# THE EFFECTS OF EYEBLINKS ON AUDITORY PROCESSING

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**Declaration**

I hereby declare that this thesis is my original work and it has been written by me in its entirety. I have duly acknowledged all the sources of information which have been used in the thesis.

This thesis has also not been submitted for any degree in any university previously.

Ching Shi Min, April

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**Abstract**

The fact that we rarely notice the brief occlusions of vision caused by eyeblinks has been linked to an active suppression of visual processing in primary visual cortex. The present study sought to determine whether this suppression is a unimodal or cross-modal phenomenon. To this end, participants completed an active auditory deviant detection task using simple tones. Deviants were slightly louder as compared to standards. For data analysis purposes, trials were classified into blink and no-blink trials depending on whether a blink occurred within 150ms before or after sound onset. Participants were less likely to detect auditory deviants on blink as compared to no-blink trials. Moreover, on blink trials, participants were less likely to detect an auditory deviant the closer their blink's apex was to sound onset. In the event-related potential (ERP), eyeblinks were associated with a decreased central-posterior N100 amplitude for both detected and missed deviants and an increased anterior N100 and P300 amplitude for detected deviants only. Together, these results suggest that eyeblinks cause cross-modal perceptual suppression and point to a compensatory amplification mechanism that may operate before and/or after a blink's maximum.

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## Introduction

Vision appears deceptively stable. Visual change always seems fluid and continuous despite incessant interruptions from natural eye movements. Each blink, for example, introduces a blackout of about about 100-150 ms, 10 to 15 times a minute (Stern, Walrath, & Goldstein, 1984). Yet we usually fail to notice these mini-blackouts. This phenomenon has been linked to a blink-mediated suppression of vision.

Aside from its existence however, little else is known regarding blink suppression. For example, it is unclear whether its effects are confined to vision or involve other modalities and mental processes. To address this question, the present study used electroencephelography (EEG) to detect the influence of spontaneous blinks on neural activity during a difficult auditory detection task. To explain the rationale behind the chosen experimental design, an outline of the current blink suppression literature will first be presented and discussed. Following that will be a revisiting of the experimental design, a review of the EEG markers of interest, and finally a description of the hypotheses.

### The Visual Effects of Blinks

Blinking is the rapid closing and opening of the eyelid which serves to lubricate the exposed eyeball and expel foreign material. Blinking behaviour displays large variance and is sensitive to both internal and external states (Stern et al., 1984). Researchers distinguish between three types of blinks - voluntary blinks which are elicited purposefully, reflex blinks which are involuntary responses to disruptive physical phenomena (e.g., a puff of air, dirt), and spontaneous or endogenous blinks which occur naturally without an eliciting stimulus. Each blink type shows differences in duration, time course, eyelid velocity and amplitude of closure (VanderWerf, Brassinga, Reits, Aramideh, & Ongerboer De Visser, 2003) yet visual suppression of similar

magnitude and time course has been observed in all three cases (Manning, Riggs, & Frost, 1983; Manning, Riggs, & Komenda, 1983; Volkmann, 1986). It is thought that this suppression during eye movements evolved to reduce disruption from self-elicited, necessary and harmless bodily motions (Volkmann, 1986; Riggs, Volkmann, & Moore, 1981).

The phenomenon of visual suppression refers to the fact that eyeblinks are often unperceived or perceived to be of lower intensity and shorter duration than they actually are. Riggs, Volkmann and Moore (1981) demonstrated this phenomenon using a Ganzfeld experiment. Participants' heads were enclosed inside hollow aluminium spheres, creating a homogenous and featureless visual field (thus "Ganzfeld" or whole-field). The participants viewed changes to Ganzfeld illuminance with eyes open and compared the visual effect to that of voluntary blinks. Ganzfeld darkening which was equivalent in intensity and duration to that of eyeblinks was judged to be visually stronger than an eyeblink, and the two became subjectively equal when the Ganzfeld was darkened with lower intensity and shorter duration. In other words, the visual suppression during eyeblinks causes an incomplete perception of the blink-associated blackout. This finding has been replicated in a number of studies (Riggs, White, Manning, & Kelly, 1984; Volkmann, Riggs, & Moore, 1980).

The source of blink suppression was for a while an issue of contention. Although some researchers considered it a purely optical phenomenon, a number of studies pointed instead to a neural cause. Specifically, a pioneer study by Volkmann, Riggs and Moore (Volkmann et al., 1980) demonstrated lowered visual acuity during blinks while controlling for the visual effects of eyelid closure. This control was achieved using the following technique. Participants wore opaque goggles while their retinae were stimulated with a fibre-optic light source in the mouth which projected light through the palatine bone (the roof of the mouth), thus creating visual stimuli which could circumvent the usual pupillary pathway to the retina and not be physically impeded by eyelid closure. When asked to pick the dimmer of two trans-palatine illumination events, participants were less sensitive to luminance changes and performed poorer when these

events coincided with a voluntary blink onset. Performance decrements were evident about 150 ms before blink onset and reached a maximum 30-40ms before the upper eyelid began to cover the pupil. Performance recovered gradually only 100-200ms after blink onset.

Since optical effects due to eyelid movement were controlled for, the lowered sensitivity to trans-palatine illuminance was unlikely to be due to visual masking. It was also unlikely that the lowered sensitivity could be explained by other blink-related eye movements (i.e. the involuntary downward deflection of the eyeball of about 1-5° during each eyeblink (Collewijn, Van Der Steen, & Steinman, 1985). Thus the experimenters concluded that the locus of blink-mediated suppression could not be retinal and was therefore neural. Besides luminance, other studies have demonstrated lowered sensitivity to changes in contrast (Ridder III & Tomlinson, 1993), spatial position (O'Regan, Deubel, Clark, & Rensink, 2000) and 2-D contour (Johns, Crowley, Chapman, Tucker, & Hocking, 2009) , as well as poorer detection of new visual objects (Wibbenmeyer, Stern, & Chen, 1983).

Taken together, this research established visual suppression during eyeblinks while controlling for optical effects from afferent sources, and ruled out visual masking or other blink-related eye movements as the sole mediator of suppression . Research on other types of passive eye movements such as saccades and vergences has also demonstrated visual suppression which cannot be attributed to optical effects (Volkmann, 1986). Furthermore the pre-blink onset of the suppression places its determinant not at the action of extraocular muscles but upstream at central processing - this favours a "feed-forward" theory of blink suppression where the blink command simultaneously triggers suppression-related neural processes (see Volkmann, 1986).

**The Cognitive Effects of Blinks**

Given its impact on visual processing, it is not surprising that spontaneous blinking is influenced by visual related mental activities. Intuitively, we know that blinking is inhibited when

carrying out a visual task to avoid missing important stimuli. In line with this intuition, empirical research has shown that endogenous blink rate decreases for tasks requiring visual attention (e.g., reading) relative to tasks involving non-visual activities (e.g., conversation, listening to a passage) (Bentivoglio et al., 1997; Doughty, 2001; Karson et al., 1981). However, blink rate is not merely a function of stimulus modality, but of cognition and task demands. Studies manipulating cognitive load by increasing the number of concurrent tasks (Fournier, Wilson, & Swain, 1999) or enlarging set size in a digit sorting task (Siegle, Ichikawa, & Steinhauer, 2008) or digit memorisation task (Holland & Tarlow, 1975) found that blinking rate declined with increased task difficulty. Moreover, this decline has been demonstrated using tasks requiring little visual feedback such as mental arithmetic (Holland & Tarlow, 1975) as well as auditory duration discrimination (Bauer, Strock, Goldstein, Stern, & Walrath, 1987; Goldstein, Walrath, Stern, & Strock, 1985). Blinking is also sensitive to task dynamics: during a continuous task blinks are deferred to less intensive periods such as immediately after task completion or between trials (Fogarty & Stern, 1989; Leal & Vrij, 2008; Orchard & Stern, 1991; Siegle et al., 2008) or when ensuing stimuli are known to be task-irrelevant (Pivik & Dykman, 2004). Blink rate is not only lowered to task relevant stimuli, but also stimuli possessing social or affective relevance (Nakano, Yamamoto, Kitajo, Takahashi, & Kitazawa, 2009; Schirmer, n.d.; Shultz, Klin, & Jones, 2011). Finally, there is evidence that blinking varies as a function of time on task. Blinking rate increases the longer participants engage in a task (Stern et al., 1984) and this is thought to reflect the waning of arousal and attention levels. In summary, visual as well as cognitive demands influence blinking behaviour. Blinking appears to be withheld while stimuli are being encoded and its frequency follows fluctuations in cognitive load.

The above studies observed changes in blink behaviour during various tasks, suggesting that changes in cognitive activity can affect the rate of blinking. But has the reverse relationship - that the occurrence of eyeblinks themselves directly correlate with suppressed cognitive activity - been observed before ? There is significantly less exploration into the effects of blinking on

cognition, which could be verified by monitoring task performance during blinks. So far only three studies have been found to adhere to that description.

In the first example, O'Regan, Deubel, Clark, and Rensink (2000) used a change-detection procedure to study the effect of blinks. Participants viewed pictures on a computer screen which changed in some manner (e.g., changes in colour or position of existing objects, new objects appearing) during a blink. They were instructed to look for changes and were not told that the visual changes occurred during blinks. Changes were generally difficult to detect. The probability of change detection increased when the eye was closer to the change location, but this probability was only 40% even when the change location was directly fixated. The results suggest that during a blink only the global aspects of the stimulus are attended to and details are ignored. Thus visual changes escape attention, which preserves the appearance of continuity across the blink-mediated blackout.

In the second study, Thomas and Irwin (2006) tested for the effects of voluntary blinks on performance in a partial-report task (Sperling, 1963). In this task, participants were very briefly presented (106 ms duration) with a 3x3 array of letters and on some trials executed a blink on seeing the array. They were then cued by a high, medium or low pitched tone to report the top, middle or bottom row of letters respectively. Only at the shortest delay between array presentation and retrieval cue (50 ms), participants made more errors during blink than no-blink trials. These errors were mislocation errors, which involved reporting letters from the other two non-target rows instead of the correct letters.This suggested that blinks interfered specifically with the binding of item identity and item position in iconic memory. Additional control experiments indicated that visual masking and irrelevant motor responses associated with eyeblinks cannot fully explain these effects. Instead, they linked the observed binding suppression to saccade-like movements of the eyeball (Irwin & Thomas, 2010).

Lastly, there is an fMRI study that explored the effects of blinks outside the visual system (Bristow, Frith, & Rees, 2005; Bristow, Haynes, Sylvester, Frith, & Rees, 2005). This study used

a similar illumination technique as Volkmann and colleagues (1980). The authors compared the BOLD signals associated with trans-palatine vision in blocks with voluntary blinking (participants were instructed to blink at a fast regular rate) against blocks with natural blinking (participants were told to blink normally) and found a decrease in the retinotopic V3 area of the visual cortex in the former as compared to the latter condition. In line with prior behavioural work this was interpreted as lowered sensitivity to visual stimulation during blink suppression. Notably, the authors also found decreases in prefrontal and parietal areas - structures linked to consciousness and decision making (Beck, Rees, Frith, & Lavie, 2001), thus suggesting that blink suppression is not merely a sensory alteration but affects processes apart from vision..

**Rationale**

Together, the three experiments described above suggest that blinks affect processes beyond simple visual sensation. Nevertheless, because they used visual stimuli it is difficult to dissociate visual from non-visual or more general cognitive effects. It is unclear whether blinks affect such general cognitive processes directly or indirectly through a deteriorated visual percept. Furthermore, there is at present no study that explored a potential impact of blinking on the sensory and cognitive representations of non-visual stimuli. Such an impact might be expected if blinking suppresses more general cognitive processes. Additionally, it would help ensure synchrony between the different senses and multisensory integration. If blinking only suppressed vision while the other senses continued to register information, visual suppression could cause a disconnect between the senses and impair the holistic perception of environmental events. Cross-modal suppression would ameliorate this issue. Thus, the present study sought to determine whether blink suppression was a unimodal or multimodal phenomenon by concentrating on the effect of blinks on the processing of sounds.

Another shortcoming of the blink suppression literature is that most studies (but see Bristow, Frith, & Rees, 2005; Bristow et al., 2005) relied on behavioural measures, or regarded blink rate itself as a dependent measure. Thus, the perceptual consequences of blink suppression are well documented but its neural mechanisms remain unknown. Here, event-related potentials (ERP) provide an alternative and promising approach to the study of blink suppression. Their high temporal resolution enables us to explore mental processes as they unfold in time and may thus shed light on the processing stages at which blink suppression takes place. This was specifically useful to the present research objective which was to determine what modalities and processing stages are affected by blink suppression. Nevertheless, the application of ERPs to the present objective also has a potential shortcoming. Specifically, the voltage changes caused by blinking are substantial and may contaminate ERP markers for ongoing mental or cognitive processes (e.g. Hoffmann & Falkenstein, 2008). However, the development of advanced techniques for blink artefact removal make blink contamination a lesser concern. Component-based techniques can decompose spatially distinct sources for the ongoing EEG. Blink related components are then identified based on their time-course and scalp distribution, and removed from the signal. The EEG is then reconstructed without these components (see methods) and thus reflects mental activity fairly independently from concurrent eye movements.

For our purposes, EEG concurrent with an auditory detection task was employed. The EEG records electrical potentials from electrodes on the surface of the scalp. The ERP is the averaged EEG signal time-locked to events of interest, such as stimuli presentation or motor response. Deflections in the ERP may provide information regarding mechanisms that subserve stimulus processing and response preparation. ERP research on audition has created many classic experimental procedures, and the ERP changes associated with these procedures have also been extensively documented. One such classic procedure - the auditory oddball paradigm - was selected for the present purpose.

In the auditory oddball task participants listen to a stream of sounds composed of rare “deviant” stimuli and common "standard" stimuli. Two ERP components have been identified to reflect processing differences in deviants and standards. As these two components were also of interest in the present study, they are described in more detail below.

**The N100 and Underlying Processes**

The auditory N100 wave (Näätänen & Picton, 1987 for a review) is a negative ERP deflection peaking approximately 80-110 ms after the onset of auditory stimuli with a vertex-centred distribution. The N100 reflects neural recruitment for the processing of acoustic events. It can be elicited by the onset of a sound after silence, the offset of a sound of long duration, or an increment in intensity or pitch of an ongoing sound. Thus, it is typically enlarged for deviants as compared to standards in an auditory oddball paradigm regardless of whether participants attend or ignore the stimuli (Butler, 1968; Näätänen & Picton, 1987).

The N100 has been linked to three main generators (Näätänen & Picton, 1987) located in the bilateral supratemporal plane (the primary auditory cortex) (Hari, Aittoniemi, Järvinen, Katila, & Varpula, 1980; Liegeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994; Vaughan, Ritter, & others, 1970, also see Woods, 1995 for a further breakdown of the supratemporal component), the superior temporal gyrus (the auditory association areas) (Celesia, Broughton, Rasmussen, & Branch, 1968; Scherg & Von Cramon, 1986), and the frontal cortex (Alcaini, Giard, Echallier, & Pernier, 1994; Giard et al., 1994; Halgren et al., 1995).

Although the N100 potential is an aggregate of activity from several neural generators, there are methods available to isolate the supratemporal subcomponent from the other subcomponents (Näätänen & Winkler, 1999) - a boon to those wishing to study this subcomponent as an index of primary auditory cortex activity. One of these methods involves recording scalp potentials and potentials at the mastoids against a common reference at the nose

(electrical potentials are measured as the potential difference between an electrode and the reference). The supratemporal subcomponent is the only subcomponent of the N100 which reverses polarity across the sylvian fissure (Vaughan et al., 1970) and thus will be negative at the scalp and positive over the mastoids. Another method uses the magnetoencephalographic (MEG) equivalent of the N100, the N100m, which can be measured by scalp sensors placed at temporal regions These sensors are primarily sensitive to the supratemporal aspect of the N100. Together with the EEG mastoid method, MEG approaches have advanced our understanding of the supratemporal N100 (Hari, 1990 for a review) . Consequently, the supratemporal subcomponent is by far the best studied N100 subcomponent.

Näätänen and colleagues (Näätänen, Kujala, & Winkler, 2011; Näätänen & Winkler, 1999) have theorised extensively that the neural elements underlying the supratemporal N100 subcomponent are responsible for maintaining auditory feature traces - fragmented stimulus information that has yet to be integrated into the representational system. N100 characteristics as seen across various paradigms together support this view: (1) A correlation of supratemporal N100 amplitude, latency and/or scalp topography with physical acoustic features such as loudness (Picton, Woods, Baribeau-Braun, & Healey, 1976), pitch (Verkindt, Bertrand, Perrin, Echallier, & Pernier, 1995) and locus of origin (Masterton, 1992) implicate feature specific networks contributing some portion of the N100. (2) The N100 amplitude correlates with stimuli detection but not discrimination or recognition (Parasuraman, Richer, & Beatty, 1982). Inferring from this, the N100 does not correspond to the complete stimulus representation (i.e. a copy of the subjective experience of the stimulus) but just fragmented feature information which is apparently not available to voluntary discrimination. (3) The attenuation of supratemporal N100 amplitude to repeated presentations of a sound (Sable, Low, Maclin, Fabiani, & Gratton, 2004) suggests refractoriness in subserving feature-detector neurons. The feature-detector neurons express lowered responsiveness with frequent stimulation. (4) Finally, at least several seconds are needed for the supratemporal N100 to recover from stimulus-specific attenuation. For example,

participants listening to a sequence of tones at rates as slow as 11-15s still elicited lowered N100 to tones identical to the preceding one as compared to dissimilar tones (Cowan, Winkler, Teder, & Näätänen, 1993). Taken together, the subcomponent demonstrates two qualities Näätänen and Winkler (1999) state as necessary to for it to represent feature traces: feature specificity and durability.

Although the above suggests the supratemporal N100 to be fairly exogenous and stimulus specific, investigations exploring the N100 as a whole imply a significant degree of stimulus non-specific excitability (i.e. it can be elicited by any type of sound) and top-down modulation. For example, the N100 may be enhanced under conditions of highly focused attention. When inspecting the stimuli presented to only one ear for deviants and ignoring those presented to the other ear, the N100 to sounds at the attended ear is greater than that to sounds at the ignored ear (Hillyard, Hink, Schwent, & Picton, 1973). Arousal may also be a factor; one study found greater N100 amplitude to task-irrelevant sound stimuli delivered while doing mental arithmetic as compared to periods of relaxation (Eason & Dudley, 1971).

These effects are presumably carried out by stimulus non-specific neural populations linked primarily to the frontal cortex. This is supported by studies using a very long interstimulus interval, in which N100 increase to sound onset was found electrically at the vertex but not magnetically at the midpoint between mastoid and vertex (Hari et al., 1980). Näätänen and colleagues also suggest that the stimulus non-specific neuronal populations may compose a transient-detector system: a mechanism which triggers conscious attention when the strength of certain feature traces exceeds threshold (Näätänen, Kujala, & Winkler, 2011).

**The P300 and Underlying Processes**

The P300 (Polich, 2007 for a review) is typically studied using an active "oddball" paradigm, in which participants intentionally inspect the oddball stream for deviants (Pritchard,

1981). It was first characterised as a central-parietal positivity occurring about 250-500ms after the onset of deviants.

The component is regarded as an endogenous component because it is insensitive to the physical characteristics of stimuli. Instead it is influenced largely by the subjective experience of stimuli, their task relevance and associated task performance.. For example, P300 amplitude to target stimuli can be modulated by task difficulty (Kok, 2001), target frequency within the presentation stream (Duncan-Johnson & Donchin, 1977, 1982; Squires, Petuchowski, Wickens, & Donchin, 1977), target-to-target interval (Gonsalvez, Barry, Rushby, & Polich, 2007), familiarity arising from previous presentations (Curran, 2004; Rugg & Doyle, 1992), state arousal (Kok, 1990) and "arousability" due to personality traits (Justus, Finn, & Steinmetz, 2006; Mardaga & Hansenne, 2009; Stenberg, 1992), among others. P300 latency correlates with task reaction time (Kutas, McCarthy, & Donchin, 1977) and thus has been proposed to be an indicator for task difficulty (McCarthy & Donchin, 1981) and participant ability (e.g., Troche, Houlihan, Stelmack, & Rammsayer, 2009).

At present, researchers often discuss the P300 as an aggregate of two subcomponents - the P3a and the P3b - each with distinct scalp distribution, latency and associated function (Polich, 2007). A three-stimulus version of the oddball paradigm is able to distinguish the P3a from the P3b (Snyder & Hillyard, 1976). In this version, a task-irrelevant distracter deviant is included in addition to the target deviants and standards in the presentation stream. The distracter elicits a P3a while the target deviant elicits both a P3a and a P3b. The P3a has an earlier latency, a central maximum, and its behaviour can be simply described as "novelty detector". It is linked to the involuntary orienting to changes in the environment. The P3b has a parietal maximum and is elicited only to task relevant deviants that are associated with a cognitive or motor response. The P3b is also sensitive to task demand. Its amplitude decreases and latency increases with increasing task difficulty (i.e. the participant is to discriminate between very similar oddball and standard stimuli).

Unlike the N100 generators, the P300 generators are not precisely known. Findings from lesion studies have delineated frontal areas and the hippocampus for the P3a subcomponent specifically (R. T. Knight, 1996), the temporo-parietal junctions for the P300 in general (Robert T. Knight, Scabini, Woods, & Clayworth, 1989; Verleger, Heide, Butt, & Kömpf, 1994). However, given a greater cognitive modulation of the P300 as compared to the N100, it is not surprising that there are other suspected generators possibly widely distributed across the brain.

The function of the P300 has been related to context-updating (Polich, 2007). Specifically, the changes in P300 amplitude and/or latency induced by differences in stimulus attributes are thought to reflect the updating of working memory representations. Presumably, the P3a indexes processes of focal attention in the frontal lobe, if a certain threshold is crossed, and activates the P3b, which indexes memory formation and context updating in parietal and temporal regions. The updated information is then available to inform behavioural responses and ongoing mental processes.

**Hypothesis**

The current thesis aimed to determine the effect of endogenous blinks on the processing of auditory information, thereby determining whether blink suppression is unimodal or multimodal. ERPs were recorded while participants carried out a two-stimulus oddball task in which they detected deviant sounds that were slightly louder than standards. We predicted lowered detection rates when endogenous blinks occurred near the onset of deviants as compared to when no blinks were present within the same time window. For analysis, ERPs were classified into blink and no-blink misses (deviant was not detected), blink and no-blink hits (deviant was detected) and blink and no-blink standards. For missed deviant ERPs, we predicted differences between blink an no-blink trials. N100 amplitude and of P300 amplitude were expected to be smaller for the former as compared to the latter. For detected deviant ERPs, we predicted no or

little blink-associated changes because we expected weaker suppression effect upon successfully detected stimuli. Specifically, we assumed that blink hits might differ from blink misses in that blinks were differently distributed (i.e., further away from the sound onset) and in that they were otherwise less effective in causing blink suppression. Similarly, little or no blink modulation was expected for standards. Due to habituation, the N100 and P300 for these events should already be reduced. A decreased N100 amplitude for blink as compared to no-blink misses would point to a suppression of early auditory processing, whereas a decreased P300 amplitude would suggest suppression at the level of conscious processing and stimulus classification. However, based on reports from past research, a decreased N100 amplitude seems more probable than a decreased P300 amplitude since the suppression effects have been described as largely perceptual.

# Methods

## Participants

Thirty-five undergraduates participated in this study. The data from 20 participants was excluded from data analysis because their EEG recording was artifactual (N = 5), their task performance was very poor (i.e. the visual change detection task described in the oddball task subsection; N = 1), or there were not enough blink trials for analysis due to a naturally low blink rate (N = 14).

The 15 participants included in data analysis (7 females; mean age = 22.7, *SD* = 1.84)reported normal hearing and normal or corrected-to-normal vision. The Edinburgh Handedness Inventory (Oldfield, 1971) was administered to determine handedness (13 right-handed participants, 2 ambidextrous). They gave informed consent after the experimenter explained the experimental procedures. After the experiment, all participants received a monetary compensation for their time (S$10 an hour) and were debriefed about the experiment background and hypothesis.

## Procedure

All participants carried out a listening threshold test followed by an auditory oddball detection task. The listening test served to identify the sound intensity for deviant sounds used in the subsequent auditory oddball task. It determined the participants' ability to detect a threshold-level sound under simultaneous masking conditions. All sound stimuli were presented binaurally over in-ear headphones (Etymotic Research ER-4P) using a Sound Blaster SB X-Fi audio card (44100 Hz, 16 bit).

***Hearing Threshold Test.*** Hearing thresholds were determined using an adaptive, three-interval, three-alternative, forced-choice procedure (adapted from Gatehouse & Davis, 1992). A test trial consisted of three 800 ms long observation intervals. At the beginning of each interval, a 300 ms long sound was played and a number was shown on the computer screen indicating which interval was currently being presented (i.e. "1" for the first interval, "2" for the second, etc.). Five ms ramps were applied to the onset and offset of all sounds. The sound stimuli were as follows:

- a 1000 Hz sinusoid tone with a duration of 50 ms ("probe") and
- a 1000 Hz sinusoid tone with a duration of 300 ms ("carrier") .

One random interval contained the carrier and probe tones with simultaneous onset, while the remaining two intervals contained the carrier only. After the three intervals, participants were prompted to indicate which interval contained the probe. Responses were made via a button box, with one of three keys each corresponding to the first, second and third interval. Once a response was made, participants were given feedback via the computer display and prompted to initiate the next trial by pressing a button.

For all participants, the carrier tones in both types of intervals were always presented at the same, clearly audible intensity level (72% of maximum sound volume), while probe intensity was altered according to the participants' prior performance. Probe intensity was determined following a transformed staircase algorithm (Levitt, 1971) - 3-down-1-up - to determine the stimulus level corresponding to 79.4% correct responses. This stimulus level was chosen because it was estimated to yield a sufficient number of both detected and missed deviants in the subsequent oddball task. The initial trial presented a probe amplitude that was loud enough for it to be detected easily amongst the three intervals (88% of maximum sound volume). An incorrect response increased probe intensity on the next trial while three correct responses in a row decreased it, otherwise probe intensity remained unchanged.

An intensity reversal occurred when the adaptive track changed direction and an increment in probe intensity was followed by a decrement, or vice versa. Probe intensity was altered in discrete steps: during the first two intensity reversals the initial step size was 5% of the maximum sound volume, for the next ten intensity reversals step size was reduced to 2%. The threshold measurement was taken as the mean of the last eight intensity reversals.

Each participant first carried out a practice session of five trials at a fixed, easily detected probe intensity, followed by three repeats of the adaptive procedure yielding three threshold measurements. The three threshold measurements were used to calculate an average threshold. In the event that the standard deviation of the three threshold measurements was greater than 15% of the maximum sound volume, the value most different from the other two was discarded and only the two remaining values were entered into the mean. The average threshold was used for the deviants in the following oddball detection task (mean of average thresholds across participants = 76.3% of maximum signal level, *SD* = 1.18%).

***Oddball Detection.*** The carrier tones served as standards, whereas the simultaneous presentation of a carrier tone together with a probe served as deviants. Standards and deviants were presented at a stimulus-onset-asynchrony of 1000 ms. A fixation cross was presented onscreen during the entire oddball detection task. This cross was white except for a few one second epochs (7%) during which the cross turned red. These exceptions only overlapped with standards, never with deviants. Participants were asked to press a button any time the encountered an auditory or visual change. The purpose of including a visual change detection task was to prevent participants from performing the auditory change detection task with their eyes closed.

In summary, participants were presented with a total of 2900 sound stimuli split across seven blocks: 406 were deviants accompanied by a white fixation cross (probability of 14%), 203 were standards accompanied by a red fixation cross (7%), and remaining 2291 were standards accompanied by a white fixation cross. The first block comprised 500 sound stimuli, whereas the remaining blocks comprised 400 sound stimuli each. Stimulus presentation was pseudo-

randomised. Each block started with five auditory standards that were accompanied by a white fixation cross. Auditory deviants were separated by a minimum of three and a maximum of nine standards. Standards with red crosses were separated by a minimum of three and a maximum of nine standards with white crosses. Additionally, the probability of auditory deviants and red crosses was identical across the seven blocks.

Although probe intensities were initially set to the level obtained from the hearing threshold test, they were subsequently altered if participants had near-zero or near-perfect oddball detection rates by the mid-point of the first block. In such cases the experimenter would adjust the intensity, according to her discretion, upward or downward as appropriate. When necessary, the experiment was restarted with additional adjustments to the probe intensity until detection rates were satisfactory. Five participants required an adjustment of deviant intensity (an increase or decrease of 1-3% of maximum sound volume) after a few restarts (less than 3). Four participants required more extensive adjustment (4-8 restarts) as they had attained abnormally low threshold values (i.e. low probe intensity) in the hearing threshold test. Possibly they were sensitive to mild distortions in all simultaneous probe-and-carrier presentations that were more difficult to detect in the oddball task, where deviants were much rarer and auditory attention had to be sustained continuously.

Participants were asked to focus on the fixation cross on the screen, while attending to the stream of sounds and maintaining normal blinking behaviour. They were also informed that the experimental task was potentially difficult and required their full attention. Before beginning each block, participants could choose to playback the deviant and standard sound stimuli as many times as they wished. During each block, they made button responses to auditory deviants and red crosses and ignored standards. They could choose to rest for a few minutes between blocks. The duration of the oddball task and simultaneous EEG recording was 45-60min.

**Data Acquisition and Analysis**

During the oddball task, EEG signals were recorded using a 64-channel Biosemi ActiveTwo system (Biosemi, Amsterdam, The Netherlands) and a sampling frequency of 256 Hz. Ag-AgCl electrodes were mounted in an elastic cap according to the modified 10-20 system. The electrooculogram (EOG), which registers eye movements, was recorded using three electrodes, which were attached above and below the left eye and at the outer canthus of the right eye. Additionally, recording electrodes were placed at the nose tip and at the left and right mastoids. Electrode impedance was below 25 kΩ.

EEG data were processed with EEGLab (Delorme & Makeig, 2004) running in the MATLAB (Mathworks, Natick, MA, USA) environment. The scalp recordings were re-referenced against the nose recording and a 0.1 to 20 Hz bandpass filter was applied. Continuous data was visually inspected for movement and other artefacts. Infomax, an independent component analysis algorithm implemented in EEGLAB, was applied to the remaining data and components reflecting typical artefacts (i.e., horizontal and vertical eye movements) were removed (figure 1). Back-projected data was subsequently epoched using a 150 ms pre-stimulus baseline and 800 ms following stimulus onset. The epoched data was base-line corrected and visually screened for residual artifacts. EEG epochs associated with incorrectly responded standards and standards accompanied by red crosses were excluded from further analysis.

The remaining data were classified into "blink" and "no-blink" standards and detected and missed deviants for later comparison. The EOG, which indicates eyelid position (Stern, 1984), was derived by subtracting the recording taken below the left eye from that taken above the same eye. The resulting signal was then subject to a bandpass filter of 0.1-20 Hz. As waveform shape and amplitude vary extensively between participants and across the course of the experiment, blink detection in the EOG was not automated. Instead, blink-typical deflections were visually identified – these were positive, narrow deflections, with similar shape and

amplitude characteristics, which were largest at the eye and frontal scalp electrodes and receded over posterior sites. An event was classified as a "blink trial" if at least one peak indicative of eyelid closure occurred within 300 ms before or after sound onset in the EOG waveform. This time window was chosen because (1) it encompasses the duration during which auditory feature traces are integrated into representations and then reach consciousness (about 250 ms after sound onset (Näätänen , Kujala and Winkler, 2011)), (2) accomodates the fact that suppression effects begin prior to eyelid closure and (3) enables a symmetrical assessment for the relationship between blink and sound onset, for the purpose of investigating the distribution of blinks before versus after sound onset. The remaining events were classified as no-blink trials. For standards, detected deviants and missed deviants, equal numbers of blink and no-blink epochs were averaged. This was achieved by matching each blink epoch (or no-blink epoch, whichever was fewer) to the closest preceding no-blink (or blink) epoch. The epoch numbers had to be balanced because no-blink epochs usually greatly outnumbered blink epochs and averaging would have suppressed noise unequally across conditions. The remaining unmatched no-blink (or blink) epochs were was discarded from averaging.

For the ERP analysis, the N100 and P300 components were defined as the mean amplitudes across specific time windows. These time windows were derived in the following way. First, the most negative peak within 70 to 180 ms post-stimulus onset and the most positive peak within 300 to 600 ms post-stimulus onset were identified across electrodes and conditions. The identified peak latencies were then averaged (for N100: $M$ = 110.73 ms, $SD$ = 29.86; for P300: $M$ = 394.81 ms, $SD$ = 116.23) and used as centre points for the N100 and P300 components. A 20 ms time window was centred around the N100, whereas a 100 ms time window was centred around the P300. Differences in the duration of the N100 and P300 time window were introduced to help account for differences in the temporal variability of these components within and across participants. Mean amplitudes during the N100 and P300 time windows were subjected to separate ANOVAs with *Stimulus* (Standard, Detected Deviant,

Missed Deviant), *Blink* (Blink, No-blink), *Region* (Anterior, Central, Posterior), and *Hemisphere* (Left, Midline, Right) as repeated-measures factors. The factors *Region* and *Hemisphere* comprised the following subgroups of electrodes: anterior–left: AP1, AF3, AF7, F5; anterior–midline: FPz, AFz, F1, F2; anterior–right: AP2, AF4, AF8, F6; central–left: FC3, C5, T7, CP3; central–midline: FCz, C1, C2, CPz; central–right: FC4, C6, T8, CP4; posterior–left: P5, PO3, PO7, O1; posterior–midline: Pz, POz, Oz, Iz; posterior–right: P6, PO4, PO8, O2. The average

**Table 1**

*Number of Epochs Entered into ERP Averages*

| | No-blink trials | | | Blink trials | | |
|---|---|---|---|---|---|---|
| Participant | Standards | Detected Deviant | Missed Deviant | Standards | Detected Deviant | Missed Deviant |
| 1 | 1211 | 94 | 111 | 752 | 46 | 93 |
| 2 | 1803 | 138 | 122 | 243 | 14 | 13 |
| 3 | 1849 | 200 | 119 | 269 | 24 | 19 |
| 4 | 1279 | 139 | 108 | 972 | 68 | 69 |
| 5 | 1617 | 99 | 193 | 625 | 25 | 65 |
| 6 | 1688 | 119 | 157 | 376 | 30 | 38 |
| 7 | 1780 | 152 | 166 | 332 | 16 | 35 |
| 8 | 1690 | 256 | 70 | 494 | 36 | 17 |
| 9 | 1288 | 149 | 82 | 981 | 89 | 64 |
| 10 | 1792 | 120 | 211 | 425 | 17 | 38 |
| 11 | 819 | 127 | 44 | 1515 | 129 | 97 |
| 12 | 1686 | 194 | 133 | 674 | 38 | 34 |
| 13 | 2011 | 184 | 169 | 259 | 14 | 24 |
| 14 | 1390 | 163 | 119 | 704 | 49 | 31 |
| 15 | 1595 | 174 | 105 | 695 | 59 | 54 |

potential of electrodes in these subgroups rather than their individual potentials was used for statistical analysis. Here, only significant effects involving *Blink* were followed up with simple effects tests.

Additionally, mean amplitudes of the N100 and P300 for only detected deviants and standards were subjected to separate ANOVAs with *Stimulus* (Standard, Detected Deviant), *Region* (Anterior, Central, Posterior), and *Hemisphere* (Left, Midline, Right) as repeated-measures factors. This additional analysis was to determine the presence of a general oddball effect independent of eyeblinks. Thus, only effects involving *Stimulus* were of interest here.

Signals from mastoid and eye channels were analysed separately. For each channel, mean amplitude values during the N100 and P300 time windows were entered into separate ANOVAs with *Blink* (Blink, No-blink) and *Stimulus* (Standard, Detected Deviant, Missed Deviant) as repeated-measures factors. Only significant effects involving *Blink* and *Stimulus* were followed up with simple effects tests.

Table 1 presents the total number of epochs from which ERPs for each level of *Blink* and *Stimulus* condition were derived.

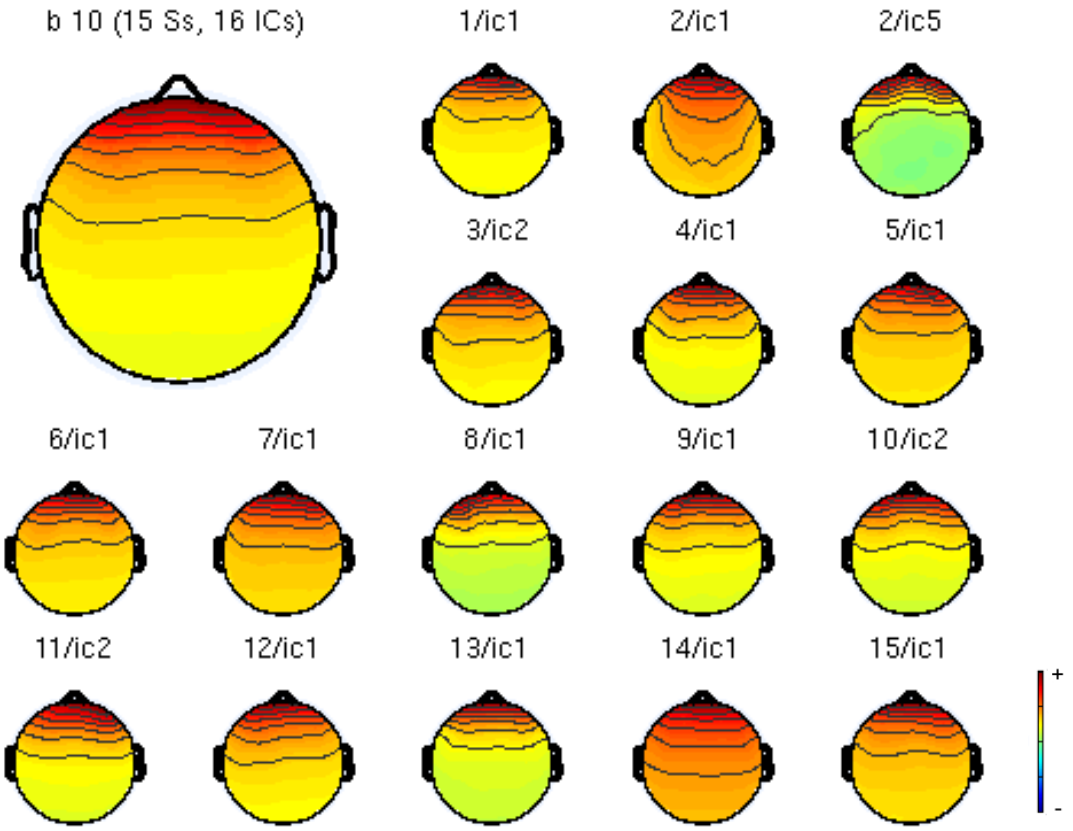


***Figure 1.*** Scalp topographies of the blink components (ic) for each selected participant and their grand average (largest plot, upper left). Titles above individual plots indicate participant ID (an arbitrary number) followed by component ("ic") number. Note that two blink related components were removed for one participant (5).

## Results

### Behavioural Measures

The behavioural data were analysed using the paired Welch t-test and the Chi-square test. One-tailed tests were used when the direction of an effect was predicted a-priori. Otherwise, two-tailed tests were used.

The percent correct detections of oddball sounds was higher for no-blink trials ($M$ = 54.50%, $SD$ = 12.65%) compared to blink trials ($M$ = 45.60%, $SD$ = 12.71%), $t(14) = 5.891$, $p < .001$, one-tailed.

Records were taken of the absolute duration between eyeblinks and sound onset in blink trials. This duration was, for pre-sound blinks, the time between full eyelid closure and the following sound onset, and for post-sound blinks, the time between sound onset and the following

**Table 2**

*Number and Mean Latencies of Pre- and Post-Sound Onset Blinks for Standard, Missed Deviant and Detected Deviant Trials.*

| Condition | Number of instances (across all participants) | Latency relative to sound onset (ms) | |
|---|---|---|---|
| | | *M* | *SD* |
| Pre-Sound Onset | | | |
| Standards | 5280 | -179.09 | 19.17 |
| Missed Deviants | 375 | -171.50 | 29.12 |
| Detected Deviants | 347 | -195.82 | 31.34 |
| Post-Sound Onset | | | |
| Standards | 5587 | 187.07 | 26.53 |
| Missed Deviants | 435 | 184.50 | 31.52 |
| Detected Deviants | 386 | 202.94 | 42.76 |
| Entire Time Window | | | |
| Standards | 10867 | 181.51 | 21.36 |
| Missed Deviants | 810 | 178.98 | 22.35 |
| Detected Deviants | 733 | 200.03 | 27.92 |

**Table 3**

*ANOVA table for N100 and P300 amplitudes.*

| Variable | df | F | *p* | |
|---|---|---|---|---|
| N100 Amplitude | | | | |
| Blink | 1, 14 | 4.9617 | .043 | * |
| Stimulus | 2, 28 | 0.7497 | .482 | |
| Region | 2, 28 | 2.5721 | .094 | |
| Hemisphere | 2, 28 | 0.5674 | .573 | |
| Blink x Stimulus | 2, 28 | 1.1879 | .320 | |
| Blink x Region | 2, 28 | 13.49 | < .001 | *** |
| Blink x Hemisphere | 2, 28 | 2.2932 | .120 | |
| Stimulus x Region | 4, 56 | 4.1026 | .006 | ** |
| Stimulus x Hemisphere | 4, 56 | 0.7555 | .559 | |
| Region x Hemisphere | 4, 56 | 1.292 | .284 | |
| Blink x Stimulus x Region | 4, 56 | 7.2618 | < .001 | *** |
| Blink x Stimulus x Hemisphere | 4, 56 | 0.4404 | .779 | |
| Blink x Region x Hemisphere | 4, 56 | 2.4465 | .059 | . |
| Stimulus x Region x Hemisphere | 8, 112 | 0.8652 | .548 | |
| Blink x Stimulus x Region x Hemisphere | 8, 112 | 0.2934 | .967 | |
| P300 Amplitude | | | | |
| Blink | 1, 14 | 1.5328 | .236 | |
| Stimulus | 2, 28 | 3.794 | .035 | * |
| Region | 2, 28 | 31.986 | < .001 | *** |
| Hemisphere | 2, 28 | 2.9428 | .069 | |
| Blink x Stimulus | 2, 28 | 1.7009 | .201 | |
| Blink x Region | 2, 28 | 24.519 | < .001 | *** |
| Blink x Hemisphere | 2, 28 | 3.6115 | 0.040 | * |
| Stimulus x Region | 4, 56 | 16.485 | < .001 | *** |
| Stimulus x Hemisphere | 4, 56 | 1.2147 | .315 | |
| Region x Hemisphere | 4, 56 | 2.1081 | .092 | |
| Blink x Stimulus x Region | 4, 56 | 4.4791 | .003 | ** |
| Blink x Stimulus x Hemisphere | 4, 56 | 0.9527 | .441 | |
| Blink x Region x Hemisphere | 4, 56 | 0.9446 | .012 | * |
| Stimulus x Region x Hemisphere | 8, 112 | 1.4764 | .174 | |
| Blink x Stimulus x Region x Hemisphere | 8, 112 | 1.0816 | .381 | |

full eyelid closure. The average duration between sound onset and full eyelid closure was greater for detected deviants ($M$ = 200.03 ms, $SD$ = 27.92 ms) than missed deviants ($M$ = 178.98 ms, $SD$ = 22.35 ms), $t(14) = 4.022$, $p < .001$, one-tailed. A chi-square test revealed no significant difference in the proportion of pre- and post-sound onset blinks between detected and missed deviant trials (see Table 2), $\chi 2(1, N=1543) = 0.129$, $p = 0.7195$. Thus, blinks were further from sound onset for detected deviants than missed deviants, while the blink distributions in relation to the sound were about the same.

There was no significant difference in reaction times for detected deviants with ($M$ = 554.3 ms, $SD$ = 82.70) and without blinks ($M$ = 559.4 ms, $SD$ = 85.42), $t(14) = 0.4213$, $p = .680$, two-tailed.

Additionally, the participants performed the visual change detection task satisfactorily (mean detection rate across participants = 96.78%, $SD$ = 4.41%).

**N100 (Scalp Electrodes)**

ERP data were submitted to a repeated measures ANOVA to determine the effects of *Blink* (with blinks, without blinks), *Stimulus* (standard, detected deviant, missed deviant), *Region*

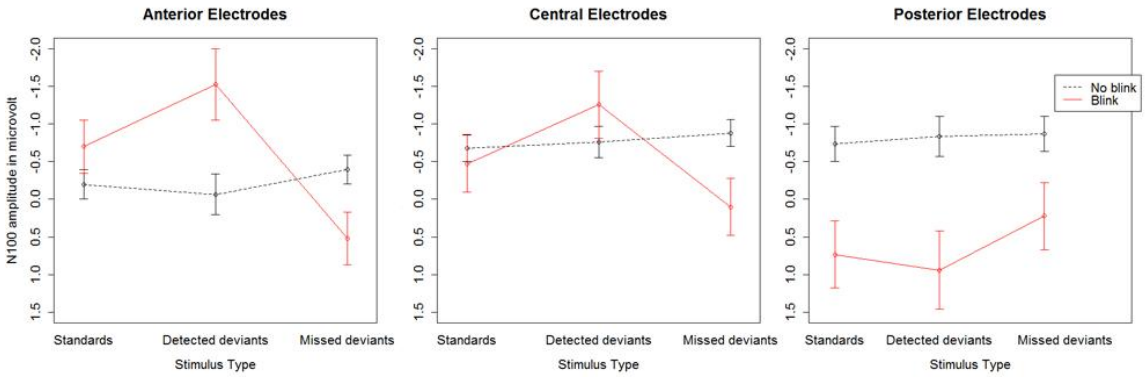


***Figure 2.*** Scalp N100 amplitude (mean potential across 120 to 140 ms) for each level of *Stimulus*, *Blink* and *Region* averaged across participants. The error bars are within-subject standard errors (Morey, 2008)

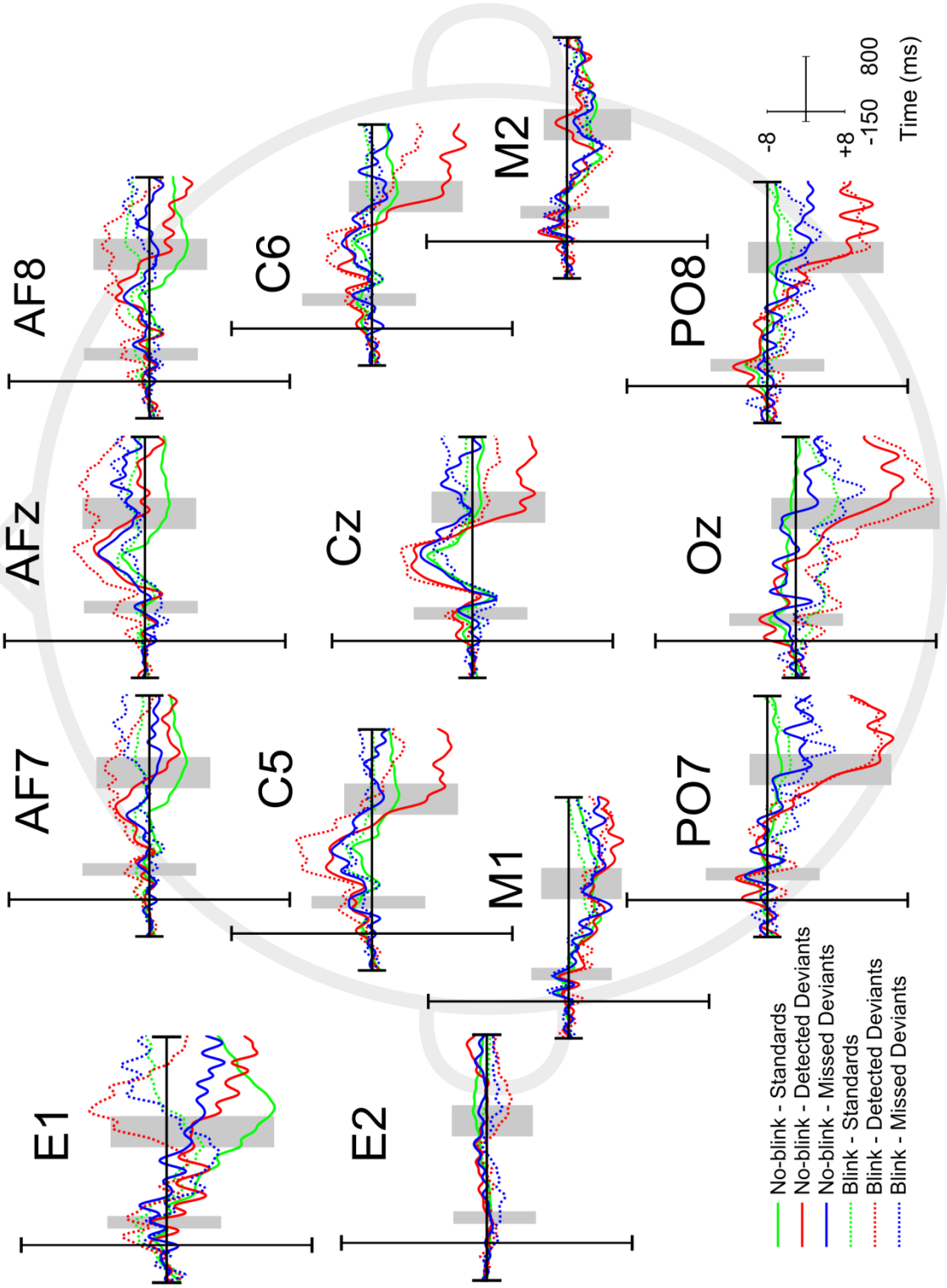


***Figure 3.*** Selected scalp, mastoid (M1 for left and M2 for right) and eye electrode (E1 for above left eye and E2 for below left eye) grand average ERPs time-locked to sound onset. No-blink trials are indicated with solid lines and blink trials with dotted lines; standards are in green, detected deviants in red and missed deviants in blue. The approximate latencies of the N100 and P300 time windows are indicated with grey rectangles.

(anterior, central, posterior) and *Hemisphere* (left, midline, right). The results are summarised in Table 3.

The *Blink* main effect ($F(1,14) = 4.96$, $p < .05$), the *Blink* by *Region* interaction ($F(2,28) = 13.49$, $p < .001$) and the *Blink* by *Stimulus* by *Region* interaction ($F(4,56) = 7.26$, $p < .001$) were significant. The latter interaction was followed up by examining the *Blink* by *Region* interaction (or the effect of *Blink* if the *Blink* by *Region* interaction was non-significant) for each level of *Stimulus* (see also fig. 3).

For detected deviants, the *Blink* by *Region* interaction was significant ($F(2,28) = 23.01$, $p < .001$) indicating that the *Blink* effect was significantly different across regions. Over anterior regions, the N100 was greater for blink than no-blink trials ($F(1,14) = 6.02$, $p < .05$), while over

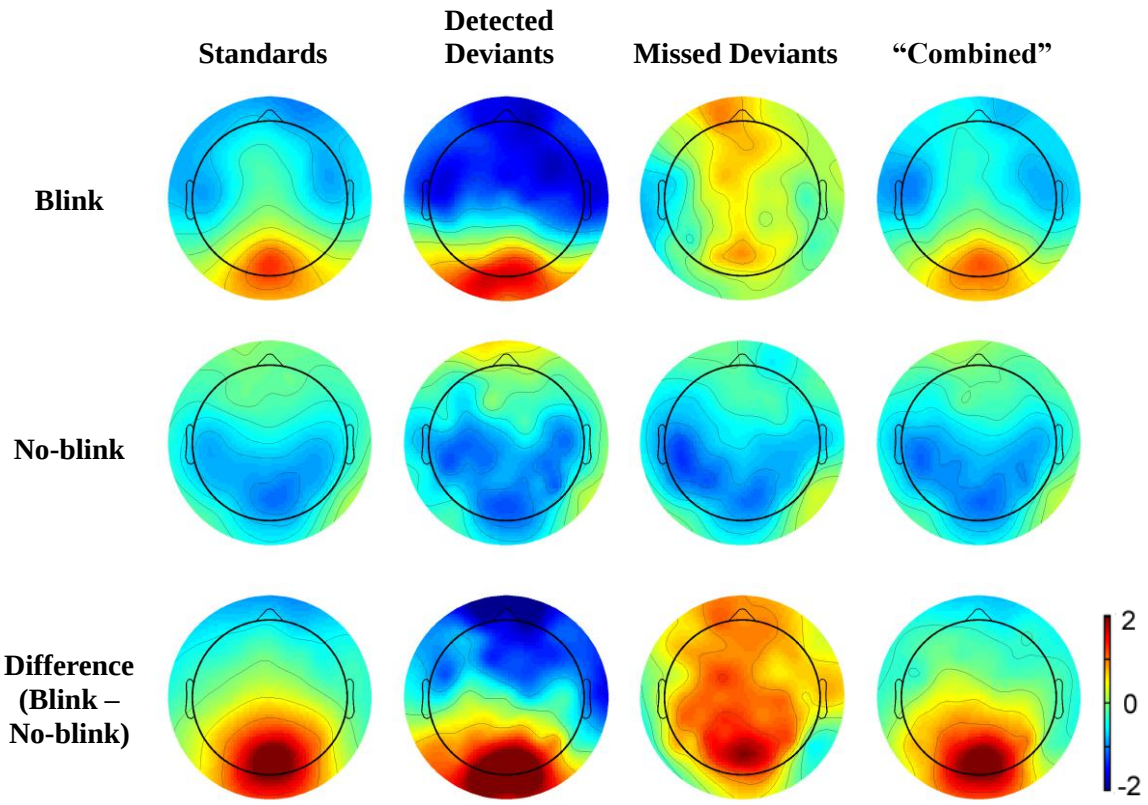


***Figure 4.*** Topographic maps (spherical spline interpolation) of mean activation (μV) at the N100 latency for each level of *Stimulus* (columns) and *Blink* (rows). The "combined" maps are calculated from all three *Stimulus* levels.

posterior regions, N100 was greater for no-blink than for blink trials ($F(1,14) = 6.50$, $p < .05$). Over central regions, the Blink effect was non-significant ($p > .1$).

For missed deviants, the *Blink* by *Region* interaction was not significant ($p > .1$). However, the *Blink* main effect was significant ($F(1,14) = 4.73$, $p < .05$) revealing a larger N100 for no-blink as compared to blink trials.

For standards, the *Blink* by *Region* interaction was significant ($F(2,28) = 18.09$, $p < .001$). Follow-up analysis revealed a *Blink* effect for posterior sites only. Here, the N100 was larger for no-blink than blink trials ($F(1,14) = 30.23$, $p < .001$).

ERP data were submitted to a second repeated measures ANOVA to determine the effects of *Stimulus* (standard, detected deviant), *Region* (anterior, central, posterior) and *Hemisphere* (left, midline, right). No effects involving *Stimulus* were found ($p > .1$).

**N100 (Mastoid and Eye Electrodes)**

ERP data from channels above the left eye, below the left eye and the two mastoids were entered into separate two-way ANOVAs with *Blink* and *Stimulus* as repeated-measures factors.

The eye channels were examined for ERP effects that might be attributed to residual eye movements artifacts. At the channel above the left eye, a significant *Blink* by *Stimulus* interaction was found ($F(2,28) = 4.69$, $p < .05$). An analysis by *Stimulus* showed that *Blink* was significant only for detected deviants ($F(1,14) = 11.00$, $p < .01$) for which the N100 was greater for blink than no-blink trials. All other effects were non-significant ($p > .1$). No effects were significant at the channel below the left eye ($p > .1$).

ANOVA results for both mastoids were also non-significant. However, due to our interest in determining whether the N100 blink effect has a supratemporal source, we conducted planned comparisons to determine if the mastoid *Blink* effects for detected deviants, missed deviants and standards were present and reversed in direction compared to the scalp. A significant *Blink* effect

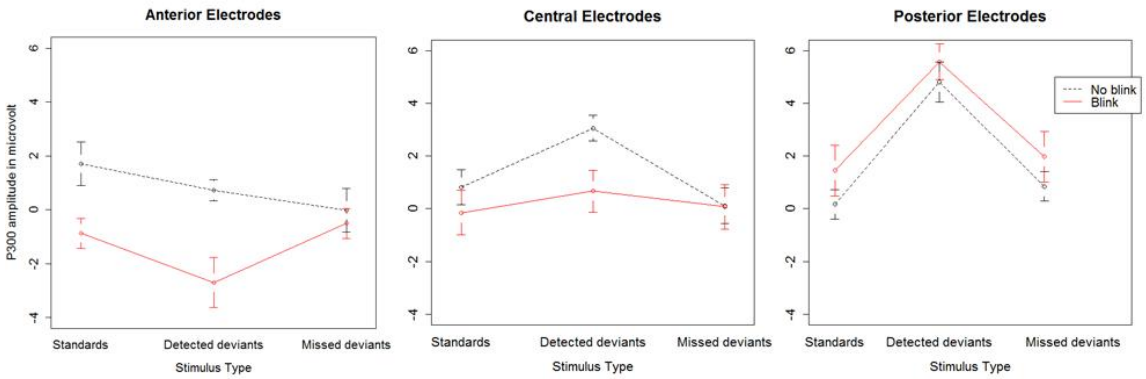


***Figure 5.*** Scalp P300 amplitude (mean potential across 470 to 570 ms) for each level of *Stimulus*, *Blink* and *Region* averaged across participants. The error bars are within-subject standard errors (Morey, 2008).

was found only for missed deviants at the right mastoid channel ($t(14) = 2.52$, $p < .05$, one-tailed) where, opposite to that seen at the other channels, voltages were more negative for blink than no-blink trials. The effect of *Blink* was not significant for detected deviants and standards at the right mastoid ($p > .1$), nor for any *Stimulus* level at the left mastoid ($p > .1$).

**P300 (Scalp Electrodes)**

ERP data were submitted to a repeated measures ANOVA to determine the effects of *Blink* (with blinks, without blinks), *Stimulus* (standard, detected deviant, missed deviant), *Region* (anterior, central, posterior) and *Hemisphere* (left, midline, right). The results are summarised in Table 3.

The *Stimulus* main effect ($F(2,28) = 31.986$, $p < .05$), *Blink* by *Region* interaction ($F(2,28) = 24.519$, $p < .001$), *Stimulus* by *Region* interaction ($F(4,56) = 16.485$, $p < .001$), and *Blink*, *Stimulus* and *Region* interaction ($F(4,56) = 4.48$, $p < .01$) were significant. The latter interaction was followed up by examining the *Blink* by *Region* interaction (or the effect of *Blink* if the *Blink* by *Region* interaction was non-significant) for each level of *Stimulus* (see also fig. 5).

For detected deviants, a simple interaction of *Blink* by *Region* was found ($F(2,28) = 24.72$, $p < .001$) indicating different *Blink* effect across regions. At anterior and central regions,

*Blink* was significant (anterior: $F(1,14) = 16.74$, $p < .01$; central: $F(1,14) = 5.54$, $p < .05$) where the no-blink P300 was greater than the blink P300. Over posterior regions *Blink* was non-significant ($p > .1$).

For missed deviants, the interaction of *Blink* by *Region* was not significant ($p > .1$). There was also no simple main effect of *Blink* ($p > .1$).

For standards, the interaction of *Blink* by *Region* was significant ($F(2,28) = 35.92$, $p < .001$). Only over anterior sites was the *Blink* effect significant ($F(1,14) = 16.41$, $p < .01$) with greater no-blink P300 than blink P300. For central and posterior regions *Blink* approached significance (central: $F(1,14) = 4.03$, $p = .064$; posterior: $F(1,14) = 3.95$, $p = .067$).

Analysis yielded a significant *Blink* by *Hemisphere* interaction ($F(2,28) = 3.61$, $p < .05$) and *Blink* by *Hemisphere* by *Region* interaction ($F(4,56) = 2.75$, $p < .01$). The latter interaction was followed up by examining the *Blink* by *Hemisphere* interaction (or the effect of *Blink* if the *Blink* by *Hemisphere* interaction was non-significant) for each level of *Region.*

At anterior sites, *Blink* by *Hemisphere* as a simple interaction effect was not significant ($p = .081$). However there was a simple effect of *Blink* ($F(1,14) = 12.38$, $p < .01$) where the no-blink P300 was greater than the blink P300.

At central sites, there were no significant *Blink* by *Hemisphere* ($p > .1$) or *Blink* effects ($p > .1$).

At posterior sites, the *Blink* by *Hemisphere* interaction was found to be significant ($F(2,28) = 7.31$, $p < .01$). Following up the *Blink* by *Hemisphere* interaction by *Hemisphere* showed that the *Blink* effect was present only at midline sites ($F(1,14) = 4.85$, $p < .05$) where the blink P300 exceeded the no-blink P300. *Blink* was not significant at left ($p > .1$) or right sites ($p > .1$).

ERP data were submitted to a second repeated measures ANOVA to determine the effects of *Stimulus* (standard, detected deviant), *Region* (anterior, central, posterior) and *Hemisphere* (left, midline, right). The analysis revealed a significant *Stimulus* by *Region*

interaction ($F(2,28) = 17.61$, $p < .001$) and a marginally significant *Stimulus* main effect ($F(1,14) = 3.405$, $p = .086$). Following up the *Stimulus* by *Region* interaction by *Region* showed that the *Stimulus* effect was present only at posterior sites ($F(1,14) = 16.75$, $p < .01$) where the detected deviant P300 exceeded the standard P300. *Stimulus* was not significant at anterior ($p > .1$) or central sites ($p > .1$). No other effects involving *Stimulus* were found. ($p > .1$).

**P300 (Mastoid and Eye Electrodes)**

ERP data from channels above the left eye, below the left eye and the right mastoid were entered into separate two-way ANOVAs with *Blink* and *Stimulus* as repeated-measures factors.

At the electrode above the left eye a significant *Blink* by *Stimulus* interaction was found ($F(2,28) = 3.84$, $p < .05$). Following up by *Stimulus, Blink* was significant only for detected deviants ($F(1,14) = 22.83$, $p < .001$) and standards ($F(1,14) = 14.78$, $p < .01$). For both cases the no-blink P300 was greater than the blink P300. No significant effects were found for missed deviants ($p > .1$). All other effects were non-significant. No effects were significant at the channel below the left eye ($p > .1$) or either mastoid ($p > .1$).

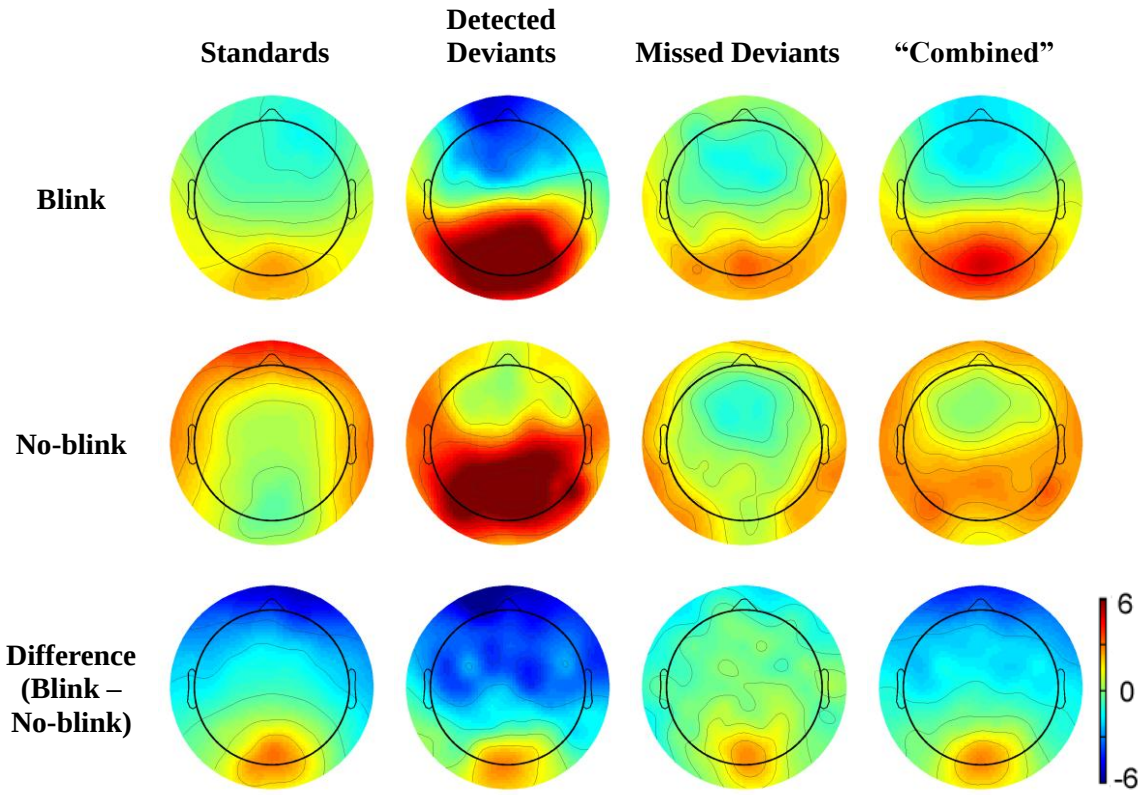


***Figure 6.*** Topographic maps (spherical spline interpolation) of mean activation (μV) at the P300 latency for each level of *Stimulus* (columns) and *Blink* (rows). The "combined" maps are calculated from all three *Stimulus* levels.

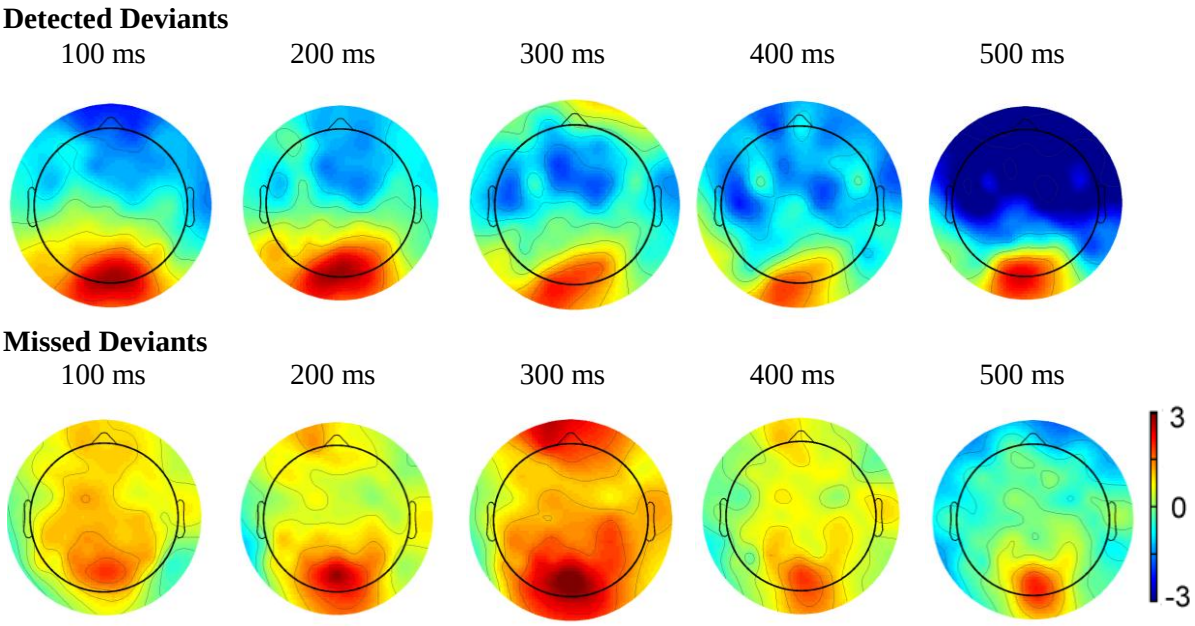


***Figure 7.*** Topographic difference maps (spherical spline interpolation) of blink minus no-blink ERPs (μV) at time points between 100 and 500 ms.

## Discussion

The present study examined the effect of blinking on auditory processing. It revealed evidence for such effects in the participants' detection of and ERP responses to deviant tones. In the following discussion we will examine these effects and explain how they advance our understanding of the functional significance of blinks.

### Behavioural Results

Analysis of the behavioural responses revealed that the detection of deviant tones declined in the presence of blinks and improved with increasing duration between blink apex and deviant onset. The latter finding demonstrates that the former finding cannot be completely attributed to factors such as time-on-task and fatigue.

There were no differences between detected and missed deviant trials with regard to the proportions of pre- and post-sound onset blink occurrences. This suggests that, with the time window currently used to categorise trials under the *Blink* factor (-300 to 300 ms), the effective time range of suppression for both pre- and post-sound blinks overlapped and caused an equitable degree of interference. Together with the fact that pre- and post-sound blinks are distributed almost equidistant from sound onset (see Table 2), the effectiveness of pre- and post-blink suppression are probably of similar duration. This interpretation agrees with the reported timescale for the visual effects of suppression, in which sensitivity begins to decline at least 150 ms before full eye closure and subsequent recovery takes about 100-200 ms afterward (Volkmann et al., 1980).

**ERP Results**

Based on the second set of ANOVA analyses, the ERPs show an overall adherence to previous oddball studies. Detected deviants elicited a greater P300 than did standards. Contrary to some previous studies (e.g., Butler, 1968), there was no general oddball effect on the N100. This may be due to the acoustic similarity between standards and deviants (e.g., Cranford, Rothermel, Walker, Stuart, & Elangovan, 2004) . Likely late attentive mechanisms were necessary to accurately discriminate between the two. Of greater interest for the purpose of the present study was the modulation of these ERP components by blinking. Specifically, we sought to determine how blinking affected the processing of deviants that were missed, deviants that were detected and standards. The following paragraphs discuss these effects, based on the first set of ANOVA analyses, first for the N100 and then for the P300.

***N100.*** Missed deviants elicited the expected blink suppression effect. The N100 for missed deviants was decreased in the presence of blinks across the entire scalp. In addition the difference was present and inverted in polarity at the right mastoid electrode, indicating changes in the supratemporal generators in the right hemisphere and inhibition of sensory processing in the auditory cortex.

The N100 to detected deviants was also modulated by blinking. However, blink effects differed across the scalp. Over posterior regions, the N100 was greater for no-blink than blink trials and thus showed a similar pattern as that observed for missed deviants. Over anterior regions, the N100 was greater for blink than no-blink trials. No N100 effects were found at the right mastoid, indicating that the effect on the supratemporal generator together with the associated auditory suppression was either absent or obscured by other neural activity. The latter possibility seems more plausible.

Firstly, it is difficult to explain the profound spatial heterogeneity of the blink effect as modulation of the N100 generators alone. It is more likely that the detected deviants were subject

to a scalp-wide N100 decrement as in the missed deviants, together with a blink-related anterior negative shift.

Secondly, although missed deviants overlapped more closely with blinks than did detected deviants, the difference in overlap was only about 20 ms (Table 2). Moreover, blink suppression has been reported for an interval of 300 or more ms centred around full eyelid closure and described as a gradual rather than a step process. Thus, it seems more likely that blink suppression was simply a bit weaker than entirely absent for detected as compared to missed deviants.

Thirdly, the dramatically increased anterior negativity for blink as compared to no-blink epochs undoubtedly added to the potentials recorded at the mastoid and, if its source was not temporal, reduced any chances of identifying inversion effects of the supratemporal N100 subcomponent. Notably, the blink enhanced anterior negativity was of longer duration than the ordinary N100 latency range and clearly marked the emergence of an additional process. Based on its frontal scalp topography as well as prior fMRI work that revealed frontal cortex activation during blinking (Bristow, Haynes, et al., 2005), one may infer a frontal origin and speculate that this negativity reflects the recruitment of resource allocation processes. For example, it may reflect the automatic recruitment of attentional resources at blink margins to help compensate for the temporary suppression in sensory sensitivity. This suggestion is in line with existing evidence of a negativity called N200. This negativity, also known as the mismatch negativity (MMN) (Näätänen, 1995; Näätänen et al., 2011), has an anterior topography, has a latency of 100 to 200 ms relative to stimulus onset, and is elicited by deviants in an attended or ignored stimulus sequence. The MMN has, like the N1, generators in temporal and frontal regions, which are linked to preperceptual auditory processing and the orienting response to stimulus change respectively. It is most likely the frontal generator that is implicated here.

The N100 to standards was decreased in the presence of blinks only at posterior sites. Given that the auditory N100 is reduced for standards relative to deviants, blink effects during

standards may act primarily on visual processes supported by the primary visual cortex. Hence, the ERP effects are restricted to posterior sites (Berg, 1986; Hoffmann & Falkenstein, 2008).

***P300.*** For missed deviants, there were no blink effects in the P300. This may not be surprising given that blinks suppressed the preceding N100 and reduced deviant detection rates. Thus, missed deviants were not perceived differently from standards and no P300 ensued.

For detected deviants, blinks reduced P300 amplitude over anterior and central sites but not over posterior sites. The distribution of this reduction implicates the more anterior "novelty" P3a instead of the P3b. We postulate that the P300 effect is another indicator of blink suppression, but one which is visible only in stimuli that reach awareness. Blink suppression might affect P3a-related functions which mediate the conscious processing of detected deviants and standards, while these functions were simply not engaged for the missed deviants. Blink suppression for detected deviants makes them appear more diminished and less attention-grabbing than they ought to be.

For standards, blinks reduced the ERP amplitude over anterior sites only. This appears to be a weaker degree of suppression relative to that observed for detected deviants. One may be surprised to observe such suppression in a P300 time window for standards, given that these are typically not considered relevant for this component. However, it is conceivable that attention to standards waxed and waned during the course of the experiment. Stimuli with more attention likely produced a more positive potential in the P300 time window than stimuli with less attention. As a consequence, perception of the former stimuli would be subject to blink suppression and an average across the two may not completely remove this effect. Thus, we see a P300 suppression albeit to weaker degree than that observed for detected deviants.

One ought to be cautious when interpreting late latency effects due to a possible carry-over from earlier processes (e.g., the possible MMN contribution described in the N100 section). However, we believe that the present P300 effects are genuine and independent of earlier components. The reason is because the blink-modulated P300 decrement was not correlated with

the N100 enhancement, as the latter was present in the detected deviants but absent in the standards.

Analysis also revealed an additional P300 blink effect that was present at all levels of *Stimulus*. The P300 at midline posterior sites was increased during the presence of blinks. Given its topography and insensitivity to response or stimulus type, this effect can be identified as the blink-related occipital positivity (Berg, 1986; Hoffmann & Falkenstein, 2008) which peaks about 250 ms after blink maximum. This positivity is thought to underlie the visual evoked response from the reopening of the eye.

**What Happens during an Eyeblink?**

The present findings demonstrate the influence of eyeblinks on performance in an auditory task as well as an ERP component which is intrinsically tied to auditory cortex function. Suppression was seen both in the ability to distinguish sounds by intensity and in the early perceptual processes indexed by the auditory N100. Both show eyeblinks to affect perceptual processes aside from vision and thus lend support to the concept of blink suppression as an automatic, cross-modal mechanism.

What follows is a discussion of the processes that potentially underlie blink suppression, based on the feature trace model of auditory processing (Näätänen et al., 2011; Näätänen & Winkler, 1999). As described earlier, blink suppression emerges gradually across a time window, which extends from at least 150 ms before blink onset to at least 200 ms after. The present blinking effects on the supratemporal N100 overlapped with this time window thus indicating that the locus of suppression is preattentional and prerepresentational. The N100 decrease could be interpreted as failure to “refresh” feature traces, possibly by inhibiting either feature detector activity or the elaboration of incoming feature information. Weakened feature traces when integrated would create poorer and less informative representations of the sound

stimulus in sensory memory, decreasing the probability of the representation triggering attention and change detection. Nevertheless, there are instances in which stimulus representation that emerge during blinking still successfully trigger attention and become perceived consciously in spite of ongoing suppression. Specifically, depending on the temporal relationship between blinking and stimulus onset, compensatory mechanisms set in that amplify executive aspects of stimulus processing. This scenario would apply to the detected deviants, which displayed an increased frontal N100 amplitude as well as a decreased P3a amplitude. The latter suggests that, despite being correctly categorised and responded to, they might appear less divergent from standards than they ought to be.

Before closing this discussion, let us consider the observed amplification mechanism a bit further. Likely it does not inhibit the channels through which suppression takes place, instead amplifying other processes to counteract suppression. Given the great overlap between blink distributions for detected and missed deviants, suppression occurring both before and after the blink maximum can be recovered from and the activations of the suppression and compensation effects must be fairly close to each other. This mechanism might be more readily activated at the early onset and late offset of blinks. Given the latency of the effect, its trigger should presumably lie at preattentional processes.

The frontal MMN generator, as discussed earlier, is considered to be the most probable source of this compensatory anterior negativity given its timing, duration, topography and related functions. In addition, this source presents a scenario that fits well with the preceding discussion about the suppression process. Postulating from the characteristics of the frontal MMN, the compensation mechanism would involve the attention-call process operating at sensory memory. The impoverishment of suppression-affected sensory representations would be offset by allowing an easier call to conscious attention, possibly by increasing the sensitivity of underlying change detection functions to deviance, or by lowering the threshold these functions require to exceed in order to activate conscious attention. The former scenario would mean that triggering

compensation simply relies on the quality of the stimulus representation (i.e. whether the representation contains sufficient information to indicate deviance in spite of suppression) and is mediated automatically.

**Caveats**

The interpretations given above hinge on a few assumptions that deserve special consideration. Firstly, the experiment relied on endogenous rather than reflex and voluntary blinks because the latter have been reported to cause dual-task confounds and subsequent ERP effects (Verleger, 1991). Moreover, we generalised from previous work that the blink effects seen here should be comparable across blink types (Manning, Riggs, & Frost, 1983; Manning, Riggs, & Komenda, 1983). Nonetheless, it would be important to ascertain this fact in future studies. Secondly, the present ERP data were interpreted on the grounds that eye movement artefacts were fully removed from the signal and thus did not contribute to the modulation of target ERP components. However, this point is difficult to ascertain because ERP sources cannot be unambiguously identified.

Moreover, inspection of blink and no-blink ERPs revealed clear differences that could have a brain or eye basis. We would like to make the case that they are brain based because we employed similar ERP "cleaning" methods as have been used by previous studies and as have been found acceptable. With these methods only a residual posterior positivity has been reported peaking at about 250 ms after full eyelid closure and with a likely neuronal origin (Hoffmann & Falkenstein, 2008). Because blinks were not time-locked to the ERP in the present study, the present data contained this blink-related influence smeared across the entire averaging epoch. The difference scalp maps (figure 7) illustrate this as a positive potential of about 2-3 μV in posterior regions, which is present in all blink conditions and absent in all non-blink conditions. Given the

timing of this component identified in prior work (Hoffmann & Falkenstein, 2008), it is likely to reflect post-suppression processes such as a visual evoked potential from reopening the eye.

Apart from our use of validated data cleaning procedures, another argument can be made for a brain basis of the observed blinking effects. This argument rests on a comparison of the scalp topography of our blink effects against that expected by residual eye movements in the ERP. Given that eye movements associated with blinks produced a positive deflection in the ongoing EEG, residual eye movements should produce a more positive ERP for blink as compared to no-blink trials and this difference should be most pronounced over frontal electrode sites and decline towards central and posterior regions. Furthermore, it should show no polarity inversion over the mastoids. As none of the condition differences reported in the present study fit such a pattern, they are unlikely to be caused by movements of the eye.

**Implications and Questions for Future Research**

The results reveal several things about blink suppression: its multimodality, the timings and natures of some of the underlying processes, and a possible compensatory mechanism. While that still leaves a lot to be explained, these findings present a foundation for more detailed study of blink suppression as a cognitive phenomenon. From a bigger perspective however, understanding blink suppression would not merely describe a single specialised phenomenon, but also elucidate further the relationship between perception and cognition.

Given the early preattentive nature of blink suppression effects, earlier ERP components may be of interest in future studies. A good target is the auditory P1 (or P50) (Frederick, Boop, Garcia-Rill, Dykman, & Skinner, 1994) which is linked to automatic and preattentive sensory gating. This component was not analysed presently as P1 effects are usually quantified as the extent of habituation to repeating stimuli(e.g., Gillette et al., 1997) , instead of

amplitude changes to oddballs. The MMN is another obvious candidate for study, given its speculated involvement in the compensation mechanism.

We may also ask whether there are any differences between the effects of eyeblinks that come before stimuli and the effects of eyeblinks that come after. Although the distribution of eyeblinks in the behavioural results suggest that eyeblinks are equally suppressive pre- and post-sound onset, it is possible that each suppresses different processes to create a performance deficit. One approach to this question would be a comparison of ERPs containing pre-stimulus blinks to those containing post- stimulus blinks. However, we were unable to do so for the present study due to a lack of data for the generation of satisfactory ERPs. Resolving this issue in future studies could yield useful information.

The present findings have important implications for the treatment of blinks in behavioural and neuroimaging research. Currently, this research operates on the implicit assumption that blinks are distributed equally across experimental conditions. However, as the literature on blinking and cognitive or emotional load demonstrates, this assumption may not be warranted. Thus, many experiments may inadvertently create conditions that elicit differences in the frequency of blinking, which may then produce differences in the behavioural and neuroimaging results. In the future, investigators need to monitor blinking, consider how the design of an experiment may affect blinking behaviour, and include potential changes in blinking into their explanations of how certain experimental stimuli or tasks affect ongoing mental processes.

**Conclusions**

In conclusion, the current study showed that blinks reduce sensory and cognitive responses to auditory events thereby providing original evidence that blink suppression is a cross-

modal phenomenon. Additionally, the present results revealed a process that seems to counteract suppression both before and after the blink maximum and that seems to help compensate for a transient lack in sensory awareness. Taken together, the findings present a clearer picture of the processes that maintain perceptual stability across modalities.

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