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# Language Deficits Assessed in an Aphasic Patient

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Language Deficits Assessed in an Aphasic Patient

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Abstract

Aphasia is often the result of traumatic brain injury, stroke, brain disease or infection that has affected the language control centers of the brain. Here, event related potentials (ERPs) were used to explore differences in brain activation between an aphasic patient and controls in response to specific stimuli. Previous research indicates that aphasics have a dampened ERP response to different violations of sentence structure. Our aphasic patient, WD, and seven undergraduate controls were studied in three language processing tasks while continuous electroencephalography (EEG) was recorded. First, participants listened to a series of audio sentences that contained subject verb violations. Next, participants listened to a series of audio sentences that contained context mismatches. Finally, participants completed fill in the blank sentences by voicing the completing word aloud. The P600, N400, and P300 ERP components were examined in brain areas of interest, particularly in frontal regions. WD showed quantitative and qualitative ERP changes in all tasks revealing an atypical neural response despite typical behavioral performance.

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# Language Deficits Assessed in an Aphasic Patient

Aphasia is defined by the National Aphasia Association as "an impairment of language, affecting the production or comprehension of speech and the ability to read or write" (National Aphasia Association, 2014). This is most often due to stroke or traumatic brain injury but can also be the result of a brain infection or brain cancers. The National Institute of Neurological Disorders and Stroke (NINDS) estimates that approximately 1 in 250 Americans suffer from aphasia, however, there is a broad spectrum of the severity of aphasia symptoms. Some patients can be so severely inhibited with global loss of function that they are generally unable to communicate and participate in society whereas some patients will exhibit only mild, specific losses of function in language and speech. There are also several varieties of aphasia based on location of injury and presentation of symptoms including global, Broca's, mixed, Wernicke's, and anomic aphasia.

Aphasia is a prime example of the correlation between location of injury and loss of function. It provides one of the most clear cut cases of impairment because Broca's area, and the left hemisphere in general, are so specifically linked to language processing. The present study examined a Broca's aphasic patient, WD, diagnosed with aphasia following a traumatic brain injury suffered from a vehicular accident. Case WD offers a unique opportunity to link his language impairment from traumatic brain injury to shifts in neural processing.

Typically, damage to Broca's area in the left frontal lobe of the brain results in difficulty producing words or retrieving words. Broca's aphasics are often only able to speak in shorter, stilted sentences. Because they have no deficit in language

comprehension, they are very aware of their limitations, which can be a frustrating reality for such individuals. It can be difficult to diagnose a single variety of aphasia because injury often spans more than one focused area affecting multiple parts of the brain. For example, in the case of a stroke, brain damage may include Broca's area but may be diffuse enough to also impair other cognitive processing skills. Therefore, the diagnosis of aphasia is made based on a combination of physiological evidence with the help of brain imaging and EEG studies as well as evaluation of a patient's ability to speak, comprehend speech, read and write. While it is not usually possible to cure and completely resolve aphasia, many methods of treatment are currently in use, including speech therapy, to improve symptoms over time after a brain injury event. There is also evidence of brain plasticity in patients to transfer function to uninjured parts of the brain. Given the rather high incidence of aphasia, many patients that are diagnosed with aphasia do continue to be high functioning members of society with treatment and find ways to cope and participate in daily life. Because aphasics are often highly functioning, as is the case of WD, we were keen to examine whether and how much brain activity changes in such a case. In the present study, electroencephalogram (EEG) and event related potentials (ERPs) were used to draw these assessments.

*Event Related Potentials*

Event related potentials are neural responses to specific stimuli measured with electroencephalogram. These ERPs reliably occur a certain number of milliseconds after the onset of the stimuli; a phenomenon which is called latency. ERP waveforms can be positive or negative-going in response to different stimuli. ERPs are named based on latency and waveform direction. For example, the P600 ERP is positive-going, meaning

a waveform with a positive amplitude, and occurs at a latency 600 milliseconds after stimulus onset. Several ERP studies over the years have shown that certain event related potentials are elicited in healthy patients in response to specific language mechanisms and are key in understanding how language is processed within the brain. These ERPs are therefore potential tools key in diagnosing brain changes in abnormal brains such as an aphasic brain.

*Studying ERPs in healthy subjects*

The P600, a positive going waveform amplitude around 600 milliseconds after stimulus onset, is associated with the processing of grammatical anomalies or incongruities (Gouvea, Kazanina, Phillips & Poeppel, 2009). It has been elicited in response to subject-verb agreement violations as shown by Herten, Kolk, and Chwilla in 2005. For example, several syntactic mismatches in sentences elicited the P600 event related potential in healthy patients, which makes it extremely useful in determining differences in brain activation in abnormal brains.

The N400 event related potential, a negative going waveform at 400 milliseconds after stimulus onset, is elicited in response to context-mismatches in written and auditory sentences (Kutas & Federmeier, 2010) as well as in the Stroop color-word matching task (Liotti et. al, 2000). In 2008, Lau, Phillips, and Poeppel reviewed the N400 event related potential within several contexts: storing and accessing lexical information, combining lexical representations with context, and selecting and controlling retrieval of lexical representations. The consistency of elicited ERPs between healthy subjects has been shown in several studies.

The P300 event related potential is implicated in decision-making and selection processes. This is another positive waveform, but it peaks around 300 milliseconds after stimulus onset. Patel and Azzam (2005) reviewed the current understandings of the diagnostic abilities of the P300 event related potential. They cited the usefulness of the P300 in studying the Stroop color-word matching task, recognition, memory-updating tasks, as well as other working memory tasks.

Across typical participants, the consistency of ERP elicitation in response to specific stimuli makes ERPs a useful tool in understanding language mechanisms, but also as a comparison in studying brain changes, such as in aphasia.

*Using ERPs to study aphasia*

In 2009, Costa, Strijkers, Martin, and Thierry explored the timing of lexical access of word retrieval from long-term memory using the P2, N2 and P3 event related potentials in typical participants. Researchers used a picture-naming task where participants were showed a picture and then were asked to name it aloud. This task offers an opportunity to further examine the disconnect between knowing a word and the ability to actually retrieve the word to say aloud that many aphasics experience. That is, in studying the underlying mechanism in a healthy brain, there is potential to find new directions in studying abnormal word retrieval in aphasics. Researchers found that event related potential amplitudes began 200 msec after being shown the picture and lasted for 180 msec. This forms a time period of a healthy subject for brain activation in object naming and can be used as a standard to compare the brain activity of aphasic patients against.

Laganaro and colleagues (2010) were able to study a patient with anomic aphasia who had, by chance, participated in an EEG picture-naming task just a year prior to his stroke. Anomia is an inability to supply words in speech and in writing, particularly nouns and verbs. Anomic aphasics' grammatical form, comprehension, and reading abilities are left intact. The individual at the time of this study was a 68-year-old male and retired psychologist who had suffered a stroke in his left temporal lobe. With the prior EEG data in hand, researchers were able to compare brain activation before and after the stroke in the same patient as well as to controls, a rare event.

Behaviorally, the patient exhibited severe expressive aphasia with difficulties finding words, reading aloud, and comprehending auditory and written sentences. A picture-naming task was used that involved picture viewing and a required response of labeling aloud. The event related potential data revealed that the patient had ERP changes 250 msec after picture onset that was different from his pre-stroke responses as well as different from age-matched control participants. The researchers concluded that these results suggest the important of "the role of left temporal cortex in lexical-phonological processing from about 250-450 msec during word production" (Laganaro et al., 2010, p. 346).

Wassenaar and Hagoort (2005) studied event related potential activation in word-category violations in aphasia patients. They used three groups: healthy controls, Broca's aphasia patients, and non-aphasic patients with a right hemisphere lesion. Patients were asked to visually read sentences that were either correct or contained word-category violations and to pay attention to comprehending the sentences and noticing errors. This study was completed in conjunction with a continuous EEG recording. It was shown that

the healthy controls and right hemisphere lesion groups showed a P600 effect whereas the Broca's aphasia group had a delayed or absent P600 component.

In 2006, Wassenaar, Brown, and Hagoort studied event related activation in subject-verb agreement violations using an oddball paradigm task with aphasia patients. In this task, participants were asked to listen to sentences that had subject-verb disagreements. The oddball paradigm task involves singling out and differentiating different tones. Using EEG, they recorded event related potentials in patients with Broca's aphasia specifically and compared them to non-aphasic patients with right-hemisphere lesions as well as to healthy, control subjects.

Healthy subjects and non-aphasic patients with the right-hemisphere lesion both showed a P600 event related potential in response to agreement violations. The Broca's aphasia patients, however, had a depleted or absent P600 event related potential. This suggests that Broca's aphasics do not have a similar sensitivity to subject-verb agreement violations as a result of aphasia. However, the Broca's patients did have P300 activation during the oddball task with no statistically significant difference from the healthy control or right-hemisphere lesion groups suggesting that aphasia did not affect this cognitive process. This study showed the subtleties of what event related potentials are affected by Broca's aphasia and which are left intact.

In 2002, Marchand, D'Arcy, and Connolly used a combination of ERPs with behavioral tasks to develop a more comprehensive way of assessing deficits in aphasics. They used the Peabody Picture Vocabulary Test-Revised during which a picture is shown with a congruent or incongruent spoken word. For example, a hammer might be shown with the word "book". This task was presented while running continuous EEG and the

N400 event related potential was analyzed. They found that the N400 event related potential was elicited in specific response to incongruent spoken words. They used this EEG study in conjunction with a language comprehension text and found that the ERP data was positively and linearly correlated with the neuropsychological data of language comprehension. That is, the ERP data provided a useful of a means to explore and describe language deficits in aphasia patients, much like behavioral tasks may be used.

Kumar and Goswami (2013) studied the reaction times in tasks associated with frequently and infrequently used words. Participants were to press '1' when a frequent word was presented and '2' when an infrequent word was presented while recording continuous EEG. It was found that Broca's aphasia patients had overall slower and less accurate reaction times. Both groups showed faster reaction times for frequent words and both groups showed a longer latency interval on infrequent words when analyzing the N400 event related potential. This shows that Broca's patients have a semantic categorization deficit as compared to controls.

*The present study*

The current study aimed to further explore differences in brain activation in an aphasic patient. Several tasks were examined. In the first task and second tasks, subject/verb agreement violation and context mismatch were presented as a means of establishing a baseline comparison of ERPs between patient WD and controls. Both these studies were replicated based on the current literature and were used as controls. I hypothesized that the P600 ERP elicitation would be dampened or absent in patient WD as compared to controls when listening to subject-verb agreement violations and that the N400 ERP would be dampened or absent in WD as compared to controls when listening

to context mismatches. If this hypothesis is correct, it would be due to brain injury in the language control centers of the brain. The current study also sought to explore the mechanism behind the difficulty Broca's aphasics' experience with word retrieval. This task was newly designed and executed by this researcher. This third task was newly developed for this study to explore the P300 and P600 ERPs elicited during word retrieval in a fill in the blank task. For example, a sentence might read "Jane spread butter on her + _____", and the participant would be asked to complete the sentence based on the context. I hypothesized that there may be a dampening or absence of ERP elicitation in patient WD when compared to controls during attention throughout sentence comprehension, word retrieval for sentence completion, and initiation to speak.

## Method

### *Participants*

Seven male students were recruited as participants from the College of William and Mary Research Participation Pool, and either received course credit in exchange for their participation or volunteered their time. Participant controls were all right handed and between the ages of 18 and 23 with a mean age of 20.7, SD=1.8. Controls self-reported no prior brain injury or other known brain pathologies or abnormalities.

The focus of this case study was a 30 year old male Broca's aphasic, referred to as WD. WD was 17 years old when he was a pedestrian struck by a vehicle in 2001. He suffered several open, depressed skull fractures and bilateral temporal contusions noted on CT brain scan, as well as several other traumatic, bodily injuries. During surgery, WD underwent debridement of the depressed skull fracture as well as debridement of the underlying contused temporal lobe. WD remained in a coma for several days before

stabilizing at a chronic vegetative state with slow but steady improvement. WD was discharged from inpatient rehabilitation 3 months later with dramatic progress from his initial admittance to the hospital. Upon discharge, patient WD was diagnosed with a dense Broca's Aphasia, though the severity of his aphasia has improved since his accident. This initial diagnosis has not been re-evaluated since the time of discharge in 2002.

*Apparatus*

For data acquisition, continuous EEG data was recorded using a DPBA-1 Sensorium bio-amplifier (Sensorium INC., Charlotte, VT). A 10-5 cap system with 74 AgCl electrodes (Electrode Arrays, El Paso, TX) was used for each participant. Participants were fitted with the EEG cap and the ground electrode and M2 reference electrode were filled with gel and attached to the center of the forehead and the tip of the nose, respectively, with electrode adhesive. Electrodes HEOGR, HEOGL, VEOGR+, VEOGR-, VEOGL+, VEOGL- were filled with gel and attached to the corner of both eyes and above and below each eye to record vertical and horizontal eye movements and monitor for ocular artifact. The scalp electrodes were then filled with gel and impedances were monitored using Acquire. During the tasks, the experimenter ensured each task ran correctly and that Acquire recorded data continuously and correctly. Upon completion of the tasks, the electrode cap was cleaned and sterilized.

*Procedure and Stimuli*

Upon arrival, participants were asked to remove all electronics prior to entry into the EEG room and were tested for an allergy to Quik-Gel conductive gel. Participants' skin was cleaned using exfoliating scrub and alcohol pads at electrode sites

on the face. A file was created for each participant. Participants were instructed to keep as still as possible and to limit eye blinks and facial muscle movement. Participants were then presented with three tasks that were run with continuous EEG and were given a short break between trials.

Stimuli were presented using E-Prime. The first task explored subject verb agreement in audio-recorded sentences. Thirty sentences were played aloud in sequence, 24 of which contained subject-verb agreement violations and 6 of which contained correct subject-verb agreement. The correct and incorrect sentences were mixed together. This task attempted to establish whether the aphasic brain could establish subject-verb agreement due to possible difficulty with maintaining information across clausal boundaries.

The second task explored context mismatch in audio-recorded sentences. Thirty sentences were played aloud in sequence, 24 of which contained context mismatches with the last word of the sentence and 6 of which contained correct sentence contextual agreement with the last word of the sentence. The correct and incorrect sentences were mixed together. This task attempted to evaluate auditory comprehension and attention as well as ability to store, access, and combine lexical representation within sentence context. In both the first and second tasks, participants listened actively to the sentences and were not required to indicate which sentences contained violations. The correct and incorrect sentences were coded with triggers named with numbers that onset at the beginning of each sentence (subject-verb violation=10; subject verb correct=20; context mismatch=30; context agreement=40). Participants were asked to keep as still as possible and to listen carefully to each sentence. Each sentence was about 5 seconds long

with the subject-verb agreement occurring 1-2 seconds after the start of the sentence, depending on the sentence.

The third task consisted of thirty fill in the blank sentences. These sentences appeared on the computer monitor one word at a time to limit horizontal eye movement and required one word to complete the sentence based on context. The words appeared on the monitor every 0.8 seconds followed by a cross then a blank line. Participants were asked to think of the word that completed the sentence when they saw the cross appear on the screen, and then were asked to speak that word aloud when they saw a blank line appear on the screen. A trigger was placed with the blank line. There was 1 second between each sentence and 0.8 seconds between each word, the cross, and the blank line. This was to ensure that the responses were rapid and more automatically driven. This task evaluated the participants' ability to maintain attention while reading each word to understand the context, to retrieve the correct word to complete the sentence, and to initiate speaking.

*EEG data processing*

All data processing and analysis was completed using EEGLAB and ERPLAB for MATLAB. All data files were preliminarily cleaned by rejecting all obvious artifacts such as any muscle movement and channel drift that occurred during each trial. Due to the relatively small number of trials in this experiment, artifact rejection was fairly conservative.

For the first and second tasks, triggers were placed at the onset of each sentence due to programming limitations. In order to have a trigger placed at the onset of the second word in each subject-verb pair or context mismatch pair, it was determined at

what time (in seconds to the 3rd decimal place) the subject-verb pair or context mismatch pair was complete in each sentence. In other words, just prior to the verb or final word of the sentence was spoken. This time was determined using Audacity for each audio sentence. The event value was then manually edited in each participant's data so that the trigger occurred just prior to the onset of the second word in each subject-verb or context mismatch pair.

Next, bad segments and bad channels were identified and any bad channels were replaced by averaging spherically from the surrounding channels. Ocular artifact was accounted for, and blinks and horizontal eye movement components were removed. Bins, which segmented the data into pieces, were assigned based on type of trigger event. Each trigger was binned based on its identifier (subject-verb correct was bin 1, subject-verb incorrect was bin 2, context correct was bin 3, context mismatch was bin 4, and fill in the blank was bin 5). This segmented the EEG data into 2000 msec epochs and data was plotted topographically 1000 msec before and after the trigger onset. Trials that had previously been marked with artifact and were rejected were not included in data analysis. After data cleaning and binning was completed, epochs were baseline corrected using the 100 msec prior to trigger onset and the ERPs in each bin were averaged together for each participant.

*Analyses*

The averages of peak ERP waveforms were computed for each control for each task at 18 electrodes of interest and at a specific latency for each task. These electrodes of interest were distributed among six regions of interest according to two topographic factors, laterality (left, center, right) and anterior/posterior. The regions of interest were

left anterior (electrodes FT7, F3, FC3), midline anterior (electrodes FZ, FCZ, CZ), right anterior (electrodes F4, FC4, FT8), left posterior (electrodes TP7, P3, CP3), midline posterior (electrodes PZ, CPZ, OZ), and right posterior (electrodes P4, CP4, TP8). These regions were chosen to examine the left hemisphere language control regions as well as any laterality and compensation that might be relevant in the right hemisphere for WD. The latency of interest for the first task was between 500 and 700 milliseconds, which encompasses the P600 component. The latency of interest for the second task was between 300 and 500 milliseconds, which encompasses the N400 component. The latency of interest for the third task included two windows, 200-400 milliseconds and 500-700 milliseconds, to encompass both the P300 and the P600 components. For each control, the peak ERP amplitudes and latencies were averaged over the 3 electrodes in that brain area to form the 6 brain areas of interest. For example FT7, F3, and FC3 were averaged together to obtain an average peak amplitude and average latency for the left anterior region of the brain. With the brain area averages and standard deviations computed from each control, a 95% confidence interval (95% CI) was obtained for the latency and amplitude over each brain area for each task. The formula used was, 95% $CI=x\pm(1.96*S_x/\sqrt{n})$, where x is the controls' mean, $S_x$ is the standard deviation, and n is the sample size. The average peak amplitude and average latency was calculated over each brain area (left anterior, central anterior, right anterior, left posterior, central posterior, right posterior) using the same 18 electrodes and the same latency windows for each task from WD's data. WD's data were converted into z-scores and were then compared to the 95% CI calculated from the controls to determine where WD fell within the 95% confidence interval. The 95% CI includes two standard deviations above and

below the mean. If WD was beyond two standard deviations from the mean (z-score>±2), then he fell outside of the 95% confidence interval and the difference of WD's data point from the controls was statistically significant and, therefore, WD was significantly different from the expected population mean.

Scalp maps of ERPs were also qualitatively compared between WD and controls for each task over the latency of interest.

## Results

*Electrophysiological data*

For the first task, the P600 ERP was measured from 18 electrodes over 6 brain areas of interest as shown in Table 1, and was topographically represented in Figure 2. WD fell within 1.15 standard deviations from the controls' mean and was well within the 95% CI for latencies in all 6 brain areas of interest, 95% CIs [618.25, 667.07], [600.96, 659.52], 588.01, 673.61], [626.41, 691.59], [622.88, 691.02], [601.70, 676.48]. Controls showed a P600 waveform in the left anterior region with a mean peak amplitude of 2.19 μV at a mean latency of 622.66 msec. In comparison, in the left anterior region WD showed a negative waveform with a peak amplitude of -1.53 μV at 644 msec. However, WD did show a large P600 component in the right anterior region with a peak amplitude of 10.12 μV at 644 msec compared to the controls which had a mean peak amplitude of 0.67 μV at a mean latency of 630.81 msec. A scalp map from a left anterior electrode, FC3, showed a positive localization in controls and negative voltages in WD over the time period of elicitation of the P600 component as shown in Figure 5.

The N400 ERP was measured for the second task from 18 electrodes over 6 brain areas of interest as shown in Table 2, and was topographically represented in Figure 3.

WD fell within 0.14 standard deviations of the controls' mean and was well within the 95% CI for controls' latencies in all 6 brain areas of interest, 95% CIs [344.04, 450.82], [356.20, 446.98], [382.96, 439.87], [357.99, 465.25], [354.73, 463.17], [356.38, 469.90]. Controls showed the N400 waveform in the all areas of interest, see Table 2 for peak amplitude values and standard deviations. WD showed a large N400 in the left anterior region with amplitude of -11.12 μV at 405.33 msec compared to the controls with mean amplitude of -7.03 μV at a mean latency of 397.43 msec. However, WD showed a dampened N400 in the central anterior and left posterior regions, as shown in Table 5. WD was 2.01 and 1.49 standard deviations away from the controls' mean in these two areas, respectively. Most dramatically, the N400 waveform was absent in the right anterior, central posterior, and right posterior regions. WD was 7.83, 4.57, 5.80 standard deviations away from the controls' mean in these three areas, respectively. Scalp maps showed overall less negativity in WD and some positive activation over the time period of elicitation of the N400 component as compared to controls, as shown in Figure 6.

On the third task, the P300 and P600 ERPs were measured in response to word retrieval during sentence completion. Behaviorally, WD completed sentences similarly to controls. For example, "He put his wallet in his ____" was completed by the word "pocket" by all participants. WD fell within 1.12 standard deviations from the controls' mean on all latencies throughout this task for all 6 brain areas of interest, 95% CIs [317.82, 374.74], [309.82, 377.04], [315.03, 376.29], [316.44, 378.42], [296.85, 381.91], [316.16, 392.22]. Controls and WD showed a strong P300 component, peak amplitude values are shown in Table 3. In the left posterior region, the controls' mean amplitude was 13.31 μV at a mean latency of 347.43 msec and WD's peak amplitude was 13.919

μV at 316 msec. All of WD's peak amplitudes fell within 1.21 standard deviations from the mean of the controls and were well within the 95% CI for P300 amplitudes in all 6 brain areas of interest, 95% CIs [9.06, 14.16], [11.41-15.45], [10.30, 13.78], [10.82, 15.80], [11.29, 16.75], [10.23, 15.75]. Five out of seven controls also showed a strong P600 component and all controls showed positive voltages in the 500-700 msec range, as shown in Table 4. WD showed no P600 component and, in fact, showed a downward slope into negative voltages after the P300 component during the 500-800 msec range as shown in Figure 4. The mean peak amplitude in the left posterior region for controls was 21.209 μV at a mean latency of 586.67 msec and for WD was -11.01 μV at 600 msec. WD was over 2 standard deviations away from the controls' peak amplitude mean in the right anterior, central anterior, right posterior, and central posterior areas (x=9.97, 9.37, 11.32, 10.88, respectively), and WD was 1.98 standard deviations away from the controls' peak amplitude mean in the left anterior region, x=10.00. Scalp maps were also compared qualitatively between WD and controls over a 200-800 msec time frame in order to encompass both the P300 and P600 components. WD's scalp map showed a P300 positive localization from 200-400 msec but then showed negative voltages after 400 msec. In comparison, controls showed more positive localization for the P300 and P600 components, but also showed more overall positive voltages from 200-800 msec, as shown in Figure 7.

## Discussion

The purpose of the current experiment was to replicate two previous ERP studies and to extend those findings to a new fill in the blank task involving word retrieval. The purpose of replicating two existing ERP studies that investigated the neural mechanisms

underlying the recognition of grammatical sentence structure and contextual sentence structure was to establish a baseline comparison between WD and the controls. With this baseline comparison, further examination of the underlying neural mechanisms in word retrieval could be more accurately and meaningfully explored.

At the time of this study, it had been 14 years since WD's accident. While WD began with a dense Broca's aphasia, as noted above, WD's language skills have drastically improved since the time of initial diagnosis. At the time of this study, WD was able to converse in mostly full sentences with clear speech. He would use many filler words throughout his speech and would repeat the last part of words. For example, when saying the word "going", he might say "going-ing". WD also repeated phrases such as "that's what they all say" in response to explanatory and declarative sentences. Therefore, with a dramatic improvement in clinical and behavioral presentation from a dense Broca's aphasia, it was especially interesting to determine which of WD's underlying neural processes would be damaged or left intact. The comparison of left and right hemispheres was established to see if there would be any compensatory effect in the right hemisphere in WD.

In the first task, WD showed a strong P600 component in the right anterior, central posterior, and right posterior regions of the brain, but showed a negative going waveform or dampened waveform in the left anterior and left posterior brain, respectively. This suggests that some areas of WD's brain are responding to the grammatical inconsistencies, but are not responding or are not responding as strongly in the left anterior and left posterior regions of the brain, respectively. This could be attributed to damage sustained in the left temporal lobe. The fact that WD is showing a strong P600

component in the right anterior and right posterior regions of the brain suggests that the right side of his brain was compensating for the damaged left side of the brain in this task.

In the second task, WD did have a present N400 component in the left anterior brain, however WD showed an absent N400 waveform in the right anterior and right posterior brain regions and had a dampened N400 waveform in the central posterior brain regions. This suggests that WD's brain is not responding to lexical inconsistencies, as would a typical control. The fact that WD did show a strong P600 component in response to subject verb agreement violations but did not show as significant of a N400 component in response to context mismatches speaks to the subtleties of which language processes were affected by WD's traumatic brain injury.

In the third task, it is important to note that in terms of the words chosen to complete the sentences, WD's and the controls' answers were considered appropriate. For example, to complete the sentence, "She sat down in the ____", WD answered, "chair". While "chair" was not the only correct answer to complete this sentence, it was a very appropriate answer. So behaviorally, WD was similar to the controls in terms of words chosen. WD did show a typical P300 component whose amplitude fell within the 95% confidence interval of the controls' mean amplitudes. However, WD did not show a P600 waveform that was strongly shown by five out of seven of the controls in the third fill in the blank task. It is possible that not all people undergo this P600 component during word retrieval, since not all the controls showed it, and that WD happens to not undergo this P600 component as well. However, while not all the controls showed a strong P600 component, all controls did show positive voltages over the 500-700 msec range. WD, on the other hand, showed very negative voltages over this time range. This

suggests that WD underwent different neural processing during word retrieval throughout this task. However, since WD still chose appropriate words to complete each sentence, it is possible that other parts of the brain were compensating in order for WD to arrive at a correct response to complete each sentence. It is plausible that the P600 component acts as a check or verification of the word retrieved based on sentence context and that WD's brain did not experience this.

*Conclusion*

It is clear that more data must be collected before reaching any solid conclusions from this study. WD's diagnosis of Broca's aphasia may still be physiologically relevant in terms of area of damage, however his clinical presentation has obviously improved with speech therapy during his inpatient therapy after discharge from the ICU and with time since the accident. It is also possible that healing has taken place in his temporal lobe since his accident, which accounts for some of the intact the ERP activation in the first two tasks. It would be helpful to have a comparison CT head scan from the present day to compare to his CT head scan from the time of the accident. Future studies should involve a more comprehensive evaluation of WD's language deficits. This could further clarify why WD showed strong ERP elicitation in response to subject verb agreement violations and context mismatch, but did not show the second ERP waveform in the fill in the blank task. Because each aphasia case is unique and often spans borders of diagnosis classification, it is difficult to extend this case study in a broader sense to other Broca's aphasics. Still, further research focused on WD could shed light onto how the brain copes years after traumatic injury and how the language processing centers in the brain can heal or compensate over time.

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Table Captions

*Table 1* Latency mean and standard deviation, peak amplitude mean and standard deviation at brain areas of interest for subject verb agreement task 1.

*Table 2* Latency mean and standard deviation, peak amplitude mean and standard deviation at brain areas of interest for context mismatch task 2.

*Table 3* Latency mean and standard deviation, peak amplitude mean and standard deviation at brain areas of interest for fill in the blank task 3 from 200-400 msec.

*Table 4* Latency mean and standard deviation, peak amplitude mean and standard deviation at brain areas of interest for fill in the blank task 3 from 500-700 msec.

*Table 5* Range of 95% CI of latency and peak amplitude and WD's z-score for brain areas of interest across subject verb task 1 and context mismatch task 2.

*Table 6* Range of 95% CI of latency and peak amplitude and WD's z-score for brain areas of interest across fill in the blank 200-400 msec and fill in the blank 500-700 msec.

Table 1 Subject Verb Task

| | Brain Region | Laterality | Latency (msec) | | Peak Amplitude (μV) | |
|---|---|---|---|---|---|---|
| WD | Anterior | Left | 644 | | -1.53 | |
| | | Center | 652 | | 6.77 | |
| | | Right | 657.33 | | 10.12 | |
| | Posterior | Left | 654.66 | | 3.38 | |
| | | Center | 657.33 | | 7.01 | |
| | | Right | 660 | | 7.40 | |
| | | | Latency Mean (msec) | Standard Deviation | Peak Amplitude Mean (μV) | Standard Deviation |
| Controls | Anterior | Left | 622.66 | 47.96 | 2.19 | 2.47 |
| | | Center | 630.24 | 31.62 | 1.89 | 3.30 |
| | | Right | 630.81 | 23.11 | 0.67 | 3.30 |
| | Posterior | Left | 659 | 35.19 | 1.39 | 3.73 |
| | | Center | 656.95 | 36.79 | 2.40 | 3.00 |
| | | Right | 638.09 | 40.38 | 1.20 | 2.68 |

Table 2 Context Mismatch Task

| | Brain Region | Laterality | Latency (msec) | | Peak Amplitude (μV) | |
|---|---|---|---|---|---|---|
| WD | Anterior | Left | 405.33 | | -11.12 | |
| | | Center | 408 | | -4.82 | |
| | | Right | 408 | | 0.79 | |
| | Posterior | Left | 406.66 | | -5.19 | |
| | | Center | 408 | | -1.26 | |
| | | Right | 408 | | 0.73 | |
| | | | Latency Mean (msec) | Standard Deviation | Peak Amplitude Mean (μV) | Standard Deviation |
| Controls | Anterior | Left | 397.53 | 57.66 | -7.03 | 1.46 |
| | | Center | 402.09 | 49.56 | -7.78 | 1.47 |
| | | Right | 411.42 | 30.73 | -7.35 | 1.04 |
| | Posterior | Left | 411.62 | 57.91 | -7.05 | 1.25 |
| | | Center | 408.95 | 58.55 | -7.01 | 1.26 |
| | | Right | 413.14 | 61.29 | -7.30 | 1.39 |

Table 3 Fill in the Blank 200-400 msec

| | Brain Region | Laterality | Latency (msec) | | Peak Amplitude (μV) | |
|---|---|---|---|---|---|---|
| WD | Anterior | Left | 330.66 | | 13.73 | |
| | | Center | 329.33 | | 14.67 | |
| | | Right | 332 | | 12.45 | |
| | Posterior | Left | 316 | | 13.92 | |
| | | Center | 321.33 | | 14.22 | |
| | | Right | 308 | | 12.65 | |
| | | | Latency Mean (msec) | Standard Deviation | Peak Amplitude Mean (μV) | Standard Deviation |
| Controls | Anterior | Left | 346.28 | 30.73 | 11.61 | 2.75 |
| | | Center | 343.43 | 36.30 | 13.43 | 2.18 |
| | | Right | 345.66 | 33.33 | 12.04 | 1.99 |
| | Posterior | Left | 347.43 | 33.47 | 13.31 | 2.69 |
| | | Center | 339.38 | 45.93 | 14.02 | 2.95 |
| | | Right | 354.19 | 41.07 | 12.99 | 2.98 |

Table 4 Fill in the Blank 500-700 msec

| | Brain Region | Laterality | Latency (msec) | | Peak Amplitude (μV) | |
|---|---|---|---|---|---|---|
| WD | Anterior | Left | 558.66 | | -10.53 | |
| | | Center | 560 | | -7.85 | |
| | | Right | 560 | | -11.66 | |
| | Posterior | Left | 600 | | -11.01 | |
| | | Center | 600 | | -9.53 | |
| | | Right | 586.66 | | -12.26 | |
| | | | Mean Latency (msec) | Standard Deviation | Peak Amplitude Mean (μV) | Standard Deviation |
| Controls | Anterior | Left | 588.57 | 36.80 | 10.80 | 10.80 |
| | | Center | 590.85 | 36.20 | 13.30 | 10.12 |
| | | Right | 589.33 | 36.98 | 12.32 | 10.77 |
| | Posterior | Left | 586.67 | 38.14 | 21.21 | 20.96 |
| | | Center | 579.24 | 40.73 | 14.87 | 11.75 |
| | | Right | 585.90 | 54.18 | 12.26 | 12.22 |

Table 5 Subject Verb and Context Mismatch tasks 95% CI

| | | Subject Verb Agreement | | | Context Mismatch | | |
|---|---|---|---|---|---|---|---|
| Brain region | Laterality | 95% CI latency (msec) | WD latency (msec) | WD z-score | 95% CI latency (msec) | WD latency (msec) | WD z-score |
| Anterior | Left | 618.25-667.07 | 664 | 0.45 | 344.04-450.82 | 405.33 | 0.14 |
| | Central | 600.96-659.52 | 652 | 0.69 | 356.20-446.98 | 408 | 0.12 |
| | Right | 588.01-673.61 | 657.33 | 1.15 | 382.96-439.87 | 408 | -0.11 |
| Posterior | Left | 626.41-691.59 | 654.66 | -0.12 | 357.99-465.25 | 406.66 | -0.09 |
| | Central | 622.88-691.02 | 657.33 | 0.01 | 354.73-463.17 | 408 | -0.02 |
| | Right | 601.70-676.48 | 660 | 0.54 | 356.38-469.90 | 408 | -0.08 |
| Brain region | Laterality | 95% CI amplitude (μV) | WD amplitude (μV) | WD z-score | 95% CI amplitude (μV) | WD amplitude | WD z-score |
| Anterior | Left | 0.36-4.02 | -1.53 | -1.70 | -8.39–-5.688 | -11.12 | -2.80 |
| | Central | -0.55-4.33 | 6.77 | 1.48 | -9.14–-6.42 | -4.82 | 2.01 |
| | Right | -1.66-3 | 10.12 | 2.86 | -8.31–-6.39 | 0.79 | 7.83 |
| Posterior | Left | -1.35-4.11 | 3.38 | 0.54 | -8.21–-5.89 | -5.19 | 1.49 |
| | Central | 0.18-4.62 | 7.01 | 1.53 | -8.17–-5.85 | -1.26 | 4.57 |
| | Right | -0.79-3.19 | 7.40 | 2.32 | -8.6–-6.02 | 0.73 | 5.80 |

Table 6 Fill in the Blank task 95% CI

| | | Fill in the Blank 200-400 msec | | | Fill in the Blank 500-700 msec | | |
|---|---|---|---|---|---|---|---|
| Brain region | Laterality | 95% CI latency (msec) | WD latency (msec) | WD z-score | 95% CI latency (msec) | WD latency (msec) | WD z-score |
| Anterior | Left | 317.82-374.74 | 330.66 | -0.51 | 554.49-622.65 | 558.66 | -0.81 |
| | Central | 309.82-377.04 | 329.33 | -0.39 | 557.34-624.38 | 560 | -0.85 |
| | Right | 315.03-376.29 | 332 | 2.59 | 555.09-623.57 | 560 | -0.79 |
| Posterior | Left | 316.44-378.42 | 316 | -0.94 | 551.35-621.99 | 600 | 0.35 |
| | Central | 296.85-381.91 | 321.33 | -0.39 | 541.52-616.96 | 600 | 0.51 |
| | Right | 316.16-392.22 | 308 | -1.12 | 535.73-636.07 | 586.66 | 0.01 |
| Brain region | Laterality | 95% CI amplitude (μV) | WD amplitude (μV) | WD z-score | 95% CI amplitude (μV) | WD amplitude (μV) | WD z-score |
| Anterior | Left | 9.06-14.16 | 13.73 | 1.21 | 1.73-21.73 | -10.53 | -1.98 |
| | Central | 11.41-15.45 | 14.67 | 0.57 | 3.93-22.67 | -7.85 | -2.09 |
| | Right | 10.3-13.78 | 12.45 | 0.21 | 2.35-22.29 | -11.66 | -2.23 |
| Posterior | Left | 10.82-15.80 | 13.92 | 0.23 | 1.8-40.62 | -11.01 | -1.54 |
| | Central | 11.29-16.75 | 14.22 | 0.07 | 3.99-25.75 | -9.53 | -2.08 |
| | Right | 10.23-15.75 | 12.65 | -0.11 | 0.94-23.58 | -12.26 | -2.01 |

Figure Captions

*Figure 1* Example of subject verb agreement sentence with correct subject verb agreement and incorrect subject verb agreement. Example of context agreement sentence with correct contextual agreement and incorrect contextual agreement. Violations shown in bold. Example of Fill-in-the-Blank sentence showed linearly.

*Figure 2* Topographic map of first trial comparing WD and control at electrode FC3 in the left anterior brain

*Figure 3* Topographic map of second trial comparing WD and control at electrode CP3 in the left posterior brain

*Figure 4* Topographic map of third trial comparing WD and control at electrode TP7 in the left posterior brain

*Figure 5* Scalp map of first trial comparing WD and controls, averaged over 18 electrodes from 550 -700 msec

*Figure 6* Scalp map of second trial comparing WD and controls, averaged over 18 electrodes from 300-500 msec

*Figure 7* Scalp map of third trial comparing WD and controls, averaged over 18 electrodes from 200-800 msec

Figure 1

The **bride walks** down the aisle.

**I lays** my head down on the pillow

A **mirror** shows your **reflection**.

The **light bulb** gives off **broccoli.**

On Christmas morning, the children opened their + _____

Figure 2

Control 1 v WD Subject Verb Task at Electrode FC3, P600 ERP

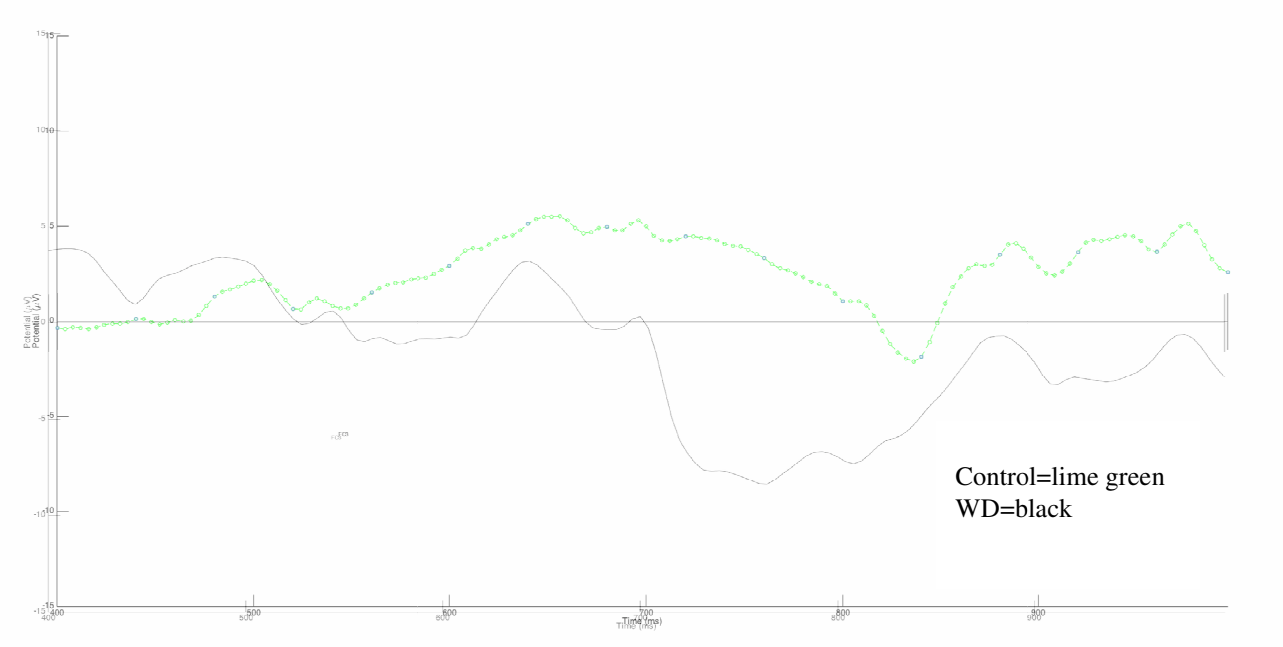


Figure 3

Control 1 v. WD Context Mismatch at Electrode CP3, N400 ERP

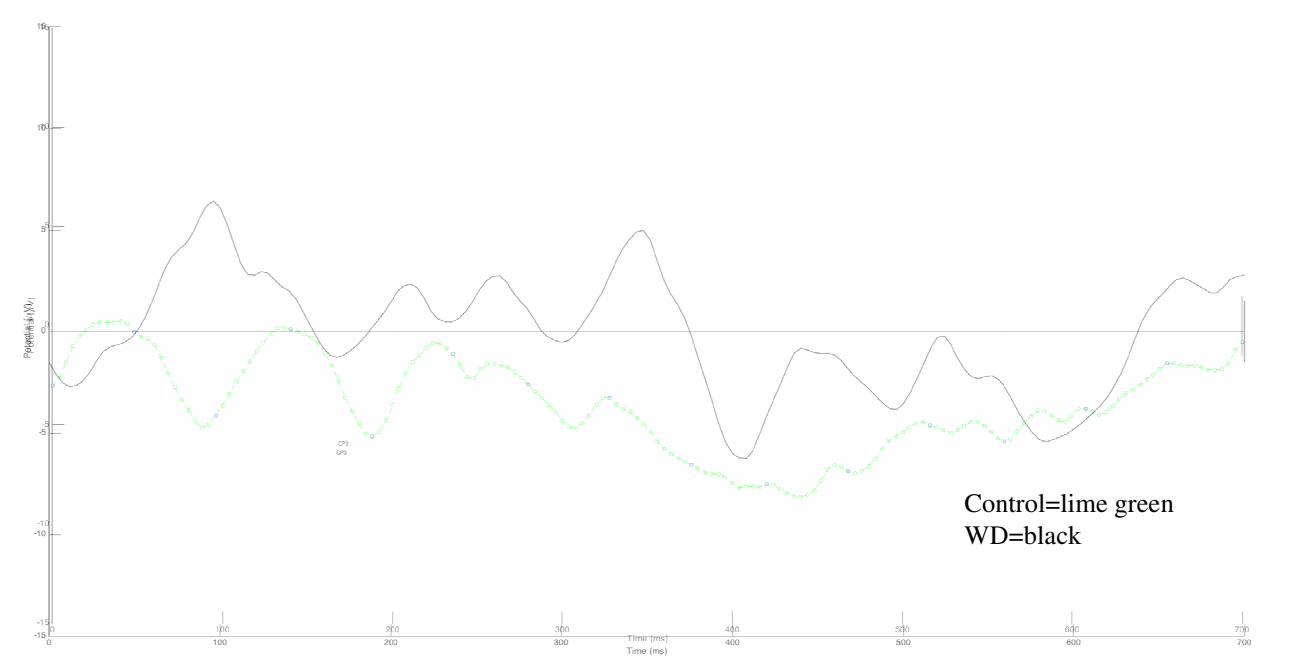


Figure 4

Controls v. WD Fill in the Blank at Electrode TP7, P300 and P600 ERP

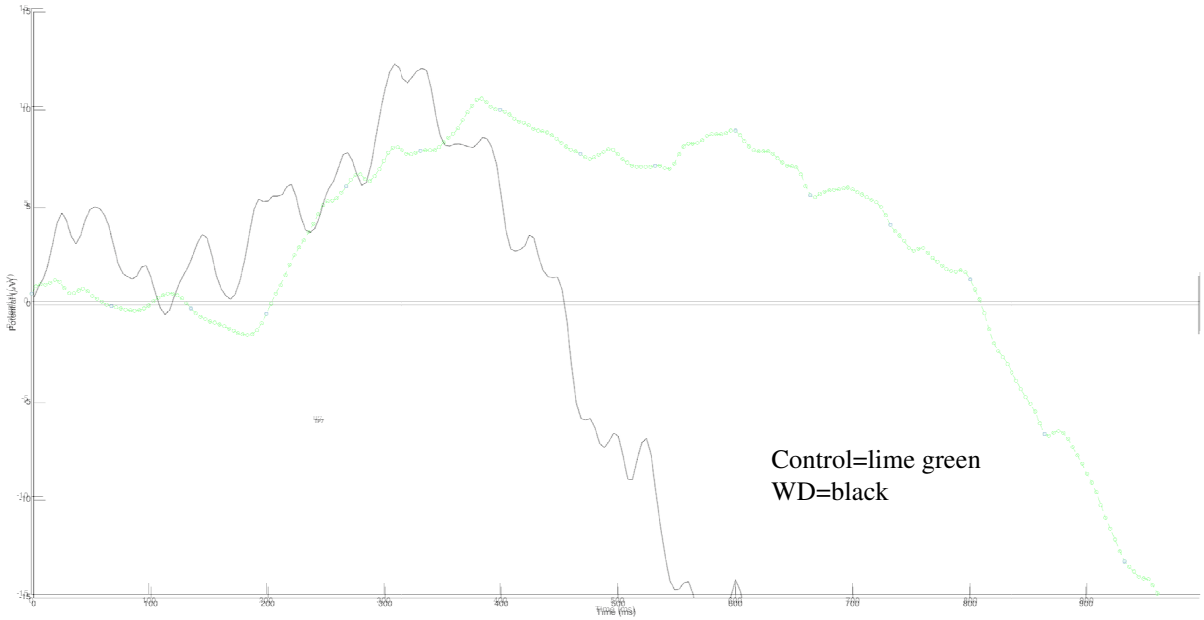
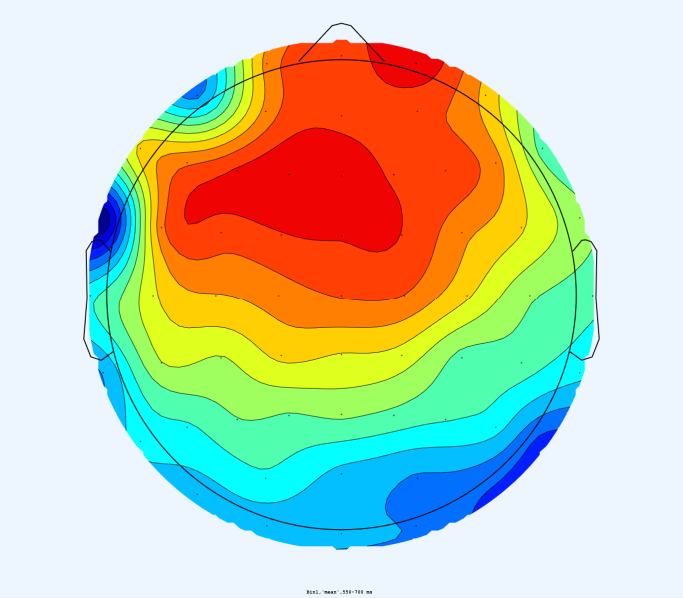


Figure 5

Control 1 Subject Verb P600 ERP



WD Subject Verb P600 ERP

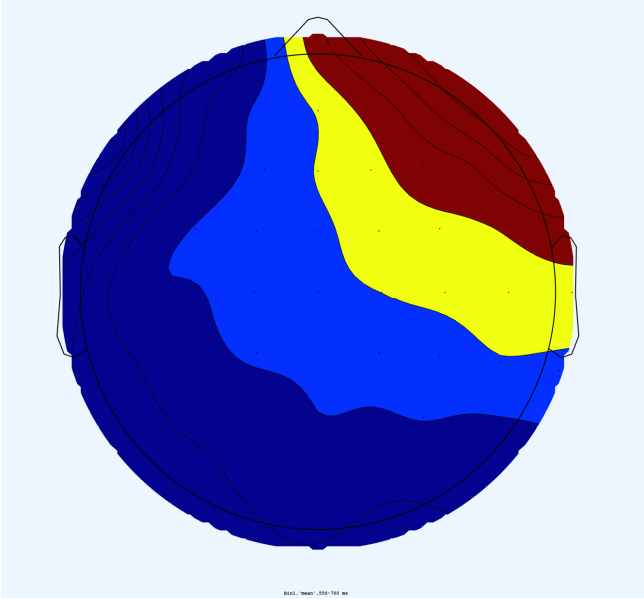
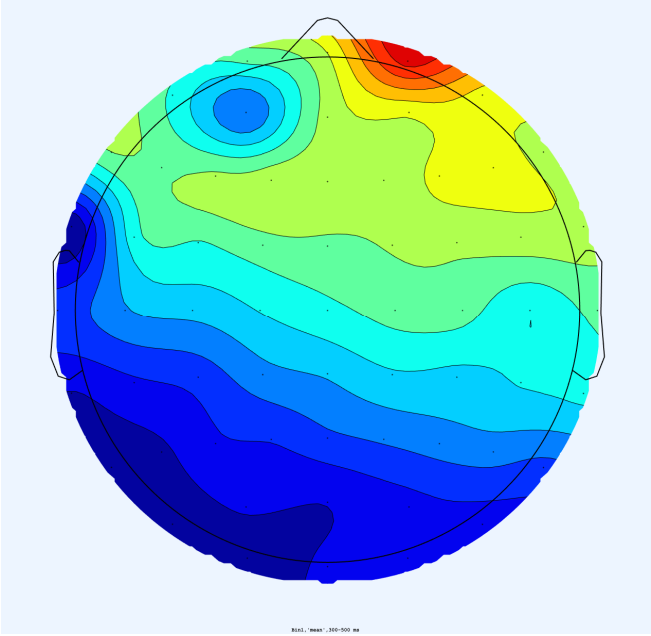


Figure 6

Control 1 Context Mismatch N400 ERP



WD Context Mismatch N400 ERP

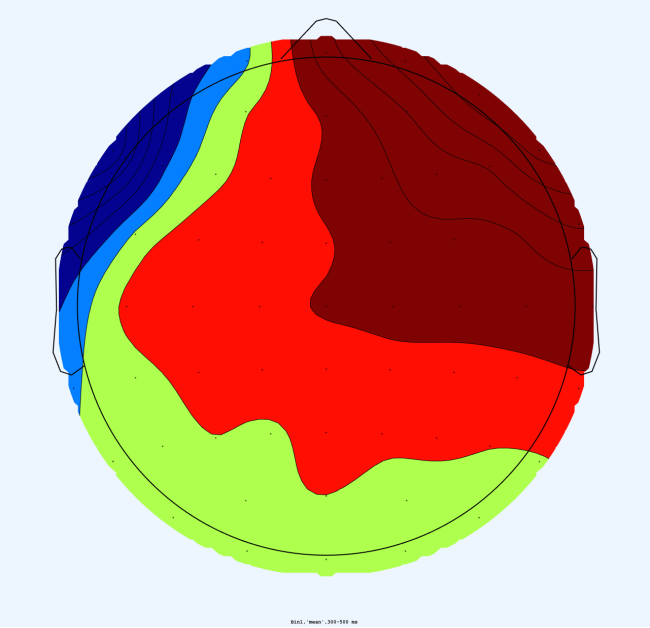
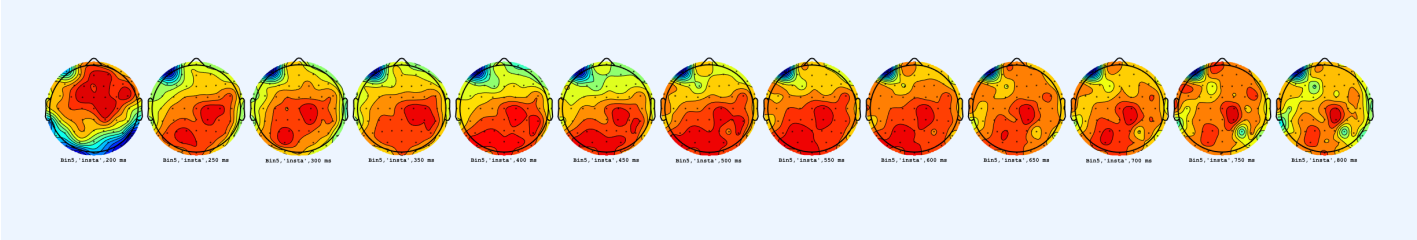


Figure 7

Controls Fill in the Blank P300 and P600 ERP from 200-800 msec



WD Fill in the Blank P300 and absent P600 ERP from 200-800 msec