Mild Traumatic Brain Injuries and Their Implications on Changes in Event Related Potentials: A look into Visual Gating (P50)

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Recommended Citation
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Mild Traumatic Brain Injuries and Their Implications on Changes in Event Related Potentials: A look into Visual Gating (P50)

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August 15th, 2018
Abstract

Concussions are a prevalent injury that affect a wide range of individuals. Commonly seen amongst individuals who play contact sports, there are many underlying factors that doctors and clinicians have yet to understand which include properties such as proper diagnosis standards or lasting impacts. In this study, we look at those impacts by using electroencephalographic (EEG) measures to study the changes in event related potentials (ERPs) associated with sensory gating and how this cognitive property is affected in those who have a self-reported concussion. Here we show that a visual attention and gating mechanism exists in both populations (control and concussed) as seen by the P50 ERP after the presentation of our visual stimulus, which is dependent on the Order the stimulus is presented (1st or 2nd). Our findings show that those who have suffered a concussion show a difference in the ability to sensory gate which is prevalent by the differences in ERPs between the two groups.
Introduction

What is a Concussion?

Mild traumatic brain injury, more commonly referred to as a concussion, occurs when the head, neck, face or surrounding area is impacted. This trauma causes both primary injury which occurs to the part of the brain where it was either directly hit (i.e. a ball hitting the front of the head would yield injury to the frontal cortex) or where the brain first strikes the skull after an impact that has not occurred directly to the head (i.e. whiplash in car accident which moves the individual in a forward motion can also cause the brain to hit the front on the skull leading to possibly primary injury of the frontal cortex). Concussions also cause injury that occurs away from the site and even at a later time, known as second injury. This can occur if the primary injury carries enough forces, leading the rebounding motion of the head bouncing in the opposite direction of the initial impact area and causing the opposite side of the brain to also strike the skull (in both examples above the secondary injury would be due to the hind-brain hitting the back of the skull). However, unlike primary injury which is typically unpredictable, secondary injury is preventable and treatable (Mendelow, & Crawford, 1997).

Figure 1: Primary and secondary injury, Mayfield Clinic (2016).
A second set of characteristics that are important to realize in the mechanistic of injury caused by concussions is the two types of acceleration forces that are present. These two forces are linear and rotational acceleration, both of which can happen simultaneously upon impact. This linear acceleration occurs when the body, head, surrounding area etc. moves in a horizontal/lateral fashion. This type of acceleration has been distinctly studied in correlation with its involvement in injury threshold (i.e. brain pressure) and has aided in developing preventative measures for injury such as helmets and air bags. The rotational acceleration occurs both internally, within the brain, as well as externally when upon impact the head gets turned to the left or right. Internally this causes the brain tissue to deform as the neurons also rotate in a similar fashion leading to shearing. Shearing, also referred to as diffuse axonal injury can ultimately lead to neuronal death, thus leading to permanent and lasting deficits (Meaney & Smith, 2011). Shearing properties, which are internal forces, can cause distortion to axons,
changes in microtubules, the rupture of blood vessels and tracts, impairments in transport (proteins, neurotransmitters, etc.) as well as aiding in neuropathological changes in brain tissue itself (Smith, Meaney & Shull, 2003).

Mechanistic injuries, which can cause neuro-metabolic alterations, commonly effects areas such as the dorsolateral pre-frontal cortex (DLPFC) and the primary motor area. Both of these areas are essential to higher cognitive functioning as well as behaviors, which are functions reported as altered when one experiences a concussion (Moore et al., 2017).

*Figure 2: Neurochemical changes due to mTBI (Gizda & Hovda, 2001).*

This worldwide ailment, which effects millions of people per year, are occurring at a largely increasing rate, nearly 75%-86% of TBIs seen are classified as a concussion (Poltavski et al., 2017). This, accompanied with the number of individuals who participate in recreational
activities and contact sports per year, leads to great concern for this type of injury if not
diagnosed or treated in an appropriate manner. This aspect, reporting and diagnosis, remains
quite elusive as there is no sound representation of concussions, as each injury presents itself
differently per individual. However, these injuries do yield some common physical and
psychological symptoms. This includes cognitive impairment (amnesia, forgetfulness, memory
loss, confusion, etc.), sleep disturbances, behavioral changes (mood swings, anger), and somatic
symptoms (headache, vision problems, nausea) (Daneshvar et al., 2011).

There have been multiple attempts over the decades to make a single parameter to
defining a concussion. One of the first parameters used to establish a definition for a concussion
is a grading system to help encompass more injuries to help with treatment. One of those systems
is defining a concussion as either mild, moderate, or severe which is based off of the Glasgow
Coma Scale (GCS), as well as as well as specific time frames for posttraumatic amnesia (PTA)
and loss of consciousness (LOC). On the other hand, the American Academy of Neurology,
Colorado Medical Society, and Cantu- revised classify concussions as Grade 1, Grade 2 and
Grade 3. While these also encompass PTA and LOC (grade 3), these definitions include
symptomology such as cognitive prognosis, confusion, amnesia and mental abnormalities lasting
for specific periods (less than 15 minutes for grade 1 and longer than 15 minutes for grade 2).
These statuses were created and are popular amongst Athletic Trainers, at both the high school
and collegiate level, because they have created a more defined parameter and elicited a better
return to play protocol for athletes (Bodin et al., 2012).

A fault in these systems is the sheer specificity of the symptoms and their duration, due to
not all patients having LOC, PTA and/or cognitive problems, yet they have sustained an impact
to the head. For this reason, it is argued that classification of concussions should become
broader, in their parameters, and encompass more people, therefore allowing a wider range of treatment. This stems from the idea of a simple vs complex concussion. Here when an individual sustains a concussion, and the injury resolves without complication in approximately 7-10 days, a diagnosis of a simplex concussion would occur. Whereas a complex concussion categorizes any individuals who have had any duration of LOC or prolonged impairment past the 10 days of a simple concussions. This broader spectrum may allow for a better diagnosis for individuals as it encompasses more into the respective “groups” (McCory et al., 2004).

Concussions, as a form of mTBI, as previously stated, have a wide range of diagnostic tools. Due to the nature of this injury, being that many arise from the participation in contact sports, such as basketball, soccer, volleyball and most prominently football, clinicians on the field (i.e. athletic trainers) rely on quick tools to diagnose. This includes methods such as the King-Devick (KD) test or the Sport Concussion Assessment Tool- 3rd Edition (SCAT3). The SCAT3 involves components such as Cognitive & Physical Evaluation, Symptom Evaluation, and balance and coordination evaluation. Yet specifically the directions state that a concussion is “clinical judgement” (SCAT 3,2013). The KD test involves a baseline screening where an individual is timed, and the number of errors are noted, as he/she reads from a pattern of numbers that increasingly gets harder. This testing is based off an individual’s eye movement, again aids in sideline removal-from-play (Galetta et al., 2011).

Classification and diagnosis of a concussion is a very subjective matter, as symptomology is not standard, and appears differently amongst the injured. The main reporting systems for concussions, self-report, is also not reliable and, more often than not, the main populations of concussed (athletes) fail to self-report their injuries. These factors can lead to longer and sustained consequences and possibly more fatal injuries. These ailments, like
cognitive impairment or attentional deficits, is why it may be pertinent to look at other factors, such as functional aspects like event related potentials (ERP), as these electrophysiological signatures can help aid in evaluating these long term effects of a concussion.

**What is an Event Related Potential?**

Previous studies have shown that concussions are known to leave lasting neurological footprint, such as, changes in the functional (Resting State) networks of the brain. Commonly, these deficits are seen within the executive functioning portions of the brain which includes items such as planning, memory, pursuing multiple tasks, and attention skills. These tasks correlate with common symptomology associated with concussions, thus providing an area of interest to study in regards to long-term effects ("Executive Functions & Self-Regulation," 2017).

While concussions present a diagnosis problem due to their large variation in aspects such as duration and symptomology, neuro imaging techniques like EEG have become more prominent in elucidating underlying changes that may have occurred. EEG reads electrical signals from the scalp, which is generated by brain activity, i.e. action potentials produced in neurons. This activity is picked up via electrodes that touch the scalp and is carried through conductance gel (Teplan, 2002). EEGs come in a variety of channel sizes from single channels to 256 channels, with greater channels leading to the ability to localize and read activity correlated to more areas ("Multi-Channel EEG (BCI) Devices," 2015). One benefit of using EEG is the portability of the equipment along with the speed of recording activity which is within fractions of seconds after the presence of a stimulus (Teplan, 2002). One of the common EEG measurements are event related potentials (ERPs) which are an informative and dynamic measurement used to chart changes in brain activity as information is processed. ERPs are
elicited by subjecting a participant to either auditory or visual stimuli, common ERPs include: N100, P50, P200, P3a and P3b, which all represent aspects of sensory gating (Lijffijt et al., 2009). Each ERP is associated with either a positive (P) peak after the stimulus has occurred, which is associated with an upward curve on the graphs, or a negative (N) downward curve after stimulus presentation (Key et al., 2005). (see Table 1).

Table 1: Event-related potential descriptions and parameters

<table>
<thead>
<tr>
<th>ERP</th>
<th>Time after stimulus present (ms)</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>P50</td>
<td>40-75ms</td>
<td>Stimulus Gating (in or out)</td>
</tr>
<tr>
<td>N100</td>
<td>90-200ms</td>
<td>Presentation of unexpected stimulus</td>
</tr>
<tr>
<td>P200</td>
<td>100-250ms</td>
<td>Allocation and filtering</td>
</tr>
<tr>
<td>N200</td>
<td>200-450ms</td>
<td>MMN, encoding stimulus change</td>
</tr>
<tr>
<td>P300 (3a&amp;3b)</td>
<td>250-400ms</td>
<td>Oddball task, Focal attention, attentional resource allocation on infrequent stimulus</td>
</tr>
</tbody>
</table>

The ERPs listed in the table above, and described below, are all commonly associated with a type of executive functioning known as sensory gating. This function acts as a mechanism to help filter out irrelevant information and protect higher cognitive functions from sensory overload. Most prominently measured by the ERP occurring 50ms after stimulus presentation (P50), sensory gating is an individual’s ability to block out repeated and competing stimuli (Lijffijt et al., 2009).
N100 reflects the neuronal activity linked to discrimination, encoding, and integrating basic stimulus. It also reflects some aspects of sensory gating as well as the properties of selective attention (Moore et al., 2017). Often times, P200 can be seen elicited in combination with N100. In these instances, P200 is referenced as being used for allocating attention as well as a filtering mechanism, while N100 is being prompted as the primary attentional aspect (Lijffijt et al., 2009). The reason these potentials are seen elicited in combination is because their times overlap with N100 occurring from 90-200ms and P200 at 100-250ms (Sur & Sinha, 2009).

The P300 component occurs when an oddball task, such as Mismatched Negativity (MMN), becomes apparent. In an oddball paradigm, when the infrequent stimulus (considered as a novel stimulus) is presented, a positive potential is presented 250-400ms after (Sur & Sinha, 2009). This potential can then be split into two separate peaks, P3a and P3b, based on their associated properties. P3a reflects aspects such as focal attention to a novel stimulus and/or distracting environment. On the other hand, P3b is seen in response to memory revision of mental events as well as changes in attentional resources allocated during a task (Moore et al., 2017). P300 is seen most over the frontal/central areas which are associated with visual, auditory, and somatosensory stimuli (Comerchero, 1999).

While the preceding ERPs are seen in an abundance amongst brain injury literature, P50 may be an even more predominant factor (Moore et al., 2017). P50 is the most positive activity occurring 40-75ms after a stimulus is present. Associated with auditory sensory gating, P50 is better known as an individual’s ability to gate in or out redundant or competing stimuli present in one’s environment (Patterson et al., 2008). If we were to correlate this term with an everyday function, it would be similar to a person listening to someone speak while music is playing. These auditory stimuli (the words of the music and words being spoken) are both competing
simultaneously for the listener’s attention and in a normal, healthy individual, they would be able to successfully block (gate) out one of these stimuli and focus (gate in) on the other. Previously, Guterman & Josiassen (1994) and Jerger et al., (1992) stated that this mechanism of sensory gating is influenced by attentional goals, with those who are able to sustain attention have a more effective sensory gating process (as cite by Yadon et al., 2009). However, as shown below, studies have found that individuals who have suffered a concussion cannot do this, and instead are receiving information from both sources.

According to Yadon (2010), sensory gating was looked at as three different mechanisms: orienting, filtering, and habituation. In the orienting phase, the brain increases it response to novel stimulus. Next is the filtering phase, which is the response of the brain to the second stimulus of an identical pair (the typical measurement for the click paradigm). The last phase is habituation where the brain’s response to stimulus becomes reduced. By measuring these three mechanisms in relation to the ERPs P50, N100 and P200, Yadon was able to show that sensory gating is a multistage process.

P50, a multistep cognitive task, is thought to involve two areas of the brain. In the early phase it involves the tempo-parietal region and the prefrontal cortex while the hippocampus is more engaged in the later phase (Kumar et al., 2005). Due to the multimodality nature of this operation, it is thought that sensory gating can occur as early as the P50 and as late as N100, giving sensory gating a range of event related potentials to look at (Lebib et al., 2003).

**Concussions and ERP**

Using an auditory paired click paradigm, along with the Immediate Memory and Delayed Memory Tasks (DMT), Lijffijt et al., studied the potential protective measures of P50, N100, and P200 using a 32 electrode EEG cap on 56 participants. Parameters for defining the ERPs
included the following: N100 was considered the first negative peak between 80-150ms, P50 was defined as either the most negative peak between 35-85ms or, if not detected there, the most positive peak preceding N100, and lastly P200 was the most positivity between 150-250ms. The study showed that there was a relationship between strong P50 sensory gating in combination with fewer commission errors and slower reaction times on the DMT. This indicates that P50 is aiding in protecting higher cognitive functioning by inhibiting interfering stimuli, while N100 was correlated with working memory and sensory processes as emanate by the relationship of higher DMT scores and a pronounced N100 wave with the presentation of the first stimulus. Like N100, P200 was found to be more associated with working memory than attention as provided by the number of correct detections in the DMT and the P200 difference scores. In combination these results suggest that there is a relationship between stronger P50, N100, P200 gating and higher task performance, thus suggesting the role that gating is a mechanism to protect higher cognitive functioning (2009).

In a 1999 study done by Comerchero and Polich sixteen subjects participated in an EEG study that presented both auditory and visual oddball stimuli, two auditory and two visuals split into easy and difficult tasks. ERP analysis showed that easy tasks with target stimuli showed P300 component most centered at the parietal lobe while difficult tasks with non-target stimuli elicited P300 at the frontal and central electrodes. The researchers also stated that amplitude effects of non-target stimulus showed a clear P3a and P3b components. This was seen because the non-target stimulus elicited a frontal P300 which resembles the P3a as non-target is considered a novel stimulus (1999).

Twenty-four participants who had experienced mild head injury, were presented with a three-stimulus oddball auditory task and asked to respond when a specific frequency was heard,
and reaction time were recorded. The digit symbol task was also given to assess verbal short-
term memory as well as the ability to rapidly shift ones’ attentional resources. These participants
showed impairment in the Digit Symbol Task, suggesting a decrease in short-term memory and
attention. With the oddball task, participants showed a decrease in N100 amplitude, slower P3b
latencies, and enhanced negativity following the P3a amplitude. All of this, in combination with
significant latency between controls and injured, suggests that mild head injury can cause
impairments in both episodic memory and attention. The researchers suggested the possible
cause of this was damage to cortical connections which show a plausible link between eye
movement and attention (Potter et al., 2001).

Duncan et al. sites a 2005 study by Solbakk et al where three different groups, 1) mTBI,
2) patients with frontal brain lesions and 3) healthy controls looked at visual stimuli while ERPs
were recorded. Eighteen participants who has moderate to severe TBI and twenty-one controls
went through this study where 360 tones were presented while ERPs were recorded at nineteen
electrodes site representing frontal, central, parietal, temporal, occipital and midline areas.
Results noted that affective stimuli elicited a larger P300 response in groups other than mTBI
where this visual ERP was seen to be reduced.

In another study done on twenty participants who had experienced a concussion at least a
year prior to the study, had accompanying impaired attention and memory and sat through EEG
readings of auditory evoked potentials. Auditory P50 is measured when paired stimuli are
presented in a 0.5 second intrapair intervals and 10-second inter-stimulus interval design through
a pair of headphones. In this study P50 was defined as the positive peak appearing between 40
and 80ms after the first stimulus was presented. The results showed a significant on the P50 ratio
by group (concussed vs control) and post-hoc comparison also showed significant ratio between
TBI subgroups and controls as well. These findings suggest that non-suppression of sensory gating is prevalent amongst patients with mild, moderate and severe TBI. This study also suggests that this may be due to injury in the hippocampus from rotational forces the brain is subjected to (Arciniegas et al., 2000).

Kumar et al. (2005) tested thirty individuals who had sustained a concussion that averaged five months prior to the beginning of the study. The participants in this study showed deficits in neuropsychological tests which included Digit Symbol Substitution Test (DSST), Digit Vigilance Test, Verbal Working Memory test, and other forms of attention and memory tasks. Results showed that 60% of people showed gating deficiencies amongst these tasks, most importantly in the DSST. Accompanying that was 87% of participants reporting post-concussive symptoms such as headache, mood change, anxiety, and memory deficits. This study also concluded that the DSST in combination with the Digit Vigilance Test were significant predictors for post-concussive symptoms (2005).

Individuals who have a self-reported a concussion, such as those in the 2002 study done by Bernstein, were shown to perform worse on the DSST, and dual tone discrimination and working memory task, as well as a reduced P300 ERP. Using EEG task and recordings on a twenty-one electrode cap, Bernstein tested 13 mTBI and 10 control participants. Concussed individuals reported symptoms such as, unconsciousness, sleep complaints, and memory problems. All participants completed four oddball tasks, both visual and auditory, two go/no-go tasks, and a pattern visual evoked potential task. The results showed that controls significantly outperformed the concussed group on the dual task (auditory and visuals) as well as outperforming in the P3 distract task. Reduced P300 amplitude in the mTBI group was seen in the auditory oddball and duration tasks (2002).
Gjini et al., studied sensory gating in the occipital visual cortex using EEG measures on healthy individuals. Two identical visual stimulus were presented to the participants. Visual stimuli were large white circles flashed on a black background of a computer as 60 pairs with interstimulus and interpair intervals at 500ms and 8s. Results showed that there was a significant decrease in both amplitudes and latency of P100 and N150. Gjini also reported an increase in the latency of the second stimulus, as compared to the first, in P100 and N75 as well as a decrease in the power for theta waves (4-8 Hz). This suggests that participants are in fact gating out irrelevant visual information as a means of aiding in selective attention and visuospatial orientation (Gjini, Sundaresan & Boutros, 2008).

Studies involving electrophysiological measures, in combination with concussions, have primarily focused on auditory event related potentials focusing on allocation of attention and memory aspects, thus leaving a gap in the how these findings are affected when looking at visually evoked potentials (although see Lifjijt et al., 2009). This has led to a fairly novel approach in regards to EEG and concussion research. Here, we look at the long-term effects concussions have on higher cognitive functions by looking at event related potentials near P50, P100, N200 and P300. Previous findings have showed that P50 was associated with the ability to either gate-in or out particular stimulus, but in the following study we show that visual attention, in concussed, leads more towards individuals over compensating in their ability to sustain visual attention, thus alluding to a system overload when it comes to sensory processing. Here we predict that individuals who have suffered a concussion will show a lowered ability to sensory gate, through changes in their ability to habituate, as compared to those who have not.

**Methods**
Participant Recruitment & Questionnaires

All methods listed below are part of our broader study which looked at the possible effects concussion can have on different cognitive functions. I will solely be focusing on our Visual Gating Protocol.

Participants for this study were recruited using SONA systems. This is an online recruiting system students use on The City College campus which allows them to sign-up for studies in exchange for extra credit points towards their psychology class. Participants were required to be at least 18 years of age. Our exclusion criteria, which was based off of our self-report questionnaires described in the Methods section, and was only if the participants had any mental disorder or a family history of them, or reported taking psychotropic medication. Our total cohort consisted of 103 participants, however for this paper, I will explicitly focus on the population recruited for the Visual Gating Task. A total of 40 participant competed the Visual Gating Task, however 14 participants were excluded do to corrupt files or excessive noise. Therefore, our final sample population was n=26(n=12 controls n=14 concussed).

Once a participant had successfully signed up and scheduled a testing date, they were brought in to the lab to begin. First, participants were briefed on what the experiment would entail and then asked to sign a consent form. Testing began with the completion of four questionnaires. This consisted of background/family history, two behavioral questionnaires, The Dark Triad which and The Depression Anxiety Stress Scales (DASS), and two concussions questionnaires, Acute Concussion Evaluation (ACE) and The ThinkFirst. The ACE, Dark Triad and DASS questionnaires were not analyzed in this part of the study and will be reported elsewhere. We did utilize the answers of the ThinkFirst questionnaire as it relayed specific answers and was geared towards asking if a concussion had occurred, what the symptomology
was and how long it may have lasted. This aided us in defining our groups. Participants were placed in either a Control or a Concussed group based on their score on the ThinkFirst Concussion Questionnaire. While we had three separate groups of concussed (see Table 2), which consisted of mild (n=6), moderate (n=5) and severe (n=3), due to the small sample size of the concussed (n=14) all participants that reported symptoms following a hit to the head, regardless of the severity or when the injury occurred, were placed into a single group for analysis.

Table 2: Groupings for the experiment (Control and Experimental)

<table>
<thead>
<tr>
<th>Type of Concussion</th>
<th>Parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Answered they had been hit in the head and either answered symptoms lasted for seconds to minutes</td>
</tr>
<tr>
<td>Moderate</td>
<td>Answered they had been hit in the head and answered that symptoms lasted hours to days</td>
</tr>
<tr>
<td>Severe</td>
<td>Answered they had been hit in the head and most consciousness and/or answered that symptoms lasted weeks on</td>
</tr>
<tr>
<td>Control</td>
<td>Answered they either have never been hit in the head on ThinkFirst questionnaire.</td>
</tr>
</tbody>
</table>

**Trail Making Tasks**

Trail Making Tasks (TMT) were utilized to test both the visual attention and task switching capabilities of both groups, as these are common aspects that are compromised and
associated with concussions. Everyone completed four different TMT. In this pen and paper task, participants had to connect the letters of the alphabet together in the correct order, as they were scattered randomly on the page. For the second TMT, participants had to connect the letters of the alphabet (A-Z) to the correct number (1-26), which again were randomly scattered on the page. For example, A-1, B-2, C-3 etc. For each of these tasks the individual was asked to read the directions aloud, asked if they understood, and given a small test practice run. Both TMT were timed and started when the participant put the pen on the paper and began connecting the cues and ended when they set the pen down. If the participant lifted their pen, which they were instructed not to do, the researcher would tally those movements and the number of times this happened. The lifting of a pen indicates that there was either some deficit in the attention and/or task switching ability of the participant, that may indicate a type of cognitive disruption.

**Digit Number Test**

This test was utilized to screen participants’ of short-term memory, ability to recall, as well as attention. To start the test, after the directions had been iterated, the researcher would say random numbers aloud to the participant and then had them recite those numbers back. This test had eight different groupings of numbers that had an increasing number of digits grouped together from two to nine numbers. The participants had two tries at the reciting the numbers back correctly. If the participant recited the numbers back correctly on the first try, the researcher moved on the next level of difficulty. If the participant did not correctly recite the numbers back on the first try they were given a second change to recite the numbers, which if they failed again to recite the numbers back then the test was completed. The second part of the Digit Number Test was like the first test but the participant had to recite the number in the reverse order they
were given to them. The same two-try process was given for five different groupings of numbers that increased in difficulty (groupings of two to six numbers).

**EEG Set-up and Recording**

The final portion of the test was conducted in a soundproof recording booth that the participant was put into so they would be isolated from any outside stimuli. The participant was seated in a chair, with a desk and computer situated two feet in front of them in the sound insulated booth. They were then fitted with a 128-electrode cap with a Duke Equidistant electrode configuration (Figure 3 below). For this study the Z7 was the reference electrode that represented the midline of not only the cap but the participants head. Other reference electrodes included the nasium (located on the nose) and VEOG (under the left eye) both of which were used to reference unwanted movements such as blinks or twitches which may alter EEG readings.
Fig 3: Equidistant Duke layout 128 channel EEG cap (ANT, 2006).

After fitting the cap to the participant all electrodes were injected with Signa, which is a water-based gel, until the impedance reading of the electrode was 5kilo-ohms (kΩ) or less. Once all electrodes were at the correct resistance, the recording phase of the test began by recordings measurement to achieve a baseline for the rest of the tests. The baseline test required the participant sat 2.5 feet from the computer screen, asked to sit with their feet on the floor, and hands in their lap. For the first recording, the participant sat in the booth with their eyes open (EO) for five minutes. This was known as our initial EO baseline and was followed up by a similar recording with the participant’s eyes closed (initial EC baseline). These two tasks, EC and EO, were repeated for two minutes each in between the presented tasks. Baseline recordings
are useful in allowing us to interpret the connections the brain makes when it is not actively viewing or interpreting a stimulus. The results of the baseline resting state network analysis will be reported elsewhere in this paper.

E-prime version 2.010.242, a computer-based program that runs the computers visual tasks and integrates the EEG recordings, was set up on a computer outside of the recording booth. After completing baseline recordings, participants were taken through four different visually based computer tasks. Tasks were presented to each of the 103 participants in a randomized order which was generated using a Latin Square Design. These tasks included Sustained Attention to Response task (SART), Mismatched Negativity (MMN), Visual Gating Task, and Digit Symbol. The Visual Gating Task was a late addition to the study and as a result only 40 participants completed that task. In SART, a Go/No Go task, measurements of both the participant’s attention abilities as well as their response inhibition recognition to changing stimuli are measured. The directions tell the participant to click the mouse every time a number other than three is seen on the screen. The participant must pay attention to the stimulus (number) and withhold from pressing the space bar when the number three appears or the mask stimulus is presented (changing stimulus). MMN is known as an oddball task the letter M and N are repeatedly flashed across the screen in a random order with two thirds of the stimuli being the letter M making the “oddball” N. This will measure the response elicited when the participant sees and recognizes that the N is different from the M. Another EEG component was Digit Symbol which measures aspects of the participants attention and memory. On the computer, participants are shown a symbol (shape) and the number it is associated with. This is repeated through a serious of shape and number combinations. Next, only the shape appears and the participant must choose which number it was originally correlated with. The computer program
scored each trial for the number of correct matches. Our last task was the Visual Gating Task which specifically measured P50, a positive measurement appearing approximately 50ms after a stimulus is presented. It measures an individual’s ability to either gate in or out competing stimuli. In our visual task, participants first saw a welcome screen, followed by a two second pause to prepare for testing. For this task, the letters x and o were randomly flashed across the screen for a duration of three seconds and forty trials. The sampling rate was 1024 times per second. Each trial began with the presentation of a focus stimulus (+) for 1000ms. Next would be a 1000ms presentation of the first stimulus followed by a 1000ms interstimulus. Following this pursuit, the second stimulus was presented and last an intertrial interval both lasting for 1000ms. Between each trial, participants were given the opportunity to rest if needed. Together, each cycle lasted approximately six minutes and was repeated three times, meaning the participant was in this paradigm for an average of eighteen minutes (not including the amount of time they chose to break between tasks). When all tasks were completed, the participant was cleaned up and thanked for their time. Contact information was given in case there were any questions they may have had.

Table 3: All tasks, their description, and measurements completed by participants in the experimental session.

<table>
<thead>
<tr>
<th>Task Name</th>
<th>Mode of Presentation</th>
<th>Description</th>
<th>Research Question</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dark Triad</td>
<td>iPad</td>
<td>Assesses the level of Machiavellianism, Narcissism, and Psychopathy</td>
<td>Do repetitive sub-concussive blows change personality traits? (not included)</td>
</tr>
<tr>
<td>Test</td>
<td>Platform</td>
<td>Measure/Task</td>
<td>Question</td>
</tr>
<tr>
<td>--------------------------</td>
<td>----------</td>
<td>-------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>DASS</td>
<td>iPad</td>
<td>Measure level of depression, anxiety, and</td>
<td>Do concussions cause psychological symptoms/cause them to progress</td>
</tr>
<tr>
<td>ThinkFirst</td>
<td>iPad</td>
<td>Concussion History</td>
<td>Aids in categorization of control vs concussed</td>
</tr>
<tr>
<td>Trail Making Task</td>
<td>Paper</td>
<td>Timed task to connect letters in numerical and alphabetical order</td>
<td>Do concussions disrupt visual attention &amp; task switching abilities</td>
</tr>
<tr>
<td>Digit Number Test</td>
<td>Paper</td>
<td>Recitation of number from short-term memory recall</td>
<td>Do concussions disrupt short-term memory storage and/or recall</td>
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<tr>
<td>Baseline</td>
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<td>Are RSN affected by concussions.</td>
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<tr>
<td>Visual Gating Task</td>
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<td>Sensory Gating</td>
<td>Changes in P50 ERP</td>
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<tr>
<td>Mini Baseline</td>
<td>E-Prime</td>
<td>RSN</td>
<td>Do RSN connectivity change after a task was presented</td>
</tr>
</tbody>
</table>
Results

While multiple tasks were listed above, as they were part of our broader study involving concussions, the following results focus on the Visual Gating Task as there was no significance difference between the groups on any of the behavioral tasks (all $p > 0.05$). There was no stimulus type (X vs O) significance, which indicates that the two stimulus type did not elicit different ERPS as can be seen below (Fig. 5-13).

ERP analysis was conducted using Fieldtrip, a MATLAB tool box specified for MEG and EEG analysis (Fieldtrip, 2018). Eye blinks and other artifacts were detected and removed using MATLAB before performing the ERP detection and analysis. Using the left and right mastoids as reference electrodes, MATLAB excluded any participant ERPs that fell one standard deviation outside of the reference electrode amplitude. Based on the previous literature regarding ERP analysis, which used 1-4 electrodes, we created the following grouping of electrodes which include those from the literature as well as additional ones within the surrounding areas: frontal Z1-5, RR1-5, LL1-5 (see Fig. 4 Red Color), occipital activity Z9-13,
RR9-13, and LL9-13 (see Fig. 4 Blue Color), central parietal LL6-8, LA3 & LA4, L7 & L8, RR6-8, RA3 & RR4, and LB3 (see Fig. 4 Yellow Color) (Lijffijt et al., 2009 & Lebib et al., 2003). Electrode RR10 was omitted from analysis due to faulty wiring in the EEG cap. For visual inspection, a time frame of 200ms preceding and 800ms following stimulus presentation were chosen.

Figure 4: ERP Analysis Electrode grouping where red represents frontal, yellow represents central-parietal and blue represents occipital.

This time frame of interest (-200ms to 800ms) was identified and extracted via MATLAB. From there, each ERP of interest was given a specific time-frame as well which were as follows: P50 (40ms-80ms), N100 (90ms-200ms), P200 (100ms-250ms), and Slow Wave (400-
600ms). By doing this we are then given one value that represents the ERP amplitude within that time frame, which then allows us to see if there are any peak shifts or delays within the data. We opted for a 2 (Group) x 2 (Order) x 2 (Stimulus Type) repeated measures analysis of variance (ANOVA) of the average amplitude of these timeframes of interest. A Holm-Bonferroni sequential correction was applied to each grouping of electrodes which expressed Order Significance (critical value 0.0033).

Depicted below in figures 5-13 are electrodes which showed a significant main effect for Group or Order, as well as an interaction between the two.

An Order effect was seen in the P50 ERP in the frontal (LL2, LL3, LL4 and RR2) and the central-parietal electrodes (L7, LL6, LL7, RR7 and Z7). The figures below display the group average amplitude for the Control and Concussed groups 200ms before and 800ms after the presentation of the stimuli. Figure 5 shows the electrophysiological activity following 1st and 2nd stimulus presentation in both the Control and Concussed groups. A repeated measures ANOVA showed significant Order effect \( p=.009, \eta^2=.242, \text{df}=1,25, F=7.985 \) but no Group or Stimulus effect for P50. The P50 is observed in the Control group between 40ms and 50ms following the presentation of the 2nd stimulus whereas the P50 in the Concussed group is roughly 60ms after the presentation of both 1st and 2nd stimuli. An N100 response is seen in the Concussed group following the 1st and 2nd stimuli. The Control group displays a clear P100 which follows the presentation of the 1st stimulus. Upon presentation of the 1st stimulus, the Control groups displays a P100 followed by a P50 from the second stimulus whereas the Concussed group displays a P50 following the presentation of both stimuli. It should also be noted that there is an
observable P200 in the Control group after both the 1<sup>st</sup> and 2<sup>nd</sup> stimuli however, no statistical analysis was conducted on this ERP as it does not pertain to the a priori hypothesis.

![LL2 AVERAGED ERP](image)

*Figure 5*: ERPs at the LL2 Frontal electrode as a 1000ms time frame, 200ms before stimulus and 800ms after, represented as Time vs Amplitude. A P50 ERP can be seen in the Control group for the second stimulus presentation, and an N100 ERP for both presentations in the Concussed group.

In Figure 6 we see the activity elicited by the presentation of 1<sup>st</sup> and 2<sup>nd</sup> stimuli in Control and Concussed measured by electrophysiological activity. Electrode LL3 showed an Order effect for ERP P50 (p=.050, $\eta^2=.146$, df=1,25 F=4.262) but no Stimulus Type of Group effect. P50 is seen in the Control group after 1<sup>st</sup> and 2<sup>nd</sup> stimulus whereas a diminished trace is seen only in the 1<sup>st</sup> stimulus of the concussed group. There is also a decrease in N100 of the Concussed group when comparing 1<sup>st</sup> stimulus presentation to 2<sup>nd</sup>. Both the 1<sup>st</sup> and 2<sup>nd</sup> stimulus presentation
caused for an event at P100 for the Control group which is not seen in the Concussed.

**Figure 6:** LL3 frontal electrode averages ERPs indicating concussed stimulus 1 and control stimulus 2 P50 as well as concussed stimulus 1 N100 (red arrows). ERPs are shown as amplitude averages over 1000ms time frame.

Figure 7 is representation of the ERPs present when 1\textsuperscript{st} and 2\textsuperscript{nd} stimulus are presented. Electrode LL4, a frontal electrode, presented with an Order effect ($p=.044$, $\eta^2=.146$, df=1,25, $F=4.505$) for P50. There was no Stimulus Type or Group effect present. Within the Control group, there is a distinct P50 for stimulus 1 and 2, with the latter showing a quickened response and increase in amplitude. This group also presents with both P100 and P200 after the 2\textsuperscript{nd} stimulus, though no further analysis was done on either of the ERPs. For the Concussed group, a P50 is seen after the 1\textsuperscript{st} stimulus. While both groups show a P50, the Control group shows a greater amplitude, from either stimulus, as compared to that of the Concussed as well as further
ERP responses.

Figure 7: Frontal electrode, LL4, averaged ERPs showing P50 for control stimulus 1 and 2 in the Control group and 1\textsuperscript{st} stimulus in Concussed group. ERPs are shown as amplitude averages over 1000ms time frame.

The last frontal electrode, Figure 8, RR2 showed a significant Order effect for P50 ($p=0.049$, $\eta^2=0.147$, df=1,25 $F=4.292$). However, there was no Stimulus Type or Group effect seen. A P50 ERP can be seen after the 1\textsuperscript{st} stimulus presentation in the Concussed group. For the Control group, the 2\textsuperscript{nd} stimulus causes a both a P50 and P100 response. Here, it seems that the P50 response of the Control group has a higher amplitude than that of the Concussed group.
Figure 8: Averaged ERP amplitudes for Control and Concussed over 1000ms time frame. Frontal electrode, RR2, showing a P50 response for both groups. The Control group displays P50 from both stimulus 1 and 2 while the Concussed group displays it from only the 1st stimulus.

Figure 9 represents electrode L7 located within the central-parietal area which showed an Order effect for P50 ($p=0.011$, $\eta^2=0.230$, df = 1, 25 $F=7.478$). There was no Stimulus type or Group effect found by the ANOVAs. The 2nd stimulus elicited a P50 ERP for the concussed group. In the Control group, there is a N100 ERP from the 1st stimulus and a P200 from the 2nd stimulus, however, no analysis was conducted on these ERPs as they were not included in the a priori hypothesis.
Figure 9: ERPs are shown as amplitude averages over 1000ms time frame for Central Parietal Electrode L7. The Control show a prominent N100 after the 1st stimulus and P200 after the 2nd stimulus.

Figure 10 represents the central-parietal electrode LL6. This electrode showed an Order effect for P50 ($p=.043, \eta^2=.154$, df+1,25 $F=4.567$), but like those electrodes preceding it, there was no Stimulus Type or Group effect. The Control group presents with a N100 after the 1st stimulus and both a P50 and P100 from the 2nd stimulus. For the Concussed group, there is a P50 present after the 1st stimulus which has a decreased amplitude as compared to that of the control group while also occurring nearly 10ms later. It is also worth noting that while the Control group has a P50 after the 2nd stimulus, the Concussed groups lacks this, thus pointing at a difference in response between the groups. It should be noted that no analysis was done on the N100 ERP as it was not pertinent to the hypothesis on hand.
Figure 10: Central-parietal electrode L7, presenting with a P50 ERP for Concussed after the 2\textsuperscript{nd} stimulus and N100 ERP for control from 1\textsuperscript{st} stimulus. ERPs are shown as amplitude averages over 1000ms time frame.

In Figure 11, electrode LL7 which represents the central-parietal area, showed an Order effect ($p=.047$, $\eta^2=.154$, df+1,25 F=4.355) for P50. There was no Stimulus Type or Group effect were seen. The Control group show a N100 after the 1\textsuperscript{st} stimulus and both a P50 and P100 after the 2\textsuperscript{nd}. No analysis was done on N100 or P100 as it does not pertain to the hypothesis at hand. For the Concussed group, a P50 is seen from the 2\textsuperscript{nd} stimulus. Upon locating the P50 displayed by both groups, it can be seen that the amplitudes differ. The Control group also shows more ERP activity, via the presence of different ERPs per stimulus, as mentioned in previous electrodes.
Figure 11: LL7, a central-parietal electrode showing N100 and P100 within the Control group and P50 within in the concussed group. ERPs are shown as amplitude averages over 1000ms time frame.

An Order effect in the P50 was observed ($p=.021$, $\eta^2=.194$, df=1,25 $F=6.030$) in the Central Parietal Electrode RR7, (see Figure 12), but no effects for Stimulus or Group observed. The Concussed group shows a P50 ERP from the 1st stimulus being presented as well as a N100 from the 2nd stimulus. For the Control group, the 1st stimulus shows a N100 while the 2nd stimulus presents with both P50 and P200. When comparing the ERP between the groups the Control group displays both a quicker ERP and higher amplitude for both P50 and N100, suggesting that there is a difference between the groups on these variables. No further analysis was conducted on the P200 and N100 as it does not pertain to the thesis at hand.
Figure 12: P50, N100, and P100 ERPs for the central-parietal electrode RR7. ERPs are shown as amplitude averages over 1000ms time frame.

The last figure, Figure 13, represents electrode Z7, a central-parietal electrode which displayed an order effect as shown by the ANOVOA ($p=.024$, $\eta^2=.187$, df=1.25 F=5.743). However, there is a lack of Stimulus or Group effect which is in line with the results seen in the previous electrodes. The Control group shows a N100 after the 1st stimulus, as well as a P50 and P100 after the 2nd. P100 and N100 are depicted as clarification for ERPs present, but no statistical analysis was completed for these two. For the Concussed group, there is a slight P50 for the 1st stimulus, like that of the Control group.
Figure 13: Z7, central-parietal averaged ERP showing P100 and N100 for stimulus 1 with Control and P50 for stimulus 2. ERPs are shown as amplitude averages over 1000ms time frame.

Discussion

While the significance of the electrodes did not survive after the Holm-Bonferroni correction, it is important to note that it is unlikely these are false-positive as our electrodes line up with the repeated significant electrodes found within the P50 literature. For example, literature cites CZ as being significant for P50 which correlates in our Duke Layout to Z7.

Previous studies have shown that visual stimuli evoke potentials such as P100, N100, P200 and P300, rather than P50. The current study showed that visual stimuli do evoke a P50 in both the Control and Concussed groups. This is a novel way to look at not only visually evoked potential, but their roles in brain injuries, as previous studies such as Lijffijt et al., Comerchero and Poich, Solbakk et al., and Arcinieagas et al., have all focused their efforts on the well-known
auditory event potential reported in individuals with brain injuries. From the graphs presented before, our data indicates two distinct phases of sensory gating. Each phase is dependent on two aspects: stimulus order (in this instance 1\textsuperscript{st} and 2\textsuperscript{nd}), and latency from stimulus presentation. All participants, regardless of being grouped into the Control group or Concussed group, displayed a P50 ERP at some point although the latency varied. However, what stands out the most is the presentation of sensory gating in the Control group, which was not observed in the Concussed group. This is apparent in a few ways which include increased amplitude and the ERP latency in the Control Group, as well as other ERPs, such as N100, P100 and P200, which appeared after the 2\textsuperscript{nd} stimulus is presented. While these were not specifically run as part of the analysis it is important to note their presence.

The idea that sensory gating consists of multiple phases is something that has been studied using an auditory paradigm amongst a healthy population. As mentioned earlier, Yadon (2010) looked at sensory gating as three phases: filtering (gating-out), orienting (gating-in) and habituation. Our data most closely represents the habituation phase of sensory gating as this aspect is dominated by attention as well as the presentation of multiple repeated stimuli. Habituation can occur in both a short-term and long-term fashion, with Johnson & Yonovitz (2017) suggesting that mid-latency ERPs like P50 and N100 can express long-term habituation. This form of habituation occurs as the response the brain has to stimuli over an extended period of time (as cited by Yadon, 2010). This would fall-in line with our experimental design which has a total amount of time spent viewing our stimuli lasting for approximately 18 minutes. Yadon found that habituation occurred as an auditory component in all three ERPs (P50, N100 and P200), as evident by the decreased response the presentation of the eight tone had as compared to the first tone (2010). Our data, while visual in nature, presented something similar.
This occurred as the Concussed group showing a decrease or a lack of P50 after the 2nd stimulus as well as an absence of N100 and P200, whereas the Control presented these three ERPs after the 2nd stimulus suggesting that, in a clinical aspect, these groups are presenting sensory gating differently. It is also apparent that research has yet to link what implications poor sensory gating has, if any, on behavior. This could be a good indication of why we did not see any statistical difference between the Control and Concussed on behavioral tasks, yet the Concussed showed a distinctly different ERP pattern.

Upon the presentation of the 1st stimulus the Control group displayed an N100 ERP which is associated with the recognition or awareness of unexpected or novel stimuli. The N100 has also been associated with automatic attentional and perceptual processing (Herrmann, Mecklinger & Pfeifer, 1998). It is thought that as a stimulus occurs and is recognized, the reaction of N100 may act to filter out irrelevant information (Lijffijt et al., 2009). In Figures 10-13 the Controls show a consistent N100, which is then consistently followed by a P50. However, the Concussed group either shows a decreased amplitude of N100 (Figure 10) or a lack of it all together (Figures 11 and 12). This is consistent with the 2001 study by Potter et al., who showed that individuals with mild traumatic head injuries had larger N100 latencies as compared with controls.

Next, at the onset of the second stimulus, the Control group showed a P50 after presentation of the stimulus. This P50 is another component associated with one’s ability to sensory gate however, this higher cognitive mechanism seems to occur after the presentation of the first stimulus and is followed by an N100. Thus it seems that the P50 is acting as a filtering mechanism by signifying that the second stimulus is no longer deemed novel, and thus requiring less attention and cognitive resources.
The location of the visually evoked potentials (VEPs) in this study, over the central parietal area, is also in line with previous research. A number of studies have shown that VEPs, most prominently those in later phases such as N100, are elicited more closely to the temporal/parietal areas of the brain, and these are thought to come from the deeper sulci of this brain region (Ibanez et al., 2012 & Creel, 2012). The auditory P50 component has also been said to be localized near the supratemporal lobe and even more specifically within the regions crossing the temporal and parietal lobes (Knott et al., 2009). Another plausible reason for the location of our VEP has to do with retinotopic mapping. Retinotopic mapping is the way in which neurons are organized to represent the visual world, i.e. the location of a visual stimulus maps to a specific area within the visual cortex. In our study, as well as previous literature, the visual stimulus is placed at a distance in the center of the participant’s visual field. Mapping of the posterior parietal cortex has been shown to be involved in the process of attentional control (Hagler et al., 2009), an aspect of our study. Thus, it is likely that the combination of stimulus presentation (at the fixation point) and retinotopic mapping of the neurons being active by this visual-attentional event could explain why these ERP are elicited near the central parietal area rather than a different location. Overall our location of ERP activity, in comparison with previous research on VEPs and in combination with the location of the auditory P50, not only confirms that we have located a true visual P50, but also suggests that both groups are trying to filter out attentional resources by “gating out” information. However, upon viewing the graphs that are present it can be seen that the Concussed group seem to do it less effectively, as evident by the differences in the ERP patterns.

Some research has suggested that P50 may have a link to the default mode network (DMN). The DMN is one of the many resting state networks of the brain has, that is the
connections between neurons during a non-active (resting) cognitive state. The DMN is defined as a region with extremely high metabolic rates and the greatest amount of deactivation during cognitive challenges from and externally presented stimuli (Deco et al., 2011). A previous study done by Castellanos et al., showed that individuals who suffered a concussion have altered connectivity in their networks due to faulty reorganizing in the brain that is occurring after the network was disrupted by the concussive impact (2010). This network, like that of the mechanisms behind P50, is linked to high-level brain functions (Griskova-Bulanova et al., 2011). In conjunction with this, studies have suggested the areas associated with this network are more synchronized during tasks in which an individual’s eyes are open and engaged in non-specific or non-goal-directed visual information gathering (Yan et al., 2009). A study done on NCAA athletes, right after sustaining a concussion, showed reduction in connectivity of the left and right parietal cortex, which are known areas of the DMN (Johnson et al., 2012.) Here in our study, we not only found a visual P50 within this area of EEG (central-parietal) but we also saw a change between presentation of it in the Concussed group as compared to the Controls. This could explain why individuals who have sustained a concussion show more difficulty with sensory gating as is evident by the lack of P50 from the visual stimuli presented. Disruption in the network from an impact to the head could cause areas once associated with the network to become disassociated or areas from other networks to become intertwined thus causing a near sensory overload leading to a failure to filter out information.

One notable results not reported in the current results section as it was not a part of the original hypothesis was an Order x Stimulus Type x Group interaction effect. The observed differences in latency and strength of the ERPs suggest that the Concussed and Control groups process the different stimuli (X vs O) in different ways, based on the stimulus temporal location
(1\textsuperscript{st} vs 2\textsuperscript{nd}). Within the Control group we see a pattern of the 1\textsuperscript{st} stimulus eliciting a P100, often followed by a P200, and the 2\textsuperscript{nd} a P50. This could indicate that upon the presentation of the 1\textsuperscript{st} stimulus the Control group recognizes the stimulus approximately 100ms after seeing it, and then 100ms after that, at P200, the visual stimulus is filtered out. However, when looking at the Concussed group we do not see a true consistency in the appearance of any ERPs let alone P50. Yet when P50 is seen it is most notably seen after the 1\textsuperscript{st} stimulus and appearing much closer to the time frame of a P100. The 2\textsuperscript{nd} stimulus often does not show any ERP associated with sensory gating. Therefore, it may be that the Concussed group is slow to recognizing the stimulus, as seen by the latency in P50, and is not ever properly gated out, thus overloading higher cognitive mechanisms. This is consistence with concussion literature which have stated that a consistency in consequences that occur due to a concussion include cognitive deficits (Boden et al., 2012). It would also explain why the Concussed group does not consistently show a P50 to the second stimulus, like the Control group does, which would thus entail properly gating out competitive stimuli.

The above statements also bring in the relevance of the group portion of this effect, as the Control and Concussed do not show the same properties of sensory gating. This is evident, as previously described, by the lack of common ERPs between the two groups, in combination with changes in latency and amplitude of those that overlap. Due to our tasks involving multi-cognitive functions, like sustained attention and recognition, and previous studies, like that of Potter et al., in 2001 which showed that in visual tasks which involved attention, individuals who has suffered a concussion showed decrease in amplitudes, longer latencies, and lack of positive ERP as compared to their control counterparts, it is apparent that this is a factor in the three-way interaction.
The last component of the interaction, the stimulus type, has to do with the fact that our experiment showed two difference visual stimuli, that would be seen as grouped together, due to the small interstimulus time frame, in one of four possible combinations when presented (XX, XO, OX, OO). The traditional click paradigm used to gauge P50, which uses clicks of the same decibels, shows that individuals who have not suffered a concussion are able to recognize that these are equivalent and lessens their cognitive response to it (i.e. gates it out). When looked at in our experiment, we would expect to see this same reaction when and individual in the Control group was presented with either XX or OO. However, when seeing the combination of XO or OX, they would recognize these are not the similar, but in fact novel, thus eliciting different responses. This is could be a factor as to why stimulus type is prevalent for this interaction. Overall, this three-way interaction is unique, yet due to the fact that only Order had a stand-alone significant effect, it is more probable that this is occurring as a “false-positive”. However, we are in the process of designing that further explore this difference.

One noticeable consistency seen within the ERP graphs were positive peaks, so called “slow-waves”, appearing in both groups between 700ms-800ms. Within the frontal Electrodes (LL2, LL3 and LL4) the Control group is seen as having a slow wave from the presentation of both stimulus 1 and 2. Among these ERPs, the second stimulus provokes a slightly decreased amplitude as well as a faster appearance. In the Central-parietal electrodes is where the slow wave appears for Concussed group. In these electrodes, the first stimulus evokes a potential for the Concussed group but not for the Control. Interestingly enough, the Control group shows a downward trend after the N100 ERP. On the other hand, the Concussed group steadily has a prominent Peak over 750ms which precedes the initial P50 ERP. When the second stimulus is presented, the Control group spikes the slow wave ERP, whereas the Concussed group shows
none at all. In this condition, the Control groups’ slow wave is preceded by a P200. This positive potential at 700ms-800ms is thought to be associated with processing pre-and post-response normally associated with cognitive control, one’s ability to select relevant information while avoiding irrelevant information during a task (Shen et al., 2013). Another study, done on visual-spatial attention using an EEG task and fMRI, sited a Willed Attention Component occurring between 400-800ms (Bengson, Kelley & Mangun, 2015).

N100, P100 and P200 were three other ERP components seen to be associated within the same electrodes that produced P50. There ERPs, as discussed in the Introduction, have been linked to sensory-gating (Lijffijt et al., 2009, Gjini et al., 2008, Moore et al., 2017 & Comerchero, 1999). Interestingly enough, P100 has also been associated with visual processing. Due to our stimuli being visual, it makes sense that P100 would show task related activation. A 2007 study done on early stage visual processing and working memory showed that a strong P100 amplitude predicted better working memory performance, more particularly during the encoding phase. It is possible that by discriminating between our visual stimulus, X or O, the control group is able to encode that they have seen the stimulus, and are encoding it into their working memory in preparation for the next stimulus, indicated by the expression of P100. Lijffijt et al. (2009), also stated that in combination the P50, N100 and P200 gating work to protect cognition by affecting response bias, behavioral inhibition, working memory, or attention. With concussion literature showing a decrease in all of these facets, it help point to why the Control group would show these ERPs whereas these patterns are abnormal in the Concussed group.

Moving forward with these findings, future studies should revolve around a multi-modality look at P50. Being that sensory gating is primarily studied as an auditory event, many studies have cited the effects of mild-traumatic brain injuries on P50. However, less research has
been conducted on visually evoked P50’s, and a very small number of studies have investigated the effects of mild traumatic injuries on visual attention. Since we have shown that there is indeed a visual P50, as well as a difference between the Control and Concussed groups in the strength and latency of the visual P50, it would be beneficial to not only extend this study to more participants but also show the difference between both the auditory and visual sensory gating event as well as their changes in frequency bands such as Delta and Alpha which have been correlated with changes in the DMN due to brain injuries (Teplan, 2002). Concussions are known to have long lasting cognitive impacts on individuals such as poor attention and memory skills. By defining differences in an individual’s natural mechanism to protect these functions, via auditory and visual gating and the changes in their frequencies, it is possible to provide and indicator in changes between cognitive networks. By showing a change in networks, weather that is through changes in connectivity, desynchronization or communication, we could open a new parameter to having proper defining terms as to what a concussion is. This type of research can have a significant and long lasting impact, especially in the realm of sports, to how the consequences of having a concussion last well beyond the physical symptoms they manifest.
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