

**Exploring the Neural Basis of
Emotional Induced Blindness
Using Event-Related Brain Potentials**

By

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ABSTRACT

Emotional induced blindness (EIB) has been demonstrated when a target follows an emotional stimulus in a rapid serial visual presentation (RSVP) stream and the subject fails to identify the target. This paradigm has been compared to the Attentional Blink (AB), a similar event in which a relevant target replaces the emotional stimuli. However, the neural mechanisms behind these two phenomena have yet to be compared. In an effort to do so we used a traditional EIB experiment but we also controlled for the physical difference of emotional stimuli with neutral counterparts. The behavioral results confirmed the role of emotion in the blink and showed that subjects were significantly worse at identifying targets in the wake of a negatively arousing image [$F(3, 52) = 32.040, p < .0001$]. Using event related brain potentials we found surprising differences between EIB and AB as well as evidence against the current accepted theories of AB.

Chapter 1

INTRODUCTION

1.1 Emotion Induced Blindness

Imagine a scenario in which you are busy on your laptop while the TV plays in the background. You have been actively surfing the Internet and have been unaware of the show that has been on for the past twenty-seven minutes. Suddenly you find yourself engulfed in a disturbing scene from the horror movie that you have been oblivious to up until this point. The question becomes, why is this the case?

From an evolutionary standpoint emotional stimuli should capture our attention. It makes sense for survival purposes to attend to information that could be potentially harmful or even extremely helpful (Hajcak, Weinberg, MacNamara & Foti, 2010). Therefore, it comes at no surprise that emotional stimuli, even when they are task irrelevant, will grab our attention (Ohmn, Flykt, & Esteves, 2001; Most, Smith, Cooter, Levy & Zald, 2007; Hajack, Weinberg, MacNamara & Foti, 2010).

We can study the process by which emotional stimuli capture attention by examining a laboratory phenomenon known as emotion-induced blindness or EIB (Most, Chun, Widders & Zald, 2005). In the EIB paradigm, people try to detect target pictures (such as a scene that is rotated left or right) occurring in a rapid serial visual presentation (RSVP) stream of pictures. When the target picture occurs shortly after presentation of an irrelevant emotional picture, people often fail to detect it and are convinced the target was not presented. A current prevailing theory among the literature says that although emotional stimuli attract spatial attention they also occupy

more processing time. Therefore, the EIB is reflective of competition in perception caused by temporal overlap between emotional distracters and relevant targets (Kennedy & Most, 2011; Most & Wang, 2011). For example, in an RSVP stream the extra time that is spent processing an emotional target coincides with the pictures that follow this stimulus. This comes at a cost to the processing of the succeeding imaging.

An important goal in understanding EIB is to determine whether the second target (T2) is being suppressed at early perceptual stages of processing or at later stages concerned with conscious awareness. Kennedy & Most (2011) minimized the role of memory by having observers make an immediate response to the target rather than responding after the RSVP stream was over. They still found a robust EIB. Most & Wang (2011) suggested that if EIB represented a perceptual level of interference, it might only occur when the emotional picture and the target occurred in the same spatial location. In contrast, interference at a more central processing stage involved in awareness of the target should be indifferent to the relative location of the two pictures. They tested this by independently placing the target and irrelevant emotional pictures in two simultaneous RSVP streams resulting in trials in which target and distracter shared the same location or appeared in different locations. They reported impairments in target detection only when both T1 and T2 appeared in the same stream. This result supports the idea that EIB is occurring at perceptual stages in which location is still important (Most & Wang, 2011).

Another critical question about EIB is the extent to which it is automatic or susceptible to “top-down” or voluntary control. Most et al (2005) addressed this by manipulating the specificity of information that they provided their participants about the target picture to determine whether this would help them ignore the emotional

picture and reduce the blink effect. They also hypothesized that individual differences in the personality trait of harm avoidance might be a contributing factor in one's ability to suppress the negative picture. The results were not entirely conclusive, however, it became clear that one's ability to override the blink depends largely on personality as they suspected. All participants experienced EIB when the target was not specified, however, when subjects were given more information those who scored low in harm avoidance could successfully overcome EIB (Most, Chun, Widders & Zald, 2005).

1.2 Attentional Blink

On the surface, emotion-induced blindness seems to be closely related to another phenomenon known as the attentional blink (AB). This refers to the severe impairment in identifying the second of two relevant targets (T1 and T2) in a RSVP task when the second target occurs within 200 to 500ms of the first (Raymond, Shaprio, & Arnell, 1992). The EIB paradigm differs from AB in that the emotional picture, which plays the role of T1 in the AB, is irrelevant but still produces a blink. Despite the similarities however, some researchers have suggested that the blink effects in the two paradigms may be fundamentally different.

In order to see some of the differences between EIB and the AB it is important to first understand the proposed mechanism responsible for the attentional blink. The widely accepted central interference theory (Chun & Potter, 1995; Isaak, Shapiro, & Martin, 1999) suggests that after an initial screening T1 is tagged as a target and moved into a limited-capacity, central resource. During this stage T1 is consolidated into working memory and achieves a durable representation. Importantly, this limited-capacity resource acts as a "bottleneck" and can operate on only one input at a time.

Therefore, while T1 is occupying this resource, T2 has to wait for access and is vulnerable to masking by subsequent pictures in the stream. As a result, although T2 is processed on a perceptual level, it never makes it into working memory and cannot be reported. This theory can also explain the *time course* (200-500ms) of the AB. The item immediately following T1 (lag 1) is not blinked because the gate to higher level processing that is initiated by the presentation of T1, is still open. This allows both the lag 1 item and T1 to enter working memory and be reported. In contrast, when T2 occurs later in the stream, for example at lag 2, the gate is now closed. T2 is unable to gain access to working memory and is masked by subsequent images, resulting in a failure to detect it.

Notice that in the EIB, the emotional picture is irrelevant so one might assume that it does not gain access to the bottleneck leading to short-term memory suggesting that it may produce a blink via a different route, perhaps by suppressing perceptual level processing of T2 as suggested by the results of Most & Wang (2011) and Kennedy & Most (2011). On the other hand, the emotional picture may *automatically* capture attention and gain access to higher-level stages, including working memory and awareness. In this case, the mechanisms underlying the blink effect in EIB and AB would be identical.

1.3 Event Related Potentials

The existing behavioral data doesn't unequivocally allow us to decide whether the blink effects in the AB and EIB paradigms are due to early or late effects and whether they are produced by the same mechanisms. The problem is measures such as accuracy in discriminating T2 are the final product of a chain of processing stages and our independent variables could be exerting their effects at any point in this chain.

Neuroimaging methods, particularly those based on EEG with its excellent temporal resolution, allow us to examine processing at any stage from onset of the stimulus to the occurrence of the motor response. This approach has been extensively applied to the AB but only sparingly to the EIB. Therefore we may be able to adapt some of these approaches to the EIB paradigm, allowing us to determine if the same processes are at work in both. First, I provide a short review of this technique and how it has been applied to the case of the AB.

The electroencephalogram or EEG represents the integrated electrical activity of billions of neurons. This makes the EEG a good measure of overall brain activity, which is useful in assessing various levels of arousal such as those that occur in different stages of sleep. It is less useful however as a measure of specific mental activities such as attention, object recognition, etc. The problem is that these mental processes only engage a small number of brain areas and the resulting signal is buried in the background activity produced by the rest of the brain. In order to extract this specific activity, the EEG from hundreds of trials that are time locked to the appearance of the stimulus that is to be processed is averaged together. This averaging process causes the unrelated brain activity to average out to zero and strengthens that activity that is reliably related to processing the stimulus. This time-locked signal is called an event-related potential or ERP (Luck, 2005). Extensive research has uncovered consistent relationships between various parts or *components* of the ERP and specific mental processes such as visual attention, decision-making, etc.

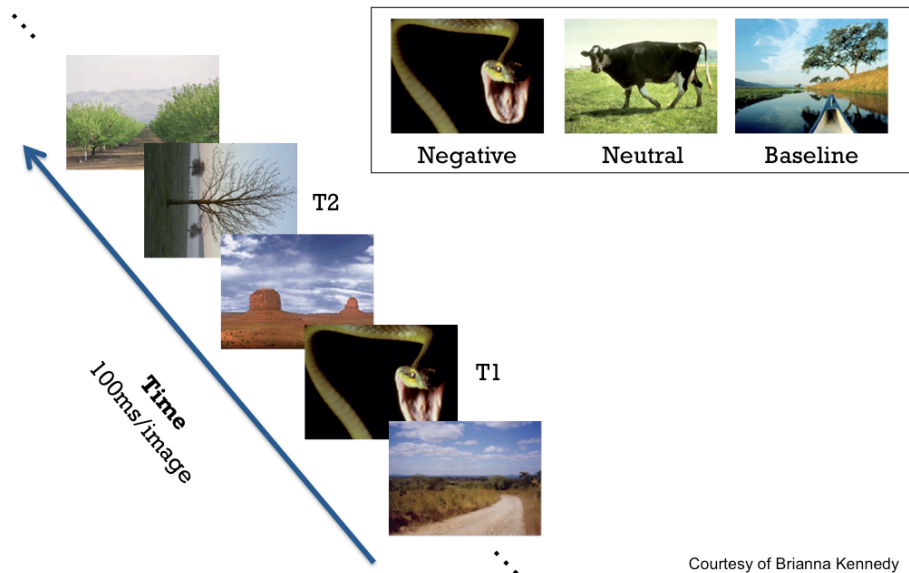
Using ERPs, researchers have confirmed some of the basic assumptions of the central interference theory as an explanation for the AB. Early peaks or components in the ERP (such as the N100 and P100), which are associated with perceptual

possessing, are not altered during the attentional blink (Vogel, Luck, & Shapiro, 1998). This provides evidence that the first stage of perceptual processing and identification posited by the central interference theory is unaffected by the blink. In contrast, a later component of the ERP called the P300, which is associated with working memory and conscious target detection, is almost completely suppressed during the blink. Consistent with central interference theories, the impairment in reporting a T2 occurring during the blink interval is likely to be a result of failures of T2 to gain access to postperceptual stages of processing associated with conscious awareness (Vogel, Luck, & Shapiro, 1998; Shapiro, Schmitz, Martens, Hommel, & Schnitzler, 2006). Research has also shown that the first target (T1) produces a robust P300. This isn't surprising because observers are supposed to report T1 and are very accurate in doing so. Accurate report of T1 suggests that it has been attended and encoded into working memory and this blocks T2 access to these resources. This failure of T2 to be encoded into working memory is reflected in an absence of P300 activity associated with T2 (Shapiro, Schmitz, Martens, Hommel, & Schnitzler, 2006). This pattern of P300 amplitude for T1 and T2 constitutes strong support for the central interference account of the blink.

1.4 Related Study

In an effort to further evaluate the proposed mechanisms of emotion-induced blindness, as well as to determine on a neural basis, the relationship between EIB and the AB, I collected ERP data during the EIB procedure. A related experiment was conducted earlier by Kennedy, Rawding, Most, & Hoffman (2012). They showed their participants an RSVP stream containing a T1 consisting of an irrelevant negative,

neutral, or scene picture (baseline). T2 was a picture rotated either left or right that could appear in the lag2 or lag8 position (see Figure 1.1).



Courtesy of Brianna Kennedy

Figure 1.1 Example of a portion of an RSVP sequence illustrating the lag2 condition. T1 could be negative, neutral, or baseline. T2 could be rotated either left or right.

If the EIB phenomenon is just like the AB we would expect a P300 to appear in response to a negative T1 even though it is irrelevant. However, Kennedy et al. did not observe any evidence of a P3 in the time interval following T1 (see Figure 1.2). Instead, they found that negative and neutral pictures produced a late P300 that appeared in the *time window corresponding to T2*. Surprisingly, this P300 occurred in the T2 P300 time window even in the lag8 condition in which T2 had not even been presented yet. Kennedy et al. hypothesized that perhaps people are using T1 as a cue for the likely temporal onset of T2. Because participants know they are likely to miss

target pictures appearing shortly after a negative or neutral picture, they may adopt a strategy of “sampling” a picture from the stream in the approximate time window corresponding to when T2 would occur. This would produce a P300 even in cases where no T2 is present. To test the idea that this P300 results from a *strategy* for detecting targets occurring shortly after T1, Kennedy et al. conducted a follow-up experiment in which T2 never followed T1 at short lags, thereby eliminating the cueing role of T1. In this case, no P300 was observed following T1, confirming that negative pictures do not automatically produce a P300.

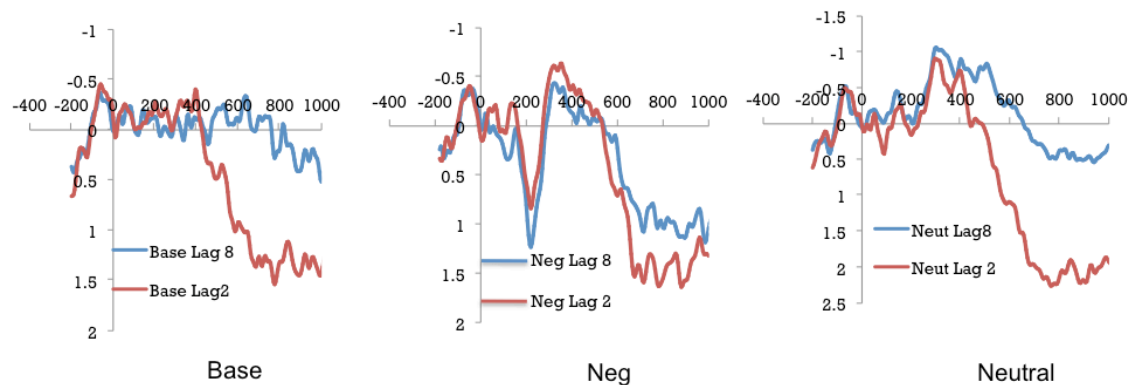


Figure 1.2 During the scene condition, there is a clear P300 beginning approximately 475ms after T2 is presented. A P300 is not observed in the lag8 condition because T2 has not appeared yet. When a negative picture is present at T1 we see a P300 with the same time course as for the base condition independent of T2. Finally in the neutral condition we see a smaller P3 when in the lag 8 conditioned compared to the lag2 condition. (from Kennedy et al., 2012)

Kennedy et al. also reported that both targets (T1 and T2) produced negative potentials over occipital sites with a latency of approximately 220msec after picture onset. This same potential has been described by other investigators (Schupp, Flaisch, Stockburger, & Junghofer, 2009) and is known as the early posterior negativity (EPN). Although not a great deal is known about the EPN, some investigators have suggested that it is specifically evoked by emotional stimuli (Schupp, Flaisch, Stockburger, & Junghofer, 2009). Kennedy et al., however, suggested that the EPN might reflect the operation of a “salience detector” which is activated by any stimulus that differs from the surrounding context. Consistent with this, they reported that neutral pictures, which portrayed people or animals in non-emotional settings, also produced a robust EPN. The rotated target picture (T2) produced an EPN as well, either because it was the only rotated picture in the stream (bottom-up salience) or because participants were set for the rotation feature (top-down salience).

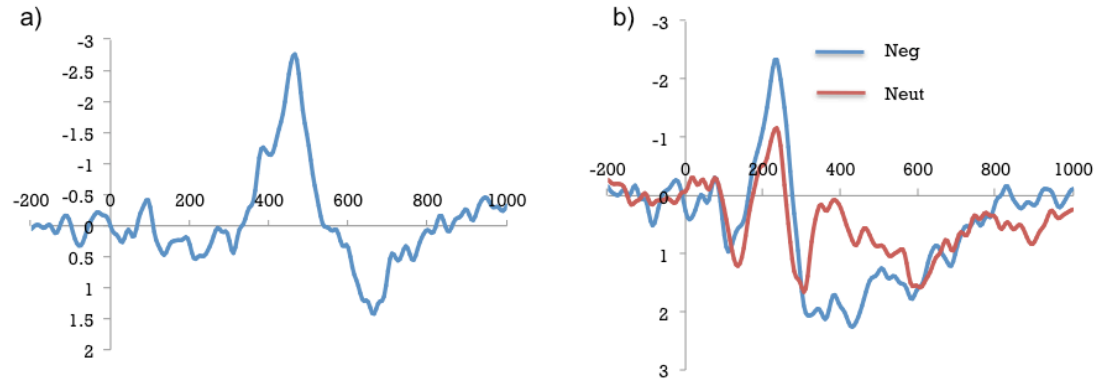


Figure 1.3 a) The EPN to T2 b) Similar components are elicited by negative and neutral T1 pictures. Note that the negative T1 elicits a larger EPN than the neutral T1.

In summary, the results here are somewhat surprising. There is no obvious P300 in the time interval after T1, where, based on the AB studies, we would expect to see it. The appearance of a P300 in a later interval independent of whether T2 is present could reflect a strategic process of looking for targets following T1. In any case it appears that T1 can elicit a blink even when it does not occupy working memory, as reflected in the absence of a P300. This brings into question the validity of the working memory bottleneck explanation of the blink. It is not clear, however, whether the attentional blink and emotional-induced blindness are unique processes or if instead the assumptions we have formed about the AB are incorrect.

Several uncertainties remain over the mechanism responsible for the EIB. Does a negative picture suppress closely following pictures because of its emotional content or its physical salience? These two features are likely to be correlated in emotional pictures, which tend to include close-ups of faces, portrayal of bright red blood, bared fangs, etc. Therefore, it could be argued that it is in fact the salient physical features of these pictures, rather than their emotional valence, that is causing the blink. There are clear physical differences between the emotional, neutral and baseline pictures, and these differences appear, at least to casual inspection, to be greatest for the negative pictures compared to baseline pictures. Perhaps these differences alone can explain the blink and emotional content is irrelevant.

This possibility can be evaluated by matching, as closely as possible, the emotional and non-emotional pictures for their physical features. I did this by using a set of stimuli introduced by Mather and Nesmith (2008). They carefully constructed pairs of pictures in which an emotional picture is paired with a control picture that is physically similar but lacks emotional content. An example is shown in figure 1.4. We

used this picture set in the current study while maintaining all other features of the experiment reported by Kennedy et al. Our intention is to determine whether the difference in blink magnitude between emotional and neutral pictures and the associated ERP components was due to differences in emotional content or differences in physical salience.



Figure 1.4 Examples of the stimuli used in the current study.

Chapter 2

METHODS

2.1 Participants

Twenty-five right-handed neurologically normal participants were paid \$10/hour for their time in this study. All subjects reported normal or corrected to normal vision and provided informed consent. Five of these subjects were eliminated from analysis due to noisy data. Since one subject failed to complete the entirety of the experiment and another subject did not exhibit a behavioral blink eighteen subjects (ages 18-29) were included in the final analysis. The University of Delaware Human Subjects Review Board approved this experiment.

2.2 Stimuli and Procedure

Stimuli were generated on a Dell 2.40 GHz computer running custom software written with Blitz3D (Sibly, 2005) and presented on a Samsung Syncmaster 2233RZ LCD display with a refresh rate of 120 Hz and a viewing window of 29 x 22 cm. Under these conditions, the total viewable area of the screen subtended approximately 20.9° x 16.1°. Testing was conducted in a dimly lit, electrically shielded room with a chinrest maintaining a 76 cm viewing distance.

Participants were first shown a blank screen with a fixation-cross of .37°x.37° located in the center. A mouse click initiated an RSVP sequence of 17 pictures at appearing at fixation. Each picture was 6.8° x 5.3° in size and appeared for 100 msec. with no interstimulus interval. A response screen containing a dialogue box asking

participants the direction of the rotated picture as well as their confidence in their own response followed the sequence. Participants were instructed to remain still and refrain from making eye blinks or eye movements throughout each trial. There were a total of 672 trials with short breaks given every 84 trials.

The RSVP stream consisted of a sequence of pictures of outdoor landscapes or city scenes. In general, all pictures were presented upright. A target picture replaced one of the images; this picture was rotated 90° to the left or right and served as T2. An out-of-category picture drawn from one of the following categories preceded T2: high negative, high negative control, moderate negative, or moderate negative control. In addition, some streams (baseline condition) substituted a scene picture for T1. T2 appeared either two (lag2) or eight (lag8) pictures after T1. The negative images and their matching control images were taken from Mather and Nesmith (2008) who matched the images on appearance, complexity, content, and focus of interest (see figure 1.4 for examples). These pictures were rated on both level of arousal as well as similarity between each control and its negative equivalent.

2.3 Electrophysiological Recording and Analysis

Continuous electroencephalogram (EEG) was collected (200-Hz sampling rate; 0.01- to 80-Hz band-pass filter; vertex reference) using a 128-channel Hydrocel Sensor Net (Tucker, 1993) with individual electrode impedances kept below 75 kΩ. EEG data were stored on a Power Mac G4 computer and processed off-line using Net Station 4.1.2 (Electrical Geodesics, Inc., Eugene, OR). Data were filtered with a 40-Hz low-pass filter. Segmentation was time-locked to onset of T1 and extended from -200

to 1,200 msec. Artifacts detected in individual channels (fast average amplitude $> 200 \mu\text{V}$, different average amplitude $> 100 \mu\text{V}$, or zero variance) or segments (greater than 10 bad channels) were eliminated from subsequent analyses. Segments with eye movements or blinks were corrected using the method described by Gratton & Donchin, 1983. Furthermore, channels containing artifacts on greater than 20% of segments were eliminated and replaced by data interpolated from surrounding electrode sites. Segments were then averaged, re-referenced to the average reference, and baseline corrected for the 200-msec interval prior to the onset of T1.

Chapter 3

RESULTS

3.1 Behavioral Data

The accuracy in discriminating the rotation direction of T1 is shown in Fig. 3.1 and reveals clear evidence for an emotion-induced blink in the Lag2 condition. Accuracy in reporting T2 is much worse when it is preceded by a T1 consisting of a negative or control picture compared to a scene picture. In addition, negative pictures produced a larger blink than their matched controls. All conditions yielded uniformly high accuracy at lag8 consistent with the idea that the blink only extends for a few hundred milliseconds following T1. A 2 (lag2 vs. lag8) x 5 (High Negative, High Negative Control, Moderate Negative, Moderate Negative Control, and. Scene) repeated measures analysis of variance (ANOVA) revealed a significant effects of lag [$F(1, 17) = 150.295, p < .0001$] and T1 type [$F(3, 45) = 20.919, p < .0001$]. There was also a Lag x T1 type interaction [$F(3, 55) = 25.838, p < .0001$]. This interaction was analyzed using separate ANOVAs on the lag2 and lag8 data. A one way ANOVA on the lag8 data revealed that there was no effect of T1 type [$F(3, 55) = .678, p = .581$]. In contrast, analysis of the lag2 data showed a significant effect of T1 Type [$F(3, 52) = 32.040, p < .0001$]. Follow-up comparisons of T1 types revealed all pairs were different from each other except for the high negative control vs. moderate negative condition ($p = .153$).

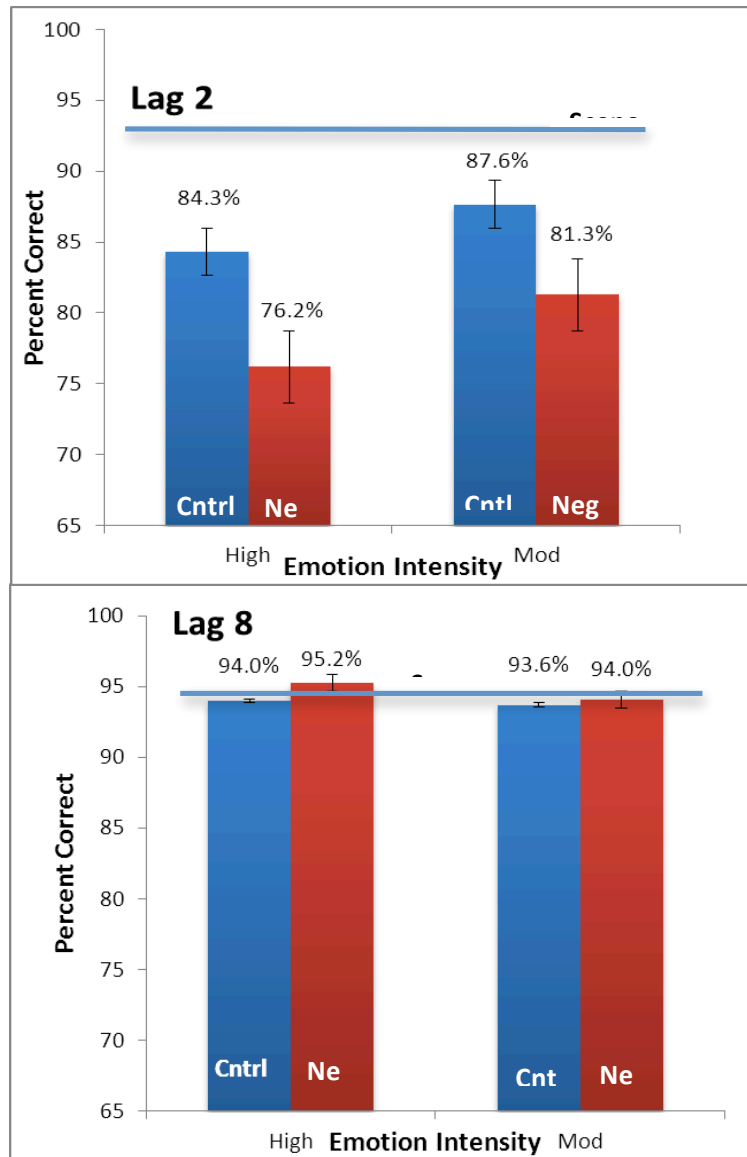


Figure 3.1 The Behavioral results show a significant difference between the emotional pictures and their controls in lag2 condition. There is no difference between any of the conditions in lag8.

3.2 EPN Component

3.2.1 EPN To T1

Figure 3.2 shows ERP waveforms for two conditions: high negative picture lag8 (HN8) and scene picture lag8 (S8 recorded over an occipital location. The S8 ERP slopes upward and has a systematic sinusoidal shape with peaks occurring every 100 msec. The sinusoidal component reflects the periodic appearance of a new picture in the stream at this same rate. The HN8 waveform is similar except for an “extra” negative component occurring approximately 220msec after the appearance of the negative picture. The only difference between these two conditions is the presentation of the negative picture in the HN8 condition. Therefore, the effect of the negative picture on the ERP can be isolated from other activity by computing the HN8-S8 subtraction waveform, which is also shown in the figure. Note that this subtraction waveform does not contain two prominent features that were present in the raw waveforms: the periodic sinusoid and the upward slope. These were common to both conditions (HN8 and S8) and are eliminated by the subtraction. The remainder is the EPN component that is present following an emotional picture and absent for the scene picture. This subtraction approach will be used to isolate the EPN in other conditions as well as to isolate the P300. These waveforms are based on averages of electrodes 95, 90, and 96, which are located over the posterior right hemisphere. These electrodes were chosen because they showed the greatest amplitude for the EPN component.

The EPN component for each T1 condition is shown in Figure 3.3. A repeated measures analysis of variance of these data revealed a main effect of T1 type [$F(2, 39)$, $p=.036$). Post-hoc comparisons showed that the high negative condition was

significantly different from all other conditions (all p 's < .05) except for the high negative control which bordered on significance ($p=.054$).

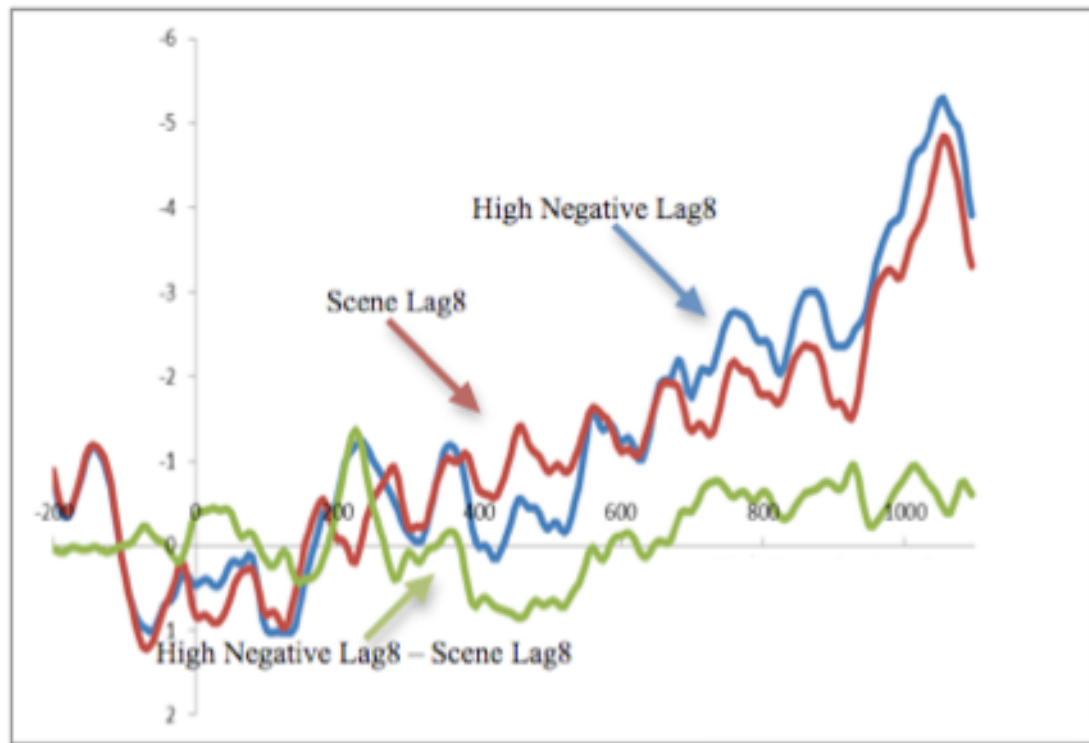


Figure 3.2 The HN8 and the S8 show similar “ramping up” with periodic peaks every 100ms reflecting the occurrence of each picture in the stream. The subtraction curve isolates the EPN component found in the High Negative Condition.

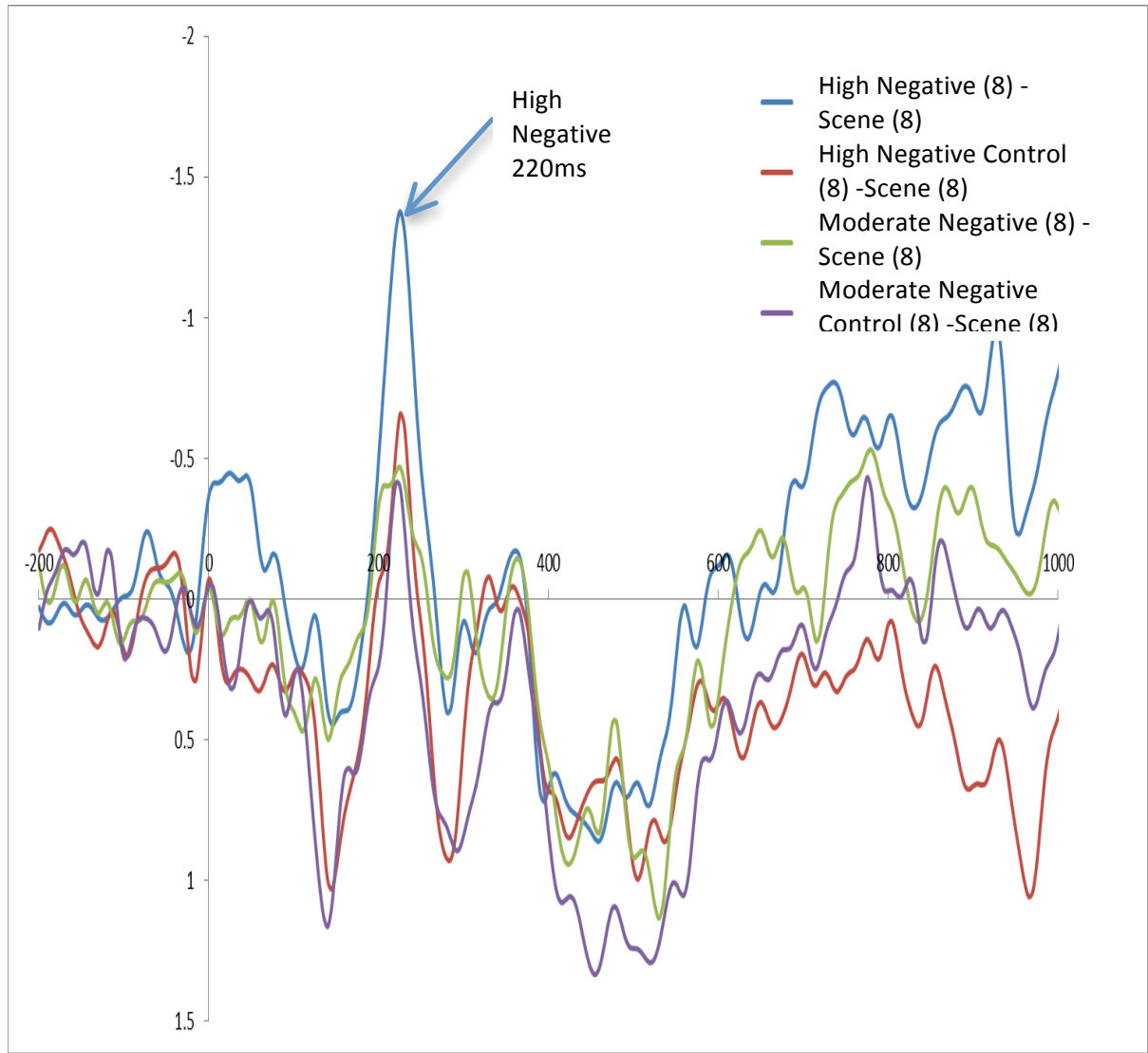


Figure 3.3 EPN components for different conditions. T1 comes on at time 0.

3.2.2 EPN elicited by T2

Figure 3.4 shows the EPN elicited by T2 in the lag2 condition as a function of the type of T1 picture that preceded it. The largest EPN occurs when a scene picture precedes T2. Presentation of an out-of-category T1 reduced the amplitude of the T2 EPN and in the case of a preceding high negative picture; it appears that the T2 EPN is

completely suppressed. A repeated measures one-way ANOVA revealed a significant main effect of T1 type [$F(3, 51)$, $p=.01$]. Post-hoc tests revealed that the high negative condition was significantly different from the high negative control ($p=.011$), the moderate negative control ($p=.006$), and the scene ($p=.004$). The difference between high negative and moderate negative conditions bordered on significance ($p=.056$).

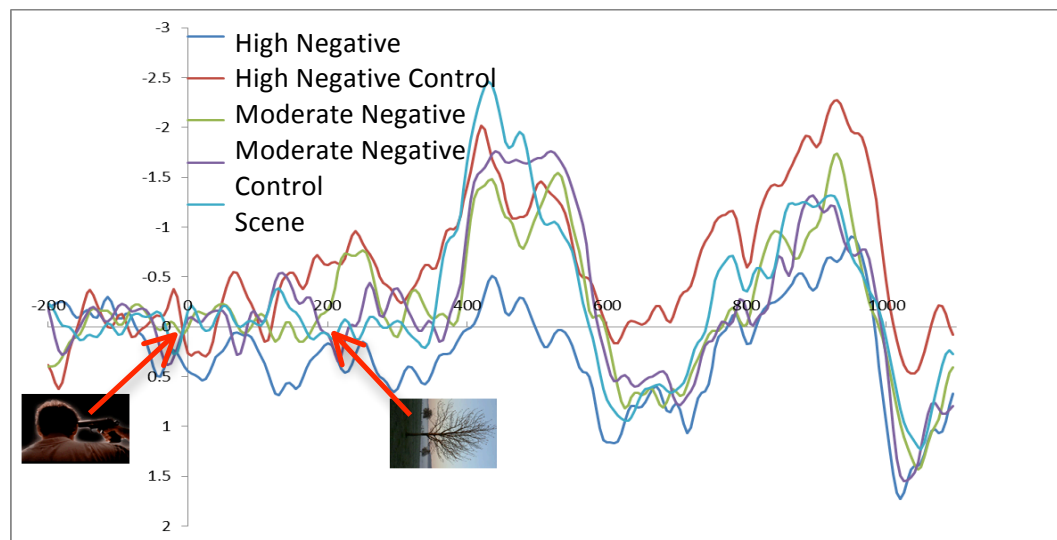


Figure 3.4 The EPN to T2 can be seen in all conditions except the high negative case. It has an onset just before 400ms and a peak at 430ms.

3.3 P300

Figure 3.5 shows waveforms recorded from posterior central sites for the scene condition for lags 2 and 8. These waveforms were recorded from electrodes 37, 53, and 54 since these showed the maximum positivity in the relevant time window. In both cases, there is a prominent positivity peaking at approximately 500msec after the target picture. These components also have similar scalp distributions (Figure 3.6).

This appears to be a P300 (note that P300, despite its name, doesn't always occur 300msec after the eliciting stimulus. It can occur at a variety of latencies depending on the nature of the stimulus and task). Both of these components are expected given what we know about the P300, therefore they give us a good idea of when and where to look for a P300 to T1 if one exists.

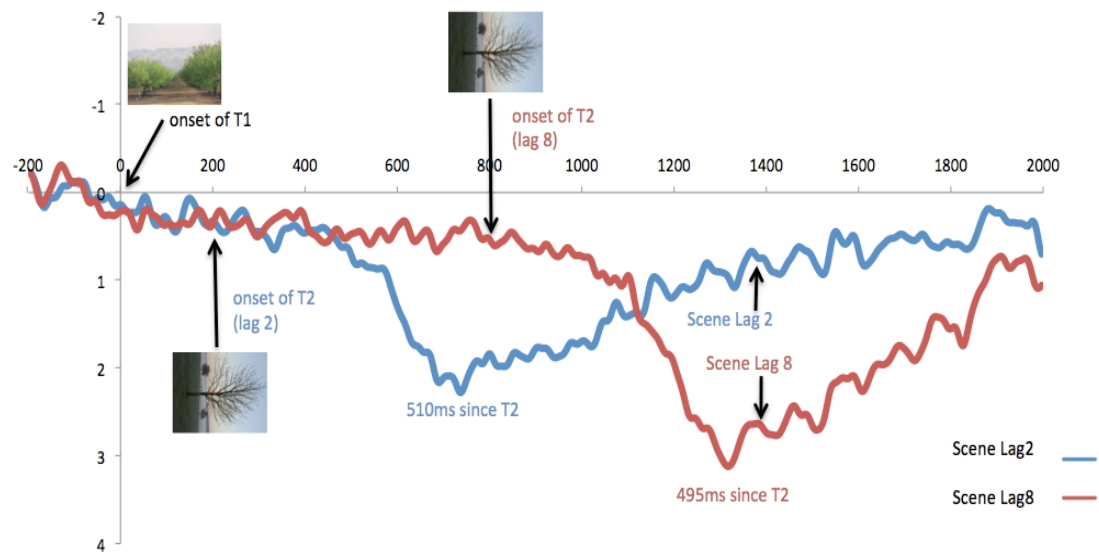


Figure 3.5 P300s elicited by T2 in the lag2 and lag8 condition have similar time courses peaking between 495 and 510ms after the onset of T2.

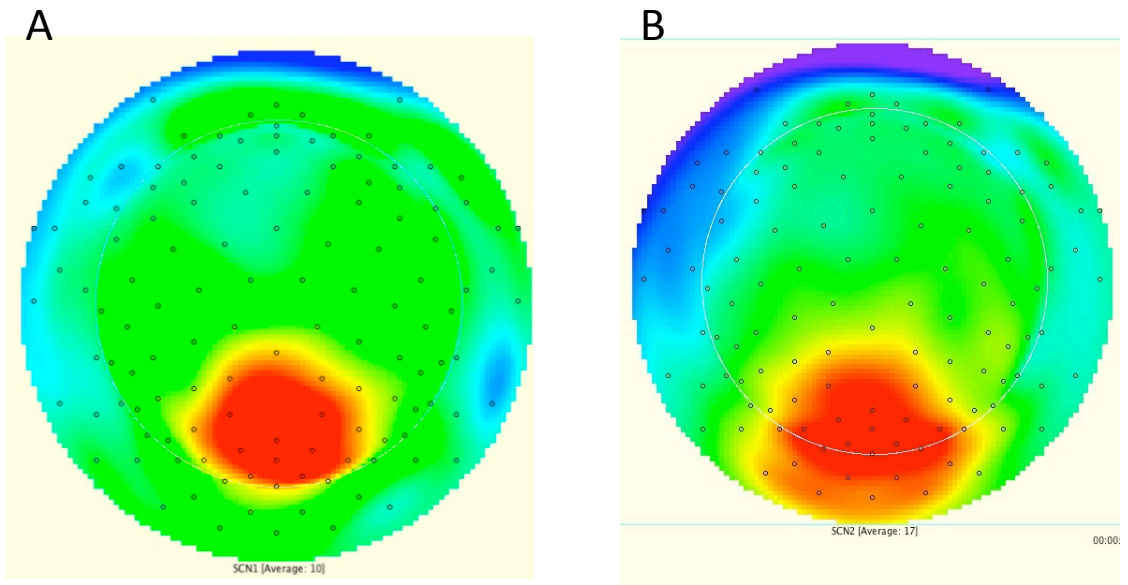


Figure 3.6 A) P300 at 710ms (510ms after T2) in the Scene lag 2 condition. B) P300 at 1295ms (495ms after T2) in the Scene lag8 condition.

Figure 3.7 shows waveforms for the S8 and HN8 conditions as well as the S2 for comparison. Both S8 and HN8 show a P300 corresponding to T2 (peaking at around 1300ms). However, despite the absence of a second target, there seems to be positivity in the HN8 condition that is similar to the positivity that occurred in response to T2 in the S2 condition. A one-sample t-test showed that the P300 for the high negative condition is significantly different than zero, [$t(17)=4.414$; $p<.0001$]. A similar test on the S8 condition in the same interval was not significant [$t(17)=1.919$; $p=.072$].

Given that the P300 peaks at approximately 500msec after T2 we would expect a similar latency for a P300 following T1. Previous research shows that emotional and neutral stimuli elicit a P300 at latency anywhere between 300-500ms following the presentation of the picture (Hajcak, Weinberg, MacNamara & Foti,

2010). In Figure 3.8, there appears to be small difference in positivity in the earlier interval corresponding to T1. To isolate this, I subtracted the S2 waveform from HN2 waveform. A t-test showed that this positivity was not significantly different from zero [$t(17)=.527$; $p=.605$] suggesting that the irrelevant T1 did not elicit a P300.

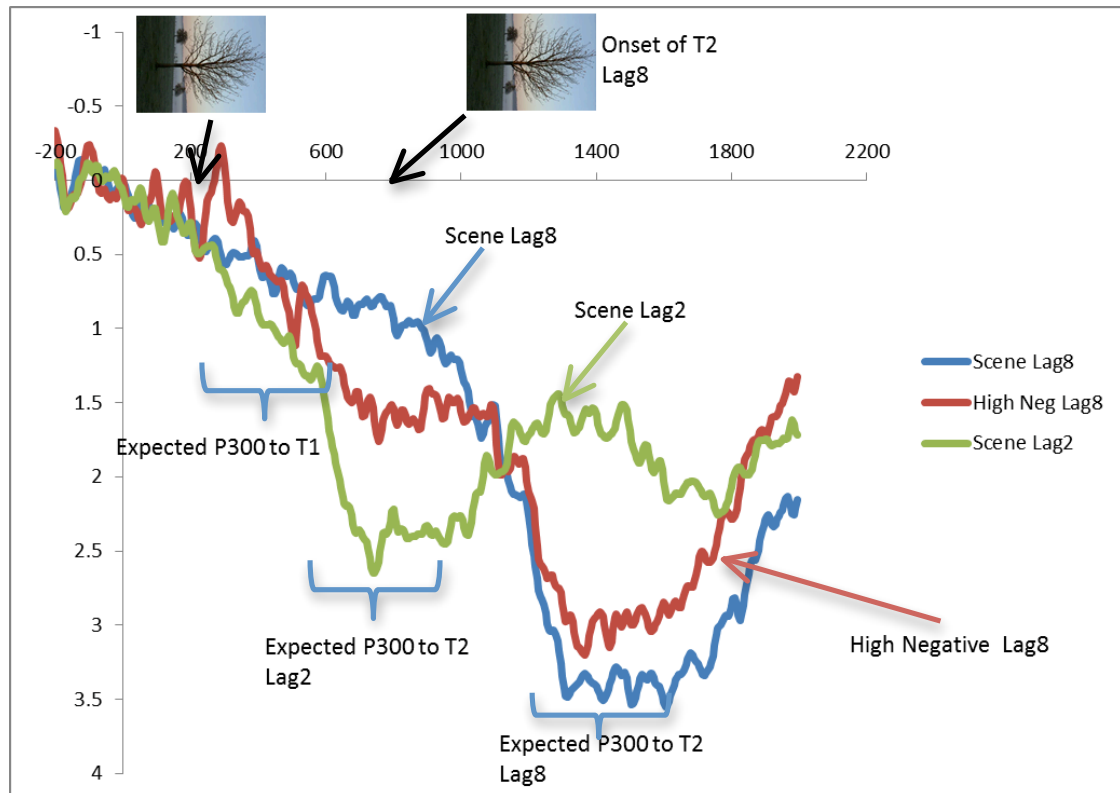


Figure 3.7 The S8, HN8, and S2 show their expected P300s to T2 for the respective lags. Surprisingly we also see a P300 in the HN8 condition during the lag2 interval.

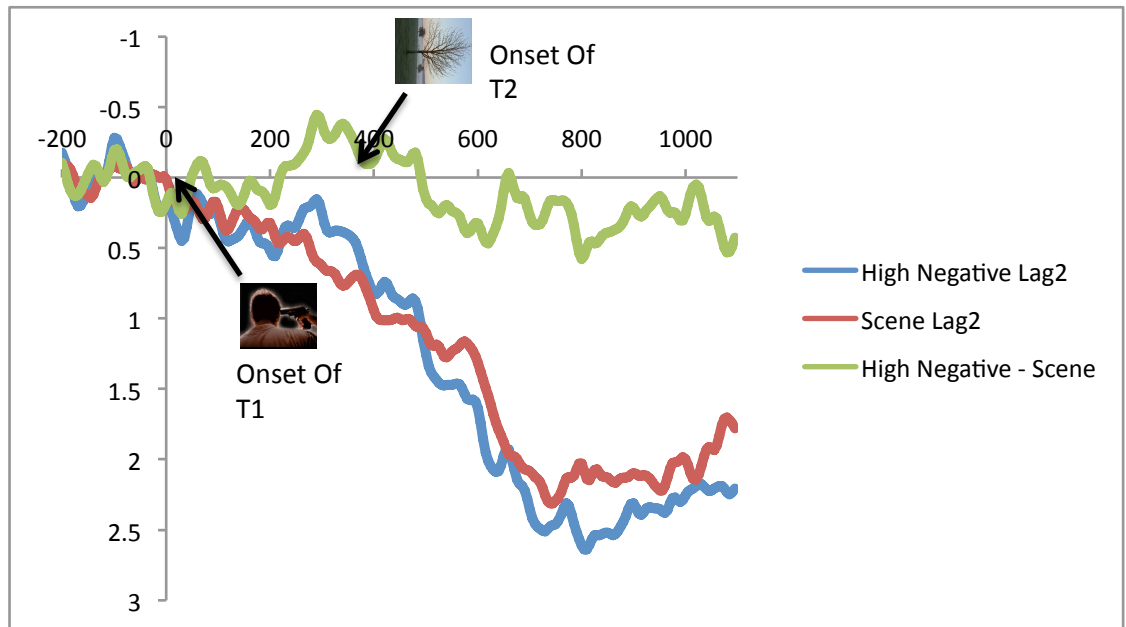


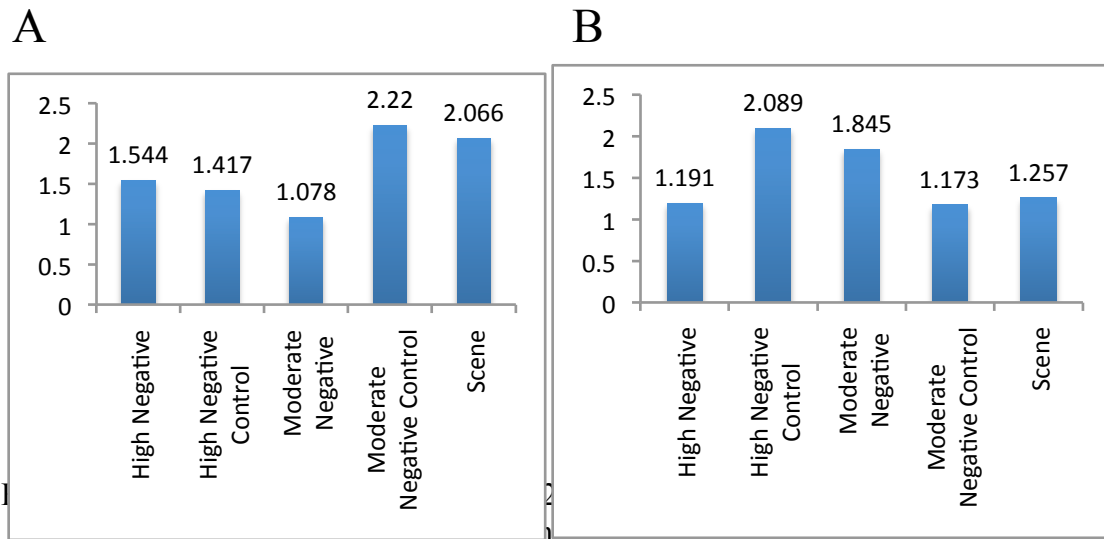
Figure 3.8 Difference curve (HN2-SN2) showing a small positivity in the range of 500-600 msec.

In order to increase our confidence that the irrelevant T1 picture did not elicit a P300, we employed a complimentary technique for measuring ERP components based on *principal components analysis* or PCA. This is a mathematical procedure for objectively discovering and quantifying ERP components (Dien, 2007). I used Dien's (2007) ERP PCA Toolkit to perform a two-stage PCA analysis. The first stage is a temporal PCA, which delivers a series of time windows containing ERP components. The second stage is a spatial PCA, which examines each time window discovered in the first stage to derive a series of spatial factors, which are clusters of correlated sensors that make the largest contributions to the ERP. The PCA revealed a clear P300 component peaking 510msec after T2 with a peak amplitude at electrode 62, which is located over central parietal areas. The latency, waveform shape, and scalp

distribution are all consistent with the conclusion that this factor is the P300 following T2. Also shown is a bar graph of P300 amplitude as a function of T1 type (Figure 3.9). The PCA did not reveal a P300 component in the interval following T1.

A one way ANOVA revealed a significant effect of T1 type on the P300 elicited by T2 [$F(3, 51) = 2.032, p = .026$]. In order to properly assess how much of the positivity in this time frame is associated only with the P300 to T2 we subtracted the lag8 conditions from the lag2. These subtraction curves, which are shown in figure 3.10, reveal similar amplitudes for different T1 types at lag2. This impression was verified by a one way repeated measures ANOVA which showed there were no significant differences due T1 type [$F(2, 39) = 1.023, p = .372$].

There was also a P300 following T2 in the lag8 condition. This P300 had a latency of 495msec relative to T2 onset and peaked at electrode 75, which is near the sensor used to measure this component in the lag2 condition (see above). A one way repeated measures ANOVA of this component revealed no significant differences between different T1 conditions [$F(2, 41) = 1.307, p = .284$].



these amplitudes indicating equivalent P300s for the lag2 conditions. B) Mean amplitude at 495msec following T2 in the lag8 condition over electrode 75. There is again no overall significant difference between these values.

3.4 Left lateralized positivity

The final component I analyzed is a previously unnamed positivity that reaches peak amplitude between 380 and 430ms after the presentation of T1 and is located over left posterior areas (maximum amplitude at sensors 63, 64, and 68). Importantly, this *left lateralized positivity* component may be related to the blink as it is largest for high negative pictures and generally follows the same pattern as the behavioral blink data (Figure 3.10). There was a main effect found using a repeated measures ANOVA [$F(3, 58) = 5.349, p = .002$]. The scene condition, which did not demonstrate this positivity (see Figure 3.11), was significantly different from the high negative ($p = .001$), high negative control ($p = .005$), and moderate negative control conditions

($p=.017$). It was bordering on a significant difference with the moderate negative condition ($p=.064$). The only other conditions to differ significantly were the high negative and the moderate negative ($p=.025$). In order to determine whether this component was related to the blink, I examined its amplitude as a function of whether participants were correct vs. incorrect on reporting the orientation of T1. If this component is related to the mechanism that is responsible for suppressing T2, we would expect that incorrect trials would be associated with a larger amplitude for this component. This comparison is shown in Figure 3.12). A repeated measures one-way ANOVA showed that the left lateralized positivity was significantly higher for correct trials versus incorrect trials [$F(1,17) = 6.103$; $p=.024$].

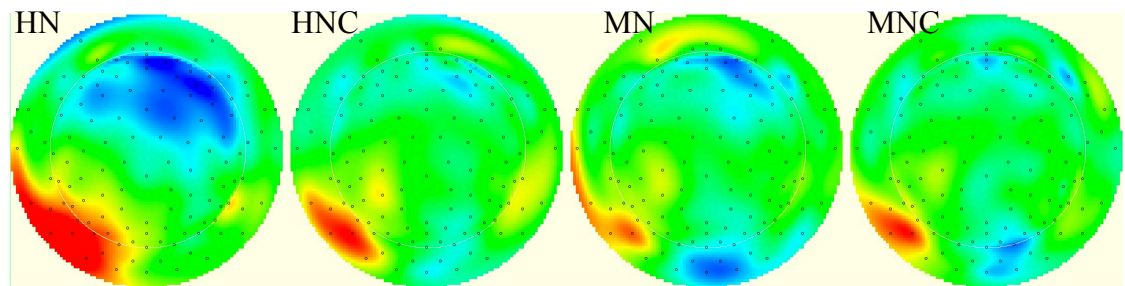


Figure 3.10 Here we can see the left lateralized positivity (400ms) in all four non-scene conditions. It is strongest for the high negative case (far left) but still present in the high negative control, moderate negative, and moderate negative control conditions (from left to right).

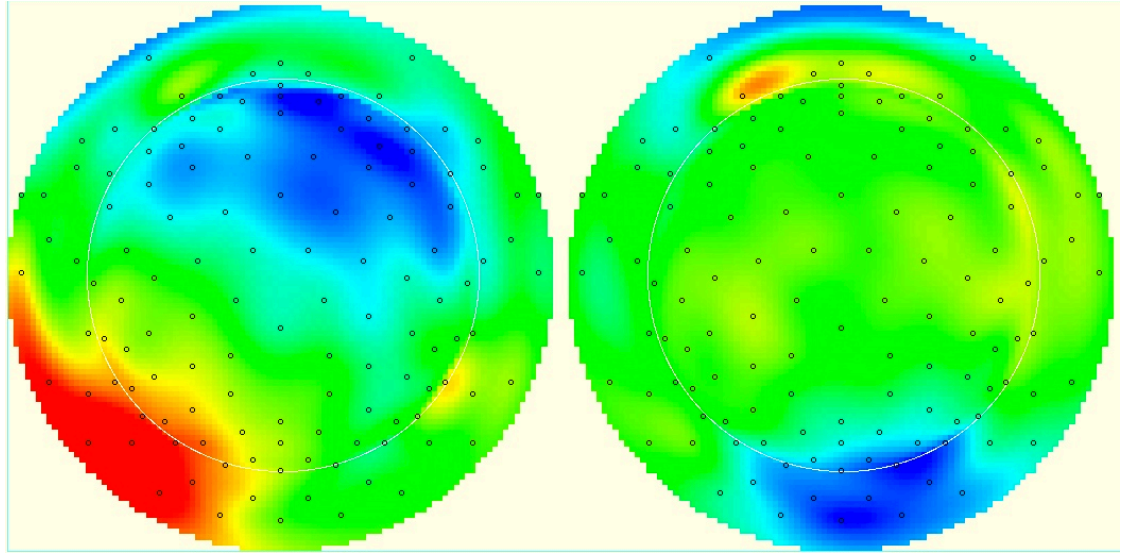


Figure 3.11 The left lateralized positivity is clearly visible in the high negative condition (left) but is absent in the scene condition (right)

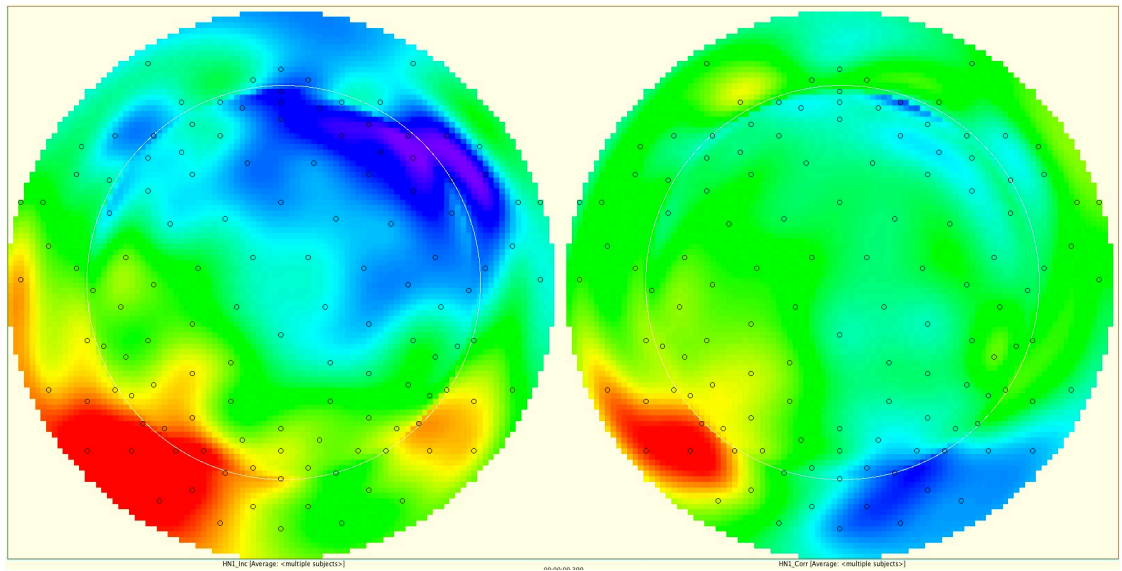


Figure 3.12 The left lateralized positivity component for incorrect (left) and correct (right) trials.

DISCUSSION

3.5 Behavioral

The behavioral evidence provides a striking replication of the emotional induced blindness effect. Participants were severely impaired in reporting a rotated target picture when it was preceded at short intervals by an irrelevant emotional picture. Importantly, this blink was larger for emotional pictures than their control pictures suggesting that the blink is related to the emotional content of the picture and not its physical salience. Furthermore, we found that the degree of emotion portrayed in the picture affects the magnitude of the blink: high negative pictures produced a larger blink than moderate negative pictures.

3.6 P300

3.6.1 Evidence Against Central Interference Theory

The results of our study call into question the central interference theory, which holds that T1 interferes with T2 by occupying working memory and preventing access by T2. We assume that in the EIB paradigm, the irrelevant picture can play the role of T1. That is, even though this picture has no task relevance and should be ignored, it may still capture attention and gain access to working memory (WM), blocking access by the following T2 and resulting in a failure to report T2. If T1 is indeed gaining access to WM, previous research suggests that it should produce a P300. Surprisingly, however, our results do not show any evidence of a P300 following T1 even though it produces a robust blink. This suggests that the blink can occur without T1 accessing working memory. Apparently, emotional pictures can produce a blink through some mechanism other than competition for working

memory. If we want to retain the claim that the AB results from working memory competition, we are forced to conclude that perhaps the AB and EIB are not as closely related as they appear. Although their behavioral similarities are striking, the relationship of the neural mechanisms behind these two paradigms is not so apparent. Perhaps working memory is to blame for the attentional blink and we must search for a different explanation of the EIB.

3.7 EPN

Earlier we proposed that the early posterior negativity (EPN) may reflect the operation of a salience detector and that negative stimuli may be more salient than the neutral stimuli that served as a control in previous EIB research. We evaluated this by using a set of pictures in which each emotional picture was paired with a non-emotional control picture that was matched as closely as possible in terms of physical features. Using these stimuli we still observed a significantly larger EPN for highly negative pictures compared to control pictures. Although the moderate negative, high negative control, and moderate negative control conditions elicited a behavioral blink, their associated EPN components were small and not significantly different from zero. Therefore, the status of the EPN as reflecting a mechanism that is causally related to the blink is uncertain. We may have simply lacked enough power to detect EPNs that are small or it may be that this component isn't causally related to the blink and that we should look to other ERP components, such as the left lateralized positivity, for insights into the mechanisms responsible for the blink. It still may be the case that the EPN reflects salience. Perhaps the high negative images were the only stimulus set that was salient enough to produce a significant EPN.

Top-down processes can also make a stimulus salient, which might be part of the reason that T2 elicits a robust EPN. When subjects are searching for a particular feature that defines a target, such as orientation, this feature could become the basis of increased salience for that picture, resulting in an EPN. In addition, when the observer is set for this feature, the triggering of the salience detector could serve to initiate a cascade of additional processes that result in entry of the salient stimulus into working memory. In this scenario, the emotional picture may suppress low-level perceptual processing of T2, eliminating its ability to trigger the salience detector and resulting in failure of the target to gain access to higher-level processes responsible for conscious awareness and report of T2. This is consistent with the data from the current experiment, which show a complete suppression of the EPN to T2 in those conditions that produce a large blink, i.e., presentation of a highly negative T1 picture at a short lag.

3.8 Left Lateralized Positivity

The left lateralized positivity immediately follows the EPN and may be causally related to the blink. We found that it was larger on trials on which the observer was incorrect at reporting T2 compared to correct trials suggesting that this component may reflect a suppression process that follows the presentation of a salient picture. The blink could be caused by a perceptual suppression of stimuli following T1 rather than competition of higher-level processing. Further research is needed to confirm these suspicions.

3.9 Summary

It is evident that emotional stimuli can involuntarily capture our attention and prevent us from attending to other information in our environment even when it may be relevant to our current goals. This can be demonstrated both inside and outside the laboratory. If you have ever had the experience of flipping aimlessly through the channels on television and inadvertently pausing on a channel featuring a gruesome horror scene you can relate to EIB. Although we may notice these effects in our everyday lives we now have some insight into the neural mechanisms behind this phenomenon. Our finding that T1 produces a blink without producing a P300 suggests that the central interference theory does not offer a good explanation for emotional induced blindness. This is because there is no indication that T1 is being encoded into working memory and therefore there is no reason to believe T1 is blocking access by T2 to these mechanisms. There appear to be two possible ways to reconcile this finding with the prominent role that central interference theory plays in explaining the AB. One possibility is that EIB and AB are produced by different underlying mechanisms, a position that is supported by various behavioral findings reported by Most and colleagues (Most & Wang, 2011; Kennedy & Most, 2011). The second possibility is that this theory is incorrect as an explanation for the AB as well, a position taken by a recent review of the AB literature (Martens & Wyble, 2010) which concludes that the AB may reflect an early perceptual suppression of T2 by T1, a possibility that is compatible with the present results.

Further exploration of the EIB is necessary to clear up many of these issues; however, by using matched stimuli we have confirmed much of what we thought to be true on the basis of the Kennedy et al. study. We have reaffirmed that irrelevant emotional stimuli can interfere with performance on other attention demanding tasks

and this effect appears to be related to the content of the emotional picture and not just its physical salience.

Perhaps we can now apply this laboratory evidence to the real world.

Advertisements may take advantage of the ability of emotional stimuli's attention grabbing ability. However, this could come at a high cost. Imagine the implications if billboards began featuring negative or emotionally arousing scenes. Although, this would attract the attention of passing drivers, it would be at the cost of other tasks. For instance, drivers could be potentially "blinded" to brake lights or other road hazards (Kennedy & Most, 2011).

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