UNIVERSITÀ
DEGLI STUDI
DI PADOVA

University of Padova

Department of Developmental Psychology and Socialization

Ph.D. School in Psychological Science

XXX Series

# On the Common Neural Basis of Temporal and Spatial Dynamics of Attention:
# Evidence from Human Electroencephalography

Thesis written with the financial contribution of University of Padova

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# Abstract (English)

This thesis investigated, by using electroencephalography (EEG) technique, the deployment of attention in time and space. Specifically –through three experimental chapters– Attentional Blink (AB) and visual search paradigms have been employed to highlight the common functional characteristics of the mechanisms which drive attention in time and space.

In Chapter 1, an overview of visual attention is presented. Specifically, I proposed a theoretical introduction regarding the two aspects of visual attention debated throughout the manuscript, namely, the AB phenomenon –that is an effect related to the temporal dynamics of visual attention– and visual spatial attention.

Results presented in Chapter 2 show how temporal dynamics of visual attention are affected by the AB effect, by analyzing how detection and encoding of a target are influenced when salient visual information is presented in temporal proximity. In line with Chapter 2, the experiment presents in Chapter 3 merged together, in a single experimental design, the AB and the visual search paradigms. In this study, I investigated whether the deployment of visual spatial attention in space is prone to the same experimental manipulations which influence detection and encoding of targets in the AB paradigm. Given the results, to assess why temporal dynamics of attention are similar both for midline- and lateral- presented information, in Chapter 4 visual spatial attention has been investigated with a visual search task, by comparing the electrophysiological activity elicited by a lateral presented target vs. a midline presented target.

Eventually, in Chapter 5, a general discussion highlights the main findings presented in this thesis, by considering them collectively, and by raising future proposals and questions in relation to the topics debated in these Chapters.

# Abstract (Italiano)

Questa tesi ha investigato, attraverso la tecnica dell'elettroencefalografia (EEG), l'allocazione dell'attenzione nel tempo e nello spazio. Specificamente – attraverso tre capitoli sperimentali– il paradigma di Attentional Blink (AB) e quello di ricerca visiva sono stati impiegati per mettere in luce le caratteristiche funzionali in comune tra i meccanismi che guidano l'attenzione nel tempo e nello spazio.

Nel Capitolo 1, viene proposta una panoramica in merito all'attenzione. Specificamente, ho proposto un'introduzione teorica riguardo i due aspetti dell'attenzione visiva trattati nel manoscritto, ossia il fenomeno dell'AB –che è un effetto relato alle dinamiche temporali dell'attenzione visiva– e l'attenzione visuo-spaziale.

I risultati presentati nel Capitolo 2 hanno mostrato come le dinamiche temporali dell'attenzione visiva vengano modificate dall'effetto AB, analizzando come la detezione e il consolidamento di un target siano influenzate quando delle informazioni visive salienti vengono presentate in prossimità temporale. In linea con il Capitolo 2, l'esperimento presentato nel Capitolo 3 ha unito assieme, in un unico disegno sperimentale, i paradigmi di AB e di ricerca visiva. In questo studio, ho investigato se l'allocazione di risorse visuo-attentive nello spazio sia soggetta alle stesse manipolazioni che influenzano la detezione e il consolidamento dei target nell'AB. In relazione a quanto emerso, per valutare perché le dinamiche temporali dell'attenzione siano simili per informazioni visive presentate lungo la linea mediana verticale e lo spazio lateralizzato, nel Capitolo 4 l'attenzione visuo-spaziale è stata investigata con un compito di ricerca visiva, comparando l'attività elettrofisiologica elicitata da un target presentato lateralmente vs. un target presentato lungo la linea mediana verticale.

Per concludere, nel Capitolo 5, una discussione generale ha evidenziato i risultati principali presentati in questa tesi, considerandoli congiuntamente, e suggerendo proposte di studio future in relazione ai temi trattati in questi capitoli.

# Chapter 1 – General Introduction

One of the most enduring issues in cognitive neuroscience concerns the neural substrate(s) underling conscious perception. This has been a topic of intense investigation for several decades, and despite a definitive understanding of the neural underpinnings of consciousness remaining elusive, there is a general consensus that conscious perception is not tied to a single neural structure but rather reflects the interplay of distinct brain areas. For instance, Dehaene, Sergent, and Changeux (2003) and Baars (1993) hypothesize that conscious perception represents the engagement of a global neural workspace. Specifically, for a stimulus to enter consciousness, neurons with long-distance axons that can connect distinct brain regions must be activated, which then allows communication between higher-level processing areas and those that are involved in sensory analysis (Antonino Raffone & Pantani, 2010). Similarly, Lamme and colleagues (Fahrenfort, Scholte, & Lamme, 2007; Lamme & Roelfsema, 2000) and Di Lollo, Enns, and Rensink (2000) predict that for information to be accessed consciously not only must it pass from sensory to higher-level structures but must also be fed back, and it is through these reentrant iterations that conscious representations are established.

Strictly related to conscious perception there is attention: if we pay attention to an object, we became conscious about it, and if we shift our attention away the object fade from consciousness (O'Regan & Noë, 2001; Posner, 1994). Based on this view, attention is at least a fundamental component of conscious perception. Aspects of visual attention related to this whole thesis will be describe in the next sections.

# 1.1 Temporal Dynamics of Visual Attention

## 1.1.1 Attentional Blink Phenomenon

A key phenomenon for studying conscious perception is the Attentional Blink (AB, Raymond, Shapiro, & Arnell, 1992), participants' typically impaired ability to perceive the second of two targets (T2) in a rapid serial visual presentation (RSVP) if it appears within 200–500 ms of a first target (T1). Paradoxically, T2 is much easier to report when it follows T1 immediately. This effect is referred to as lag 1 sparing and it is thought to reflect T1 and T2 being processed together within a single attentional episode (Chun & Potter, 1995; Dell'Acqua, Dux, Wyble, & Jolicœur, 2012; Wyble, Potter, Bowman, & Nieuwenstein, 2011). To note, the lag is the serial position of T2 from T1: for instance, if after T1 two distractors and then T2 are showed, T2 is presented at lag 3.

The AB is well-known to be an attentive (and not perceptual) phenomenon, since when the subject is asked to report only T2 (when T1 is still presented), the accuracy is high and similar regardless the distance between T1 and T2 in the RSVP. According to this behavioral evidence, functional magnetic resonance (fMRI, Choi, Chang, Shibata, Sasaki, & Watanabe, 2012; Kranczioch, Debener, Schwarzbach, Goebel, & Engel, 2005; Marcantoni, Lepage, Beaudoin, Bourgouin, & Richer, 2003; Marois, Yi, & Chun, 2004; Marois & Ivanoff, 2005) and positron emission tomography (PET, Slagter et al., 2012) explorations have localized AB effects to a frontoparietal attentional network composed of core nodes in the posterior parietal and dorsolateral prefrontal cortex that support a variety of attention tasks (e.g., Corbetta & Shulman, 2002; Desimone & Duncan, 1995). A set of additional areas have been shown to be susceptible to the AB influence, including striate (Williams, Visser, Cunnington, & Mattingley, 2008) and extrastriate visual areas (Dell'Acqua, Sessa, Jolicœur, &

Robitaille, 2006; Marois et al., 2004), and subcortical structures (i.e., basal ganglia and locus coeruleous), whose roles have been incorporated in neural instantiations of AB models (Colzato, Slagter, de Rover, & Hommel, 2011; Hommel et al., 2006; Nieuwenhuis, Gilzenrat, Holmes, & Cohen, 2005).

Evidence converging with the hypothesis that the AB engages a frontoparietal attention circuit comes from studies employing EEG and magnetoencephalography (MEG) techniques that have explored the correspondence between AB effects and non-phase-locked (Bastiaansen, Mazaheri, & Jensen, 2012) neural synchronization of scalp-recorded oscillations. AB-induced decreases in long-range phase synchronization in the beta and gamma band encompassing the frontoparietal attention network have been reported (Gross et al., 2004, Kranczioch, Debener, Maye, and Engel, 2007, Nakatani, Ito, Nikolaev, Gong, and Leeuwen, 2005. These modulations were consistently observed before T1 onset, a pattern akin to spontaneous trial-by-trial fluctuations in alpha band desynchronization which has been proposed to index an anticipatory mental state related to successful identification of RSVP targets (Hanslmayr, Gross, Klimesch, & Shapiro, 2011; MacLean & Arnell, 2011, for a review).

A key approach for isolating the stages of information processing that gives rise to the AB is the event-related potential (ERP) technique. Studies employing this approach have focused primarily on AB modulations of the P3b component. Typically observed at midparietal electrode sites, this waveform has been shown to reflect consolidation of visual information in short-term/working memory (Akyürek, Leszczyński, & Schubö, 2010). Indeed, P3b can be taken as the hallmark of a widespread state of activation following detection of visual target information aiding memory encoding (Fabiani & Donchin, 1995; Johnson, 1995; Kranczioch, Debener, &

Engel, 2003). Two complementary studies have demonstrated that the AB must reflect, at least to some extent, T2 memory consolidation being postponed when the two targets appear in close temporal proximity in an RSVP stream. By using standard RSVP trials terminating with one or more distractors following T2, Vogel, Luck, and Shapiro (1998) showed that T2-locked P3b is influenced by the AB, in the form of a sizable T2-locked P3b amplitude reduction at short relative to long lags. The neural sources of the P3b have been localized to posterior brain structures including posterior-parietal areas and the temporoparietal junction (TPJ, e.g., Johnson, 1993; Polich, 2003).

The above AB and P3b findings fit well with behavioral studies, which suggest that T2 processing is delayed during the AB, which renders it more vulnerable to interruption from subsequent stimuli. However, what cannot be determined from these measurements of the P3b is whether it is the detection or the additional encoding of T2 that is delayed. To address this question, a different associated with the detection of relevant stimuli should be considered, namely, P3a. The P3a is typically observed at midfrontal electrodes and occurs before the P3b. Lesion studies (Knight, 1991), fMRI/EEG multimodal acquisition (Bledowski, Prvulovic, Hoechstetter, et al., 2004; Bledowski, Prvulovic, Goebel, Zanella, & Linden, 2004), and neurobiological analyses (Gazzaniga, Ivry, & Mangun, 2000) have pointed to a set of frontal structures generating P3a that include anterior cingulate and lateral prefrontal cortices (Friedman, Cycowicz, & Gaeta, 2001). Whereas earlier proposals have suggested that the P3a primarily indicates novelty and sensory deviance of cross-modal stimulation (e.g., Courchesne, Hillyard, & Galambos, 1975), more recent views on P3a link it to the deployment of attention for detection of contextually salient information presented among distracting stimuli (e.g., Barceló, Escera, Corral, & Periáñez, 2006; Cycowicz &

Friedman, 1998; Koechlin, Ody, & Kouneiher, 2003; Polich, 2007), especially in tasks where such classification is difficult (Polich & Comerchero, 2003).

There are several models of AB interference but they do not all agree on the role that attentional engagement plays in the deficit. Chun & Potter's (1995) two-stage model, for example, proposes that only one target at a time can be consolidated in memory. Any subsequent target therefore should wait until the first target is fully encoded before having access to the encoding stage. Meanwhile, if a second target is too close in time to the previous target, the perceptual trace of the second one fades (or is overwritten) before it can be encoded. It is not clear, in this model, what role attention might play. The episodic simultaneous type, serial token model (Wyble, Bowman, & Nieuwenstein, 2009) holds that activation of a target is enhanced by attentional mechanisms. Accordingly, the AB deficit takes place because attention inhibits trailing distractors during the encoding of the first target. If the second target is displayed during this temporary inhibition, its activation cannot be enhanced and eventually encoded in working memory.

### 1.1.2 Lag 1 sparing phenomenon

As mentioned at the begin of this section, lag 1 sparing represent a peculiar phenomenon for which when two targets are presented in sequence, both of them are often reported correctly. Reeves & Sperling, (1986, see also Nakayama & Mackeben, 1989) have shown that the time course of attention deployment to RSVP targets is well approximated by a gamma function, with a steeply rising rate of information accumulation peaking at about 100–150 ms after target onset, followed by a gradual return to baseline. Given that RSVP items are often presented at rates close to 10 Hz, this implies that attention deployment to RSVP items is likely to be at its peak when the

item after the first target is displayed. Indeed, a considerable number of evidence shows that if the item after the first target is another target, then it is often spared from the AB.

Two computational accounts of the which have specifically addressed lag 1 sparing phenomenon, and made explicit claims concerning ERP findings, are useful in the present context. These theories make predictions regarding how T1-locked P3a, reflecting attention deployment to target(s) and associated with neural structures localized frontally, should vary as a function of whether the item which follow the first target is a distractor or a subsequent target. Both models predict that, with minimal latency variations, P3a amplitude should be greater when T1 is immediately followed by T2 relative to when T1 is followed by a distractor. According to Olivers and Meeter (2008), a distractor trailing T1 curtails attention deployment by eliciting an inhibitory response. The attentional response would, in contrast, have time to unfold to a greater extent when the item after the first target is another target, that is, when the inhibitory response –elicited by the distractor that follow the first target– is postponed by a time corresponding to the second target exposure duration. This activation asymmetry between a weakly activated first target and a strongly activated second target has been raised as the cause of order reversals in consecutive target report and for the better report of the second target relative to the first at lag 1 (Olivers, Hilkenmeier, & Scharlau, 2011). Similarly, Wyble and colleagues (2011) propose that both the targets elicit attentional responses, but are processed in the same attentional window when presented sequentially, with the first target enhancing attention deployment to the second. Attentional enhancement would be discontinued when the item which follow the first target is a distractor. Thus, both Olivers and colleagues and Wyble et al. (2011) maintain that order reversals in target report and the increased report accuracy for the

second target relative to the first are determined by the resulting asymmetry in target activations, with the second target overtaking the first on a sizable proportion of trials.

Despite some similarities, these two models differ substantially with reference to time course and localization of ERP responses following P3a. The root cause of the AB in Olivers and Meeter's (2008) model is a transient inhibition (so-called bounce response) elicited by the T1 + 1 distractor to contrast the initial attention boost to T1 and prevent access of trailing nontarget items to working memory. In support of this hypothesis, Olivers and Meeter (2008) cite ERP evidence described by Martens, Munneke, Smid and Johnson (2006), who explored the processing differences between blinkers (i.e., subjects who show average AB effects with RSVP) and nonblinkers (i.e., subjects who appear to be immune to the AB and tend to miss T2 in less than 10% of RSVP trials). Martens et al. (2006) reported that T1 elicited an initial positive component recorded in a 180–350 ms time range post-T1 at frontal electrode sites (F7 and F8), dubbed frontal selection positivity (FSP; e.g., Smid, Jakob, & Heinze, 1999), followed by a negative component observed at these frontal electrodes. Although this negative component was not parametrically investigated by Martens et al. (2006), Olivers and Meeter (2008) noted that the time course of the post-FSP frontal negative component, held to be the correlate of the bounce response, had a temporal extension of 300–500 ms after the offset of the FSP component, displaying therefore an interesting overlap with the time course of the AB.

According to the model of Wyble et al. (2011), the AB is symptomatic of the visual system's overarching goal of generating episodically distinguishable episodes. As surmised above, targets in RSVP undergo attentional enhancement, which I proposed as indexed by an increment of frontal positivity, in order to bring their early sensory (and conceptual) representations beyond a certain threshold such that targets

can be subsequently encoded as reportable episodes and stored in working memory. Upon detection of a discontinuity in target presentation (e.g., upon detection of a distractor), attention enhancement is discontinued and tokenization encompasses all target information subject to attentional enhancement. Once tokenization is under way, no further targets can be subject to attention enhancement, with increased probability for unattended targets to be missed, bringing about an AB effect.

### 1.1.3 Visual Masking and the AB

Among the factors which have been found to influence the AB, one of which is the backward masking caused by the distractor following T2. Masking is defined as the reduction in visibility of a stimulus (target) by a spatially or temporally close second stimulus (mask) (Bachmann, 1984; Breitmeyer & Ogmen, 2000). Giesbrecht and Di Lollo (1998) found that when the RSVP in an AB paradigm ended with the last target instead of an additional distractor (a mask), no behavioral AB occurred; accuracy for the last target was at ceiling. Later works showed that an AB can be found even when the last target is not masked as well as when distractors are replaced by blank intervals, but it is invariably smaller in amplitude compared with the AB found with a trailing mask (cf. Arnell & Jolicœur, 1999; Nieuwenstein, Potter, & Theeuwes, 2009; Ptito, Arnell, Jolicœur, & MacLeod, 2008). Masking is often assumed to erase the visual information from the target or to interrupt its processing, although theories which are seeking to explain masking are more complex and more nuanced (Breitmeyer & Ogmen, 2000).

As mentioned previously, Vogel et al. (1998) were among the first to study the impact of masking on ERP components in the AB. They found an almost completely suppressed P3b during the AB for masked trials at short lags. However, when T2 was

not-masked, the P3b component was not suppressed in terms of amplitude, but the onset of the P3b was delayed at lag 3 compared to lag 7 despite accuracy levels suggesting no AB (Vogel et al., 1998; see also Sessa, Luria, Verleger, & Dell'Acqua, 2007). These findings were particularly important because they suggested that the absence of an AB effect on accuracy of report of T2 could not be interpreted as an absence of AB interference on the processing of T2. The delay of the P3b provided strong evidence for either an interruption or a slowing of encoding of T2 resulting from concurrent processing of T1 (Jolicœur & Dell'Acqua, 1998). Vogel et al. (1998) argued that the perceptual representation of T2 could be sustained for a relatively long period of time if it was not followed by another item. This representation was therefore still available when the encoding of T1 was completed, allowing subsequent but delayed processing of T2. If a mask (a distractor) followed T2, however, its perceptual representation was lost and/or overwritten by the subsequent stimulus before encoding processes devoted to T1 were available for T2. This made the last target unavailable for conscious report. Interestingly, Jolicœur and Dell'Acqua (1998) reported several experiments in which visual stimuli that had to be encoded for later report (at the end of each trial) were followed by a second stimulus that required an immediate speeded response. Response times increased as the delay between these two stimuli was reduced. This finding suggested that encoding visual stimuli for later report was sufficient to delay or slow the processing of trailing stimuli (see also Jolicœur, Dell'Acqua, & Crebolder, 2000). The delay of P3b onset at short lag in the AB is consistent with the increases in response time reported by Jolicœur & Dell'Acqua (1998) or Jolicœur et al. (2001; see also Dell'Acqua, Jolicœur, Vespignani, & Toffanin, 2005).

Models including attention-related explanations do not all agree on the cause of the attentional disruption, however, a subset include distractor or masking

related impacts on attention. As for models of masking on the other hand, none appear to address possible links between the impacts of a mask on attention. One exception to this general statement pertains to the role of attention in the 4-dot masking phenomenon where this type of mask is more effective if attention is distributed among several targets (Dell'Acqua, Pascali, Jolicœur, & Sessa, 2003; Enns & Di Lollo, 1997).

## 1.2 Visual Spatial Attention

Visual spatial attention is the ability to search within a visual scene, and find the information which is necessary to our goal, and suppress the information that is unnecessary. Visual attention is usually assessed through a visual search task, which asked the subject to find a target among distractors in a visual scene. Although there are several models which try to explain how visual attention works, in order to be the most coherent as possible with the experimental evidence of the next chapters, only specific fundamental aspects of visual attention will be explained in this section.

There are two main ways to explore the visual space in front of us: one is called "overt search", and it is done by moving the eyes; the "covert search", instead, is done with no gaze shift but with attentional shift (Posner, 1980; 2016), and it is the one investigated in Chapter 3 and Chapter 4. More precisely, the subject of my investigation in this context is how visual attention is deployed in space.

According to Desimone and Duncan (1995), the information embedded in the visual field became cortical representation in visual areas. Because of the retinotopical structure of the visual areas, when two stimuli are presented they both activate them receptive field, and create competition for cortical representation (Desimone, 1998). This competition is controlled (and solved) by brain mechanisms: for instance, a stimulus which is more salient (i.e., more luminant, or more novel) than

another can win the representation competition through a bottom-up attentional process. Differently, the representation of a stimulus that is relevant for some reason (i.e., the experimental task) can be enhanced through a top-down process which also involved a strong influence of the frontal areas (i.e., Zanto, Rubens, Thangavel, & Gazzaley, 2011), and then win the cortical competition. This top-down mechanism is thought to actively maintain in visual working memory the representation of the target to-be-search, and match it with the candidates items presented in a visual scene (Desimone & Duncan, 1995). In line with this hypothesis, it has been showed that neurons in inferotemporal cortex of monkeys (Chelazzi, Duncan, Miller, & Desimone, 1998; Chelazzi, Miller, Duncan, & Desimone, 1993) exhibit a higher firing rate when, during a visual search task, it is presented a target cued at the begin of the trial.

According to Wolfe (1994), preattentive processes direct attention to items which can be good candidates to be the target. The parallel existence of bottom-up and top-down mechanisms allow the visual system to create a ranking of visual items based on their attentional priority. Attention will be directed the item with the highest priority, and if this will be not the target, attention will move on the next candidate. When a search task is simple, for instance when a subject is asked to find a blue item among green items, attention can be focused to the target with no particular effect of the distractors. In contrast, there are cases in which attentional guidance is possible, for instance searching for conjunction of features, there are not a single candidate to be target. If the target is a red circle among other red shapes and other color circles, all the circle and all the red items will be activated, and then attention is oriented to each of the possible candidates.

A widespread method to investigate the deployment of visual attention is the EEG and the ERP technique. When a lateral target is presented among distractors, an

ERP called N2pc (Luck & Hillyard, 1994a; b) is elicited about 200 ms after the attended stimulus onset. The N2pc is a greater negativity over the hemisphere contralateral to a lateral target, compared to the ipsilateral response, and it has a parieto-occipital scalp distribution. This component is derived by subtracting the average activity ipsilateral to a target (i.e., left posterior scalp activity when a target is presented on left and right posterior activity when a target is presented on right) from the average activity contralateral to a target (i.e., right posterior scalp activity when a target is presented on left and left posterior activity when a target is presented on right).

Of note, the items presented in the visual field must be balanced in terms of number and luminance. In contrast, when the competition among target and distractors is reduced by –for instance– a decrease of distractors' luminance, the *locus* of selection is anticipated to the N1 time domain, eliciting a lateralized ERP called N1pc, which looks as an anticipated N2pc (Wascher & Beste, 2010).

At the neural level, the N2pc reflects a circuitry which includes parietal and occipito-temporal areas. The activation of these areas are actually coherent with visual search task dynamics, since parietal lobes seems to be involved in attentional control (Chelazzi et al., 1993; Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991; Heinze et al., 1994; Mountcastle, Andersen, & Motter, 1981) and occipito-temporal areas are involved in the implementation of attentional selection (Chelazzi et al., 1993; Corbetta et al., 1991; Heinze et al., 1994; Moran & Desimone, 1985). According to this evidence, the parietal lobes could be responsible of an initial attentional shift toward the task-relevant item (Corbetta, Shulman, Miezin, & Petersen, 1995), and the occipito-temporal lobes could filter the irrelevant information which surround the to-be-attend item (Heinze et al., 1994).

In the last 25 years, the N2pc has been deeply investigated to further the knowledge in regard of visual attention. Mazza and Caramazza (2011) found that the N2pc amplitude is sensitive to the number of presented target in an enumeration visual search task: the greater the number of target items presented, the greater the N2pc amplitude. Nevertheless, when the task was to detect the presence of at least one target, a greater number of target did not elicit a greater N2pc. This suggest that this component reflect a top-down mechanism strongly related to the task demand. Of note, a visual inspection of Figure 4 by Mazza and Caramazza (2011), suggests that when the number of presented target increases, the contralateral portion of the N2pc is more negative, although the ipsilateral one remain the same regardless the number of presented targets. Nevertheless, Kiss, Jolicœur, Dell'Acqua, and Eimer (2008) found that when a subject was searching among homogeneous distractors (circles) for a specific shape (square), when another shape (45° tilted square) was presented instead of the target, it elicited a N2pc. Differently, when the target was absent and a different color distractor was presented, no N2pc was found. This suggest that in some specific cases the N2pc reflects a bottom-up process: when a pop-out target was presented, if it is defined by the target dimension (i.e., the shape) but not the same feature (i.e., a square) it automatically produce an attentional capture. Differently, if the pop-out stimulus is characterized by a different characteristic it does not capture attention.

It has been argued that the N2pc reflects two functions that orient attention in the visual space: target selection and distractor suppression (Eimer, 1996; Luck & Hillyard, 1994b). Hickey, Di Lollo, and McDonald (2009) found two sub-components of the N2pc, basically comparable to the contralateral and ipsilateral waves: a Target Negativity (Nt) and a Distractor Positivity (Pd). The authors suggested that the Nt (contralateral to a target) reflect the target selection mechanism, and the Pd (ipsilateral

to a distractor) reflect the target suppression mechanism. This view is quite in line with a work by Mazza, Turatto, and Caramazza (2009). In this study, the authors found an increased N2pc amplitude when a higher number of distractors was displayed, compared to a smaller number, when searching for a singleton target. By visually inspecting separately the contralateral and the ipsilateral ERP which subtracted create the N2pc, it is clear that a greater number of distractors drove a positive shift of the ipsilateral portion of the N2pc, while the contralateral wave has the same amplitude regardless of the number of distractors (Mazza, Turatto & Caramazza, 2009, Figure 2A).

Differently than the N2pc, when a stimulus is presented on the horizontal midline of the visual field, a bilateral N2 component, called N2pb, is elicited (Luck & Hillyard, 1994b; Simson, Vaughan, & Walter, 1976). This component has been study with less concern compared to the N2pc, and it is thought to reflect stimulus categorization processes, and it is larger for less frequent targets (Lange, Wijers, Mulder, & Mulder, 1998; Okita, Wijers, Mulder, & Mulder, 1985; Potts & Tucker, 2001). N2pb is calculated as the average of both left and right posterior electrodes activity. Of note, a target presented aligned with the sagittal midline of a subject does not elicit an unbalance between the two posterior scalp hemispheres, as N2pc does.

## 1.3 Merging the Dynamics of Visual Attention

There are some studies which link the mechanisms which drive visual spatial attention with the temporal dynamics of attention of the AB phenomenon. Namely, the N2pc modulations are monitored to assess whether is there any disruption of the visual search ability within the AB. In general, N2pc presented a smaller amplitude at short relative to long lags during the AB (Akyürek et al., 2010; Dell'Acqua

et al., 2006; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2006). All the previous experiments examining the N2pc in AB paradigms, however, contained a post-T2 mask. In tasks other than the AB, masked targets seemed to elicit an N2pc. This was the case for four-dot masking (Prime, Pluchino, Eimer, Dell'Acqua, & Jolicœur, 2011), metacontrast masking under certain conditions (Ansorge & Heumann, 2009; Ansorge, Horstmann, & Worschech, 2010; Jaśkowski, van der Lubbe, Schlotterbeck, & Verleger, 2002), and for pattern masking (Robitaille & Jolicœur, 2006). Again, however, these experiments did not directly compare masking to conditions where no mask was present. Only Robitaille and Jolicœur (2006) compared trials where a pattern mask (another alphanumeric character) was presented to when no mask was presented and found no masking effect on the N2pc. Although manipulations of masking have been used a number of times in the AB paradigm to study how processing of information unfolds over time, less is known about how masking affects the deployment of visual spatial attention in this paradigm. Despite that, a sizable number of masking theories exist but little place is given to attention in these proposed mechanisms. The re-entrant perceptual hypothesis (Enns & Di Lollo, 2000) suggests that less attention will accentuate masking but does not broach the impact of the mask itself could have on attention.

If an attentional perturbation in the temporal domain (i.e., AB) exerts modulatory effects on the efficiency in visuo-spatial attention allocation (i.e., in visual search) (Akyürek et al., 2010; Dell'Acqua et al., 2006; Jolicœur, Sessa, Dell'Acqua, & Robitaille, 2006) then this would be ground to parsimoniously suggest that these hypothetically distinct limits subtended a common cause. By following this rationale, next studies first isolated the attentional dynamics reflected by ERP components in an AB task (Chapter 2). After that, another study (Chapter 3) combined an AB task with

visual spatial attention, in order to assess whether the same AB-related ERP modulations found in Chapter 2 are still present also when visual information is presented in a parafoveal position and visual search abilities are required to find them. Of note, additional ERPs which reflect the deployment of visual spatial attention have been also investigated to understand whether their AB-related modulation are comparable to the other attentional ERP modulation. In order to explore more in deep the link between foveal and parafoveal attention, a last experimental section (Chapter 4) followed a different approach. Namely, through two experiments, visual attentive ERP component associated to the deployment of visual attention elicited by midline and lateral presented targets are compared, in order to observe from another perspective whether there are differences between the cognitive mechanisms which driven the deployment of attention. Based on previous results, AB should modulate frontal and posterior ERP components related to attention such as P3a and P3b (Gross et al., 2004). This modulation should be present both when the items of the RSVP are presented in the same position (Chapter 1) and also when some of them are presented eccentrically (Chapter 2, e.g., Dell'Acqua et al., 2006). Finally, there are no previous evidence which gives the possibility to predict a specific outcome of Chapter 4's experiments. Nevertheless, given the overlap between foveal and parafoveal visual attention found in past (e.g., Jolicœur et al., 2006), by comparing the bilateral posterior ERP elicited by a midline target with the contralateral and ipsilateral activities elicited by a lateral target it would be possible to infer more directly the origins of the functional and neural overlap between foveal and parafoveal attention.

# Chapter 2

Part of the content presented in this chapter has been described in the following published articles:

Dell'Acqua, R., Doro, M., Dux, P. E., Losier, T., & Jolicœur, P. (2016). Enhanced frontal activation underlies sparing from the attentional blink: Evidence from human electrophysiology. *Psychophysiology*, 53(5). https://doi.org/10.1111/psyp.12618

Dell'Acqua, R., Dux, P. E., Wyble, B., Doro, M., Sessa, P., Meconi, F., & Jolicœur, P. (2015). The attentional blink impairs detection and delays encoding of visual information: Evidence from human electrophysiology. *Journal of Cognitive Neuroscience*, 27(4). https://doi.org/10.1162/jocn_a_00752

# 2.1 Experiment 1a

## 2.1.1 Introduction

In the General Introduction evidence regarding the brain activation and circuitry involved in the AB phenomenon have been mentioned. Basically, the AB is associated to a number of connections between the frontal and parietal brain areas. Although these studies have helped isolate processing to specific attentional circuits within the brain, it is not known how these circuits interact to produce the AB. For example, it could be the case that the AB slows down the detection of T2, allowing it to be overwritten by trailing stimuli. On the other hand, it could be that T2 detection operates unimpaired, but that the ensuing attentional deployment is less effective at processing the required information.

Using a behavioral approach, Dux, Wyble, Jolicœur, and Dell'Acqua (2014) recently examined whether the AB delays target detection, memory encoding or both, and whether the AB is a T1-locked phenomenon or a manifestation of an attentional perturbation induced by distracting information trailing T1 (Dux & Marois, 2009; Martens & Wyble, 2010). Specifically, I assessed if the encoding load within a temporal attention episode/window influenced report of stimuli appearing in subsequent episodes. In a three-target RSVP paradigm, T1 and T2 always appeared sequentially, creating lag 1 sparing conditions, but T3 appeared at varying lags relative to T2. When T1 and T2 were correctly reported a much larger AB was observed for T3 compared with when only T1 or T2 was correctly reported. Thus, target load, within an attentional window and independent of distractors, influenced the AB magnitude. In addition, there was no difference between the AB observed when either T1 or T2 was missed in three-target trials relative to the AB found in standard two-target trials, suggesting the missing one

stimulus preceding T3 had an all-or-none effect on the AB observed in three-target trials.

Here, the approach of Dux et al. (2014) was combined with ERPs to investigate the influence of target load on the interplay between detection and encoding stages and the role they play in operations linked to the AB and conscious visual perception. Specifically, I used a variant of the three-target RSVP paradigm introduced by Dux et al. (2014) to explore the impact target processing load has on P3a and P3b components elicited by the last target in RSVP streams. The design differs from that employed by Dux et al. (2014) in two important aspects. First, the last target embedded in RSVP was not trailed by distractors so as to allow us to observe fully fledged P3b and P3a responses to this stimulus. As explained in the General Introduction, little or no behavioral AB is typically observed for unmasked targets (e.g., Giesbrecht & Di Lollo, 1998; Jannati, Spalek, & Di Lollo, 2011; Jannati, Spalek, Lagroix, & Di Lollo, 2012; Ptito et al., 2008). However, under these conditions, the underlying neural process evoked by T1 that produces the behavioral AB for a masked T2 should still occur, and it is this underlying neural process that is the subject of this inquiry. This approach has the benefit of allowing to capture the modulatory influence of the manipulations on the neural correlates of target processing as quantifiable parametric changes in the latency and amplitude of the P3a/b components. Second, target-present trials in the conditions of interest were intermixed in the present experimental context with target-absent trials (i.e., trials in which the last target was replaced with a distractor), so as to isolate unequivocal P3a/b responses reflecting last target detection and encoding uncontaminated by activity elicited by to-be-ignored distractors and/or by phasic oscillations induced by the RSVP rhythm (Hanslmayr et al., 2011; Ptito et al., 2008).

## 2.1.2 Method

### Participants

Forty students at the University of Padua (23 women) participated in the experiment after giving informed consent. Their mean age was 24.8 years (SD = 4.6 years), and all had normal or corrected-to-normal visual acuity and no history of neurological disorders.

### Stimuli

The stimuli were the 22 letters of the English alphabet remaining after excluding B, I, O, Z, and the digits 2–9. The stimuli were displayed in light gray (34 cd/m2) Romantri font against a black (6 cd/m2) background. Luminance measurements were performed using a Minolta LS-100 chroma-meter (Ramsey, NJ). Stimuli appeared on a 19-in. CRT monitor running at 60 Hz, placed at a viewing distance of approximately 60 cm from the participant, controlled by an i686 IBM-clone computer running MEL 2.0 software. RSVP streams were composed of distractor digits randomly selected from the available set, plus two or three different target letters (T1, T2, and T3) presented in various positions in the stream (see Design and Procedure section). Identical distractor digits in the RSVP stream were always separated by a minimum of three different stimuli. Each stimulus was displayed for 84 ms and was immediately replaced by the next stimulus (ISI = 0 ms). The lag between pairs of critical targets (i.e., T1–T2 lag in the two-target RSVP streams or T2–T3 lag in three-target RSVP streams) was manipulated by varying the number of distractors between T1 and T2 or between T2 and T3. The number of distractors preceding T1 was varied randomly across trials from 6 to 11, and each RSVP stream ended with T2 in two-target RSVP streams or T3 in three-target RSVP stream, which were replaced by a digit distractor in the same

position when the last target was not displayed. All stimuli were scaled to fit in a central, square portion of the monitor measuring 1.0° × 1.0° of visual angle.

### Design and Procedure

A schematic representation of the experimental design is illustrated in the upper portion of Figure 2.1. In three-target RSVP streams, T1 and T2 were always consecutive items. The lag between T1 and T2 in two-target RSVP streams and between T2 and T3 in three-target RSVP streams was manipulated by presenting 2 (lag 3, SOA = 252 ms) or 8 (lag 9, SOA = 756 ms) distractors between these targets.

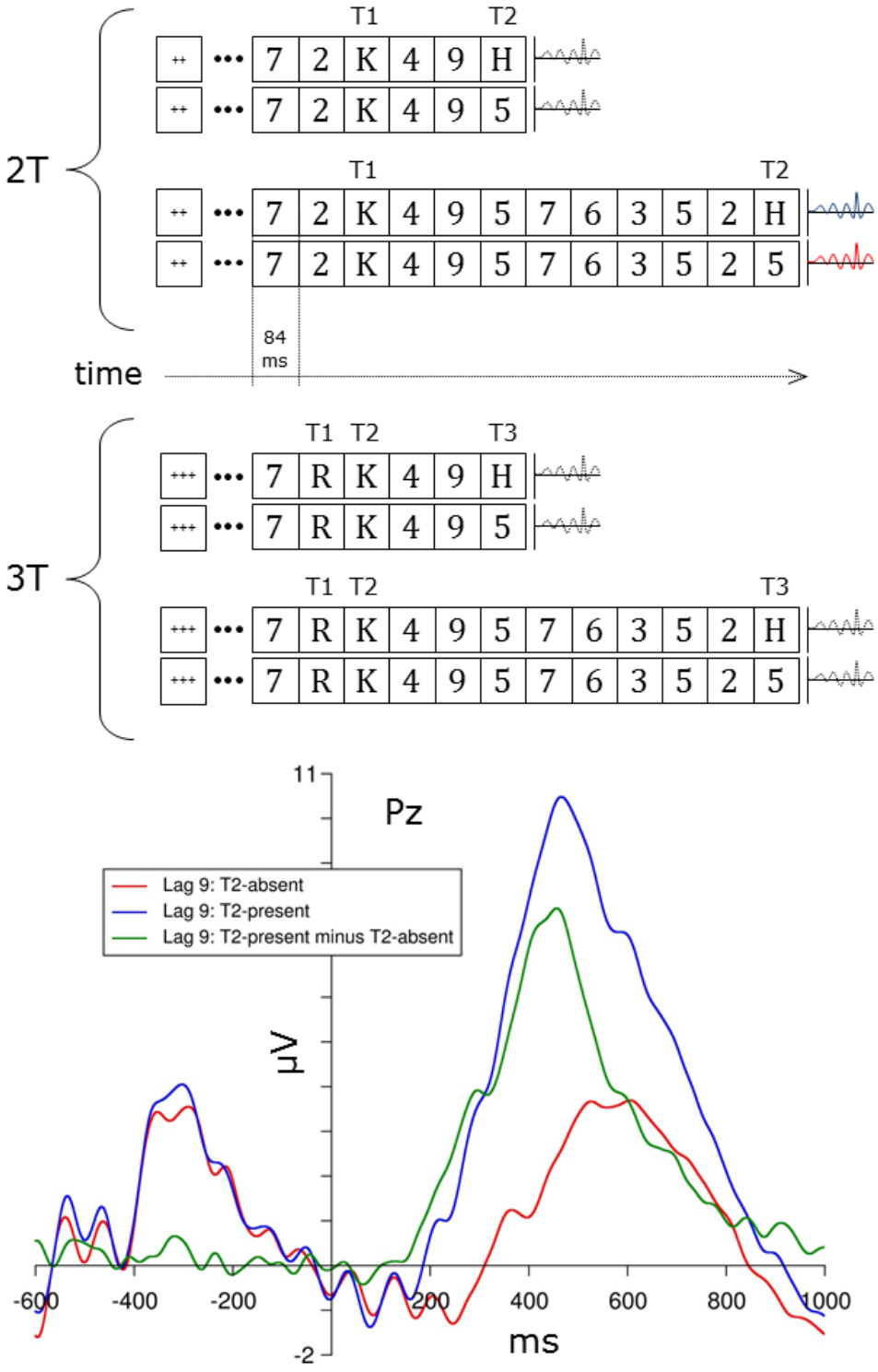


**Figure 2.1.** Top: Gantt diagrams: Design of the present experiment. In target-present trials, two letters or three letters were embedded among digit distractors in two-target (2T) or three-target (3T) RSVP streams. Both RSVP streams began with the presentation of a number of centrally displayed "+" signs equal to the number of targets included in the RSVP streams, and each character was displayed for 84 ms,

immediately followed by the next character. In half of the trials, the last target letter was replaced with a digit distractor, generating a corresponding target-absent RSVP stream. In three-target RSVP streams, the first and second targets (i.e., T1 and T2) were always consecutive letters. T1 and T2 in two-target RSVP streams and T2 and T3 in three-target RSVP streams were separated by two distractors (i.e., at lag 3) or eight distractors (i.e., at lag 9). When present, the last target in both RSVP streams was never trailed by a digit distractor. The dotted curvilinear function trailing the last character in each RSVP stream indicates (the onset of) the target monitored for ERP responses in the present experiment. Bottom graph: Illustration of the subtraction approach used in the present context to isolate difference ERPs reflecting mental operations engaged for last target processing. In the graph, T2-locked ERP functions observed at Pz in two-target trials, at lag 9, in the T2-present (blue) and T2-absent (red) conditions. Corresponding colors can be seen in the Gantt diagrams above referred to the condition of interest. T1-locked P3 responses of equal amplitude peaking at about −300 ms before T2 precede T2-locked P3 responses elicited in target-present (red) and target-absent (blue) trials. P3 responses to the last distractor in target-absent trials were non-nil, had a later onset latency, and were generally of smaller amplitude than P3 responses observed in T2-present trials. Note also that both T2-present and T2-absent trial ERPs show clear symptoms of stimulus-locked visual evoked potentials in the form of entrained sinusoidal activity at about 12 Hz, corresponding to the rate of RSVP stimulation. To derive "pure" target-related ERP activity, target-absent ERP responses were subtracted from target-present ERP responses in each condition of the experimental design (see text). The resulting difference ERP function is shown in green in the graph.

Each participant performed 648 trials, organized into 18 blocks of 36 trials each. Each lag appeared an equal number of times in each block, but their order was pseudorandomized, with the constraint that no more than three consecutive trials could be displayed at the same lag. The last target in two-target (i.e., T2) and three-target (i.e., T3) RSVP streams was displayed on half of the trials (henceforth, target-present trials) within each block and replaced with a digit distractor in the same position on the other half of trials (henceforth, target-absent trials). In four trials in each block, a target was presented in the last stream position, with no preceding targets. These trials were not analyzed in this study. Half of the participants started with nine consecutive blocks of two-target RSVP streams, followed by nine consecutive blocks of three-target RSVP streams. The opposite order applied for the other half of the participants.

Each trial began with the presentation of a number of horizontally aligned plus signs in the center of the monitor denoting the number of targets that would appear in the forthcoming RSVP stream (i.e., two or three plus signs). Pressing the spacebar initiated a trial, causing the plus signs to disappear, and the RSVP to start 800 ms later. A question was displayed 800 ms after the end of the RSVP stream, inviting report of

the targets by pressing the corresponding keys on the keyboard. Participants were instructed to report all letters in the RSVP streams, with no emphasis on their order or response speed. Feedback on an incorrectly reported target was provided at the end of each trial by replacing the plus sign in the position congruent with target order (from left to right, T1, T2, and T3 when present) with a minus sign. Experimental data were collected after exposing participants to no less than 20 RSVP streams for practice in each of two-target and three-target conditions.

### 2.1.3 EEG/ERP Recordings and Preprocessing

EEG activity was recorded continuously from 28 active electrodes (Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, C3, C4, Cz, CP1, CP2, CP5, CP6, P3, P4, Pz, O1, O2, Oz, T7, T8, TP9, PO9, PO10, P7, P8 sites) placed on an elastic ActiCap (Brain Products, München, Germany), referenced to the left earlobe. HEOG activity was recorded bipolarly from electrodes positioned on the outer canthi of both eyes. VEOG activity was recorded bipolarly from two electrodes, above (Fp1) and below the left eye. Impedance at each electrode site was maintained below 5 KΩ. EEG, HEOG, and VEOG activities were amplified, filtered using a bandpass of 0.016–80 Hz, digitized at a sampling rate of 500 Hz, and referenced offline to the average of the left and right earlobes. Independent components analysis (ICA) was used to identify blink and saccade components in the continuous EEG recordings and remove them from the data (Delorme & Makeig, 2004; Jung et al., 2000). The corrected EEG was high-pass filtered at 0.1 Hz and low-pass filtered at 20 Hz and then segmented into 1100 ms epochs starting 100 ms before the onset of the last character in the RSVP stream and ending 1000 ms after and baseline-corrected using the mean activity in the interval [−100, 0] ms. To ensure no residual artifacts remained on the EOG channels, each segment was

examined in the interval [−100, 1000] ms relative to the onset of the last item in the RSVP stream for voltage deviations greater than 80 μV in any period of 150 ms for the VEOG difference waveform or a deviation greater than 45 μV in any 300 ms period for the HEOG difference waveform. Segments with residual ocular artifacts were removed from the data set. EEG channels were flagged when the signal exceeded ±100 μV anywhere in the analysis segment. If a segment had seven or fewer flagged data channels, these channels were interpolated using a spherical spline interpolation algorithm in EEGLAB (Delorme & Makeig, 2004), for that segment. The critical analyses were carried out on separate ERP waveforms for each condition (two-target vs. three-target and lag 3 vs. lag 9) considering only trials associated with the correct report of all displayed targets and generated by subtracting the ERP waveforms elicited by distractors replacing T2 in two-target (target-absent) trials or T3 in three-target (target-absent) trials from the ERP waveforms elicited by the corresponding target-present trials (i.e., T2 in two-target trials or T3 in three-target trials). These difference waveforms isolate the response to a target character in the final RSVP position –T2 or T3, in two-target and three-target trials, respectively– from the response to a nontarget character in the same position while reducing to nil EEG oscillations in phase with the rate of presentation of RSVP items (about 12 Hz; alpha band). An illustration of the results of the present subtractive approach is reported in the lower portion of Figure 2.1.

The mean amplitude of the subtracted P3a and P3b components was quantified as the mean value in a 150 ms window centered on the peak of the waveform in individual grand averages computed at Fz and Pz, respectively. As noted above, these electrodes have previously been linked with peak amplitude of the P3a and P3b, respectively (e.g., Polich, 2003). The mean latency of the subtracted P3 components at the same recording sites was estimated using the jackknife approach (Brisson &

Jolicœur, 2007; Kiesel, Miller, Jolicœur, & Brisson, 2008; Ulrich & Miller, 2001) and deriving individual values with the solution proposed by Smulders (2010). Latency values were calculated as the time-point when individual jackknife waveforms reached 75% of the peak amplitude. The Greenhouse–Geisser correction for nonsphericity was applied when appropriate.

## 2.1.4 Results

Behavior

Separate ANOVAs were performed on the mean proportion of correct report for each target-contingent of the correct report of preceding targets –as a function of number of presented targets within the RSVP (two-target trials vs. three-target trials) and lag (3 vs. 9) as within-subject factors. Only target-present trials were considered in the analyses (see Table 2.1).

| | *Trial Type* | *Lag* | |
|---|---|---|---|
| *Target* | | *3* | *9* |
| *p*(T1) | Two-targets | .94 | .95 |
| | Three-targets | .81 | .82 |
| *p*(T2\|T1) | Two-targets | .95 | .96 |
| | Three-targets | .94 | .94 |
| *p*(T3\|T2^T1) | Three-targets | .86 | .96 |

**Table 2.1.** Mean probability of correct report of each target in each condition.
Values in the table are contingent on the correct report of preceding target(s) (e.g., T2|T1 indicates the probability of T2 correct response when T1 is report correctly.

On average, T1 report was superior in two-target trials relative to three-target trials, $F(1,39) = 256.7$, $\eta_p^2 = .868$, $p < .001$, and this effect was constant across lags, $F < 1$. An ANOVA was carried out to compare T2 report in two-target trials and T3 report in three-target trials, as a function of lag. There was a main effect of Number of targets, $F(1,39) = 34.7$, $\eta_p^2 = .466$, $p < .001$, a main effect of lag, $F(1,39) = 17.3$, $\eta_p^2 = .307$, $p < .001$, and a significant interaction between these factors, $F(1,39) = 15.9$, $\eta_p^2 = .290$, $p < .001$. False-discovery rate (FDR, Benjamini & Hochberg, 1995) corrected t tests indicated that lag effects were absent on T2 in two-target trials, $t < 1$, whereas T3 report was worse at lag 3 relative to lag 9 in three-target trials, $t(39) = 18.6$, $p < .001$, that is, a small but reliable AB effect was detected in spite of the absence of a distractor trailing T3. These findings converge with prior studies reporting small but reliable AB effects even when the last target is not masked by trailing distractors (Giesbrecht & Di Lollo, 1998; Jannati et al., 2011, 2012; Ptito et al., 2008; Sessa et al., 2007).

ERP

The various automated artifact screening procedures resulted in the exclusion of 2.4% of the segments. For most participants, less than 1% of the data were excluded. Three participants had exclusion rates between 18% and 22%. Visual inspection of their ERPs suggested their results were comparable to those of the other participants, and so their data were included in the final analyses. Thus, the final sample included all 40 participants tested in the experiment.

P3b

Difference (target-present minus target-absent; see EEG/ERP Recordings and Preprocessing section) P3 response waveforms for two-target and three-target trials at each lag are shown in Figure 2.2 for electrode Pz. Key for this study, the amplitude of P3b responses was largest and largely overlapping at lag 9 for two-target and three-target trials but was delayed and attenuated at lag 3. This target-load effect on P3b was substantially more evident in three-target trials than in two-target trials.

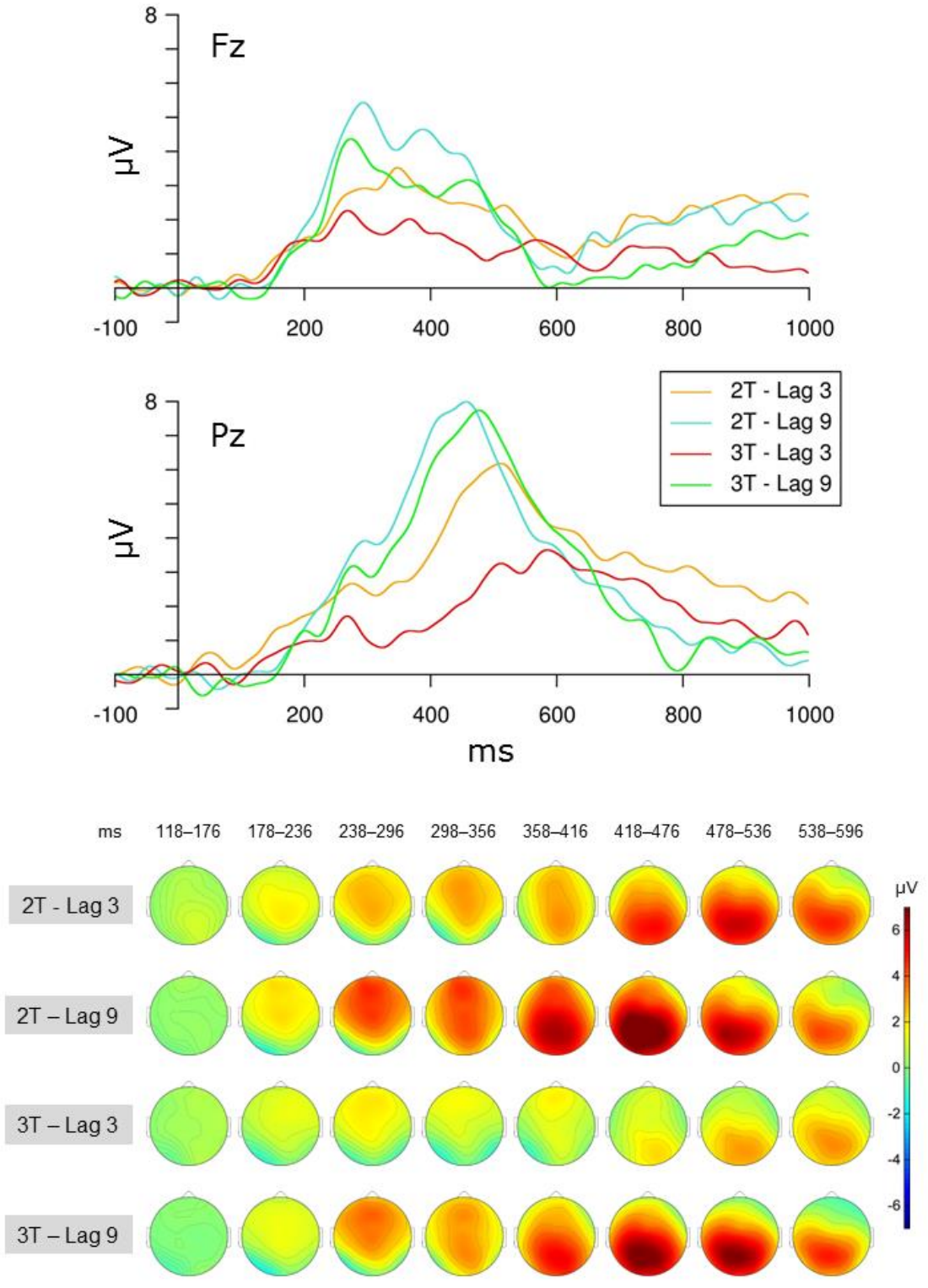


**Figure 2.2.** Results of ERP analysis. Top graphs: Difference (target-present minus target-absent) ERP responses in two-target and three-target trials, plotted as a function of lag (3 vs. 9) observed at Pz (P3b) and Fz (P3a). Bottom scalp plots: Time course of voltage topographic scalp distribution in each of the four main conditions on the experimental design.

The main analyses were performed at Pz, at the peak of the scalp distribution of the P3b component. At lag 9, the mean amplitudes of the P3b were 7.35 μV in two-target trials and 7.13 μV in three-target trials. At lag 3, the mean amplitudes of the P3b were 5.7 μV in two-target trials and 3.35 μV in three-target trials. Individual means for each of these measures were submitted to an ANOVA with number of presented targets (two-target vs. three-target) and lag (3 vs. 9) as within-subject factors. The ANOVA confirmed that the P3b amplitude was larger at lag 9 than at lag 3, $F(1,39) = 27.6$, $\eta_p^2 = .411$, $p < .001$, and larger in two-target than in three-target trials, $F(1,39) = 9.0$, $\eta_p^2 = .192$, $p < .005$. Furthermore, lag and number of targets interacted, with a larger attenuation of P3b amplitude in three-target trials relative to two-target trials at lag 3 than at lag 9, $F(1,39) = 6.6$, $\eta_p^2 = .149$, $p < .02$. To characterize the interaction further, the amplitude of the P3b across two-target and three-target trials were compared in a separate ANOVA considering only trials at lag 9 and found no significant difference, $F < 1$, $p = .72$. At lag 3, in contrast, the amplitude of the P3b was larger in two-target trials than in three-target trials, $F(1,39) = 14.1$, $\eta_p^2 = .266$, $p < .001$.

Individual latency values of P3b responses to the last target in the streams (i.e., T3 in three-target trials or T2 in two-target trials) were submitted to an ANOVA using the same model as for the mean amplitudes analyses reported above. At lag 9, the mean latencies of the P3b response were 387 ms in two-target trials and 393 ms in three-target trials. At lag 3, the mean latencies of the P3b response were 434 ms in two-target trials and 483 ms in three-target trials. P3b latency was longer at lag 3 than at lag 9, $F(1,39) = 38.7$, $\eta_p^2 = .199$, $p < .001$, and longer in three-target trials than in two-target trials, $F(1,39) = 9.4$, $\eta_p^2 = .567$, $p < .004$. Importantly, the difference in P3b latencies between two-target and three-target trials was larger at lag 3 than at lag 9, producing a significant interaction between number of targets and lag, $F(1,39) = 4.5$, $\eta_p^2 = .102$, $p <$

.05. Two-target trials and three-target trials were also compared separately at each lag observing no difference in P3b latencies between two-target trials and three-target trials at lag 9, $F(1,39) = 1.6$, $p > .2$, but a clear significant difference in P3b latencies between two-target trials and three-target trials at lag 3, $F(1,39) = 8.7$, $\eta_p^2 = .189$, $p = .005$.

P3a

The main analyses were performed at Fz, at the peak of the scalp distribution of the P3a component. At lag 9, the mean amplitudes of the P3a response were 4.8 μV in two-target trials and 3.9 μV in three-target trials. At lag 3, the mean amplitudes of the P3a response were 2.9 μV in two-target trials and 1.9 μV in three-target trials. Individual means for each of these measures were submitted to an ANOVA with number of targets (two-target vs. three-target) and lag (3 vs. 9) as within-subject factors. The ANOVA indicated a larger P3a amplitude at lag 9 than at lag 3, $F(1,39) = 27.0$, $\eta_p^2 = .413$, $p < .001$, and a larger P3a amplitude in two-target trials than in three-target trials, $F(1,39) = 6.4$, $\eta_p^2 = .147$, $p < .02$. There was no interaction between number of targets and lag in the analysis of P3a amplitude values recorded at Fz, $F < 1$, $p > .9$.

The mean P3a latency was 190 ms, and there were no significant differences across conditions in an ANOVA that considered number of targets and lag as factors, all $F$s < 1, all $p$s > .6, confirming what can be observed in Figure 2.2, namely, that, contrary to P3b, P3a latency was not subject to AB-induced perturbations.

ICA of ERPs

To further explore the interaction of detection and encoding processes in the AB, an ICA was used to decompose the ERPs into separate components using the algorithm implemented in EEGLAB (Delorme & Makeig, 2004). This was done to provide a more faithful depiction of the ERP results by decomposing P3a and P3b waveforms into maximally spatiotemporally independent signals available in the channel data and minimize to nil the influence of their potential overlap/summation on the interpretation of the above findings. The difference waves for the four main conditions in the experiment (two-target vs. three-target trials × lag 3 vs. lag 9) for each participant were first analyzed using singular value decomposition to determine the dimensionality of the signal subspace containing most of the relevant event-related activity. A scree plot of the singular values showed a clear break after the first three components, leading us to retain the first four dimensions, which accounted for 54.3% of the variance. The ICA analysis was thus restricted to this subspace of the signal space using an initial PCA. The ICA analysis isolated two components of the P3 family, namely, a later posterior component (Component I, P3b) and an earlier anterior component (Component II, P3a). The grand-averaged time courses and relative topographies for these two components, for the four main conditions of the present experimental design, are shown in Figure 2.3. The time course for the two components of interest was reconstructed in the ICA analysis for each participant and condition and submitted amplitude and latency measures to the same type of ANOVA models as were used for the analyses of the original ERPs.

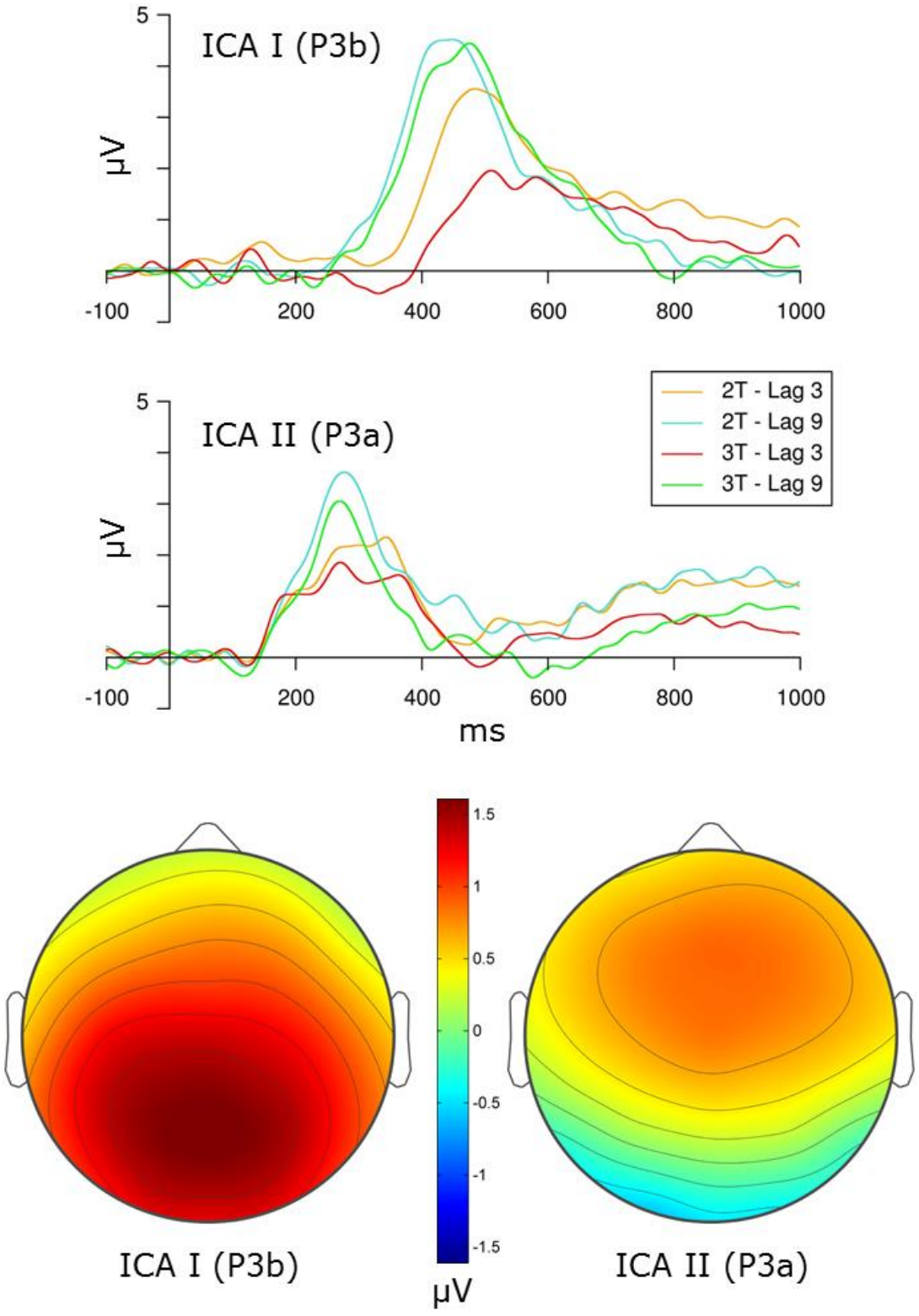


**Figure 2.3.** Results of ICA decomposition. Top graphs: ERP functions corresponding to ICA-P3b and ICA-P3a isolated using ICA. Bottom scalp plots: Scalp topographic maps of ICA-P3b (left) and ICA-P3a (right).

ICA-P3b

At lag 9, the mean amplitudes of the ICA-P3b were 4.1 μV in two-target trials and 3.94 μV in three-target trials. At lag 3, the mean amplitudes of the ICA-P3b were 3.1 μV in two-target trials and 1.7 μV in three-target trials. ICA-P3b amplitude was larger at lag 9 than at lag 3, $F(1,39) = 32.9$, $\eta_p^2 = .466$, $p < .001$. Furthermore, ICA-P3b amplitude was larger in two-target trials than in the three-target trials, $F(1,39) = 13.7$, $\eta_p^2 = .269$, $p < .001$. The difference between two-target and three-target trials was larger at lag 3 than at lag 9, which produced a significant interaction between number of targets and lag, $F(1,39) = 7.9$, $\eta_p^2 = .174$, $p < .008$. Two-target and three-target trials was also compared separately at each lag. At lag 9, ICA-P3b amplitudes for two-target and three-target trials were equivalent, $F < 1$, $p > .7$. In contrast, at lag 3, ICA-P3b amplitudes for two-target and three-target trials differed substantially, $F(1,39) = 14.1$, $\eta_p^2 = .264$, $p < .0006$.

At lag 9, the estimated mean ICA-P3b latencies were 390 ms for two-target trials and 396 ms for three-target trials. At lag 3, ICA-P3b latencies were 436 ms for two-target trials and 480 ms for three-target trials. This pattern of latencies produced a significant effect of lag, $F(1,39) = 57.65$, $\eta_p^2 = .674$, $p < .001$, reflecting the general earlier latency of ICA-P3b components of two-target and three-target trials at lag 9 relative to lag 3. ICA-P3b latency was prolonged in three-target trials relative to two-target trials, $F(1,39) = 16.0$, $\eta_p^2 = .295$, $p < .001$, the more so at lag 3 compared with lag 9, $F(1,39) = 5.7$, $\eta_p^2 = .138$, $p < .03$. Two-target and three-target trials was compared separately at each lag. At lag 9, ICA-P3b latencies for two-target and three-target trials were equivalent, $F(1,39) = 2.5$, $p > .12$. In contrast, at lag 3, ICA-P3b latencies for two-target and three-target trials differed substantially, $F(1,39) = 13.9$, $\eta_p^2 = .264$, $p < .001$.

ICA-P3a

At lag 9, the mean amplitudes of the ICA-P3a were 2.13 μV in two-target trials and 1.86 μV in three-target trials. At lag 3, the mean amplitudes of the ICA-P3a were 2.26 μV in two-target trials and 1.65 μV in three-target trials. ICA-P3a amplitude did not significantly differ between lags 3 and 9, $F(1,39) = 1.9$, $p = .13$. ICA-P3a amplitude was however larger in two-target trials than in three-target trials, $F(1,39) = 6.4$, $\eta_p^2 = .149$, $p = .019$. The difference between two-target and three-target waveforms was smaller at lag 9 than at lag 3, which produced a significant interaction between RSVP structure and lag, $F(1,39) = 3.7$, $\eta_p^2 = .128$, $p = .047$. Separate analyses confirmed that ICA-P3a amplitude did not differ between two-target and three-target trials at lag 9, $F(1,39) = 1.4$, $p = .31$, whereas this difference was significant at lag 3, $F(1,39) = 6.7$, $\eta_p^2 = .185$, $p = .019$.

The mean latency of ICA-P3a was 175 ms. There were no significant differences across conditions, $F < 1$, $p > .8$, for both main effects and for the interaction. To further ascertain the absence of any latency effects on P3a, the offset latency of the ICA-P3a was computed as the mean time-point when the descending portion of individual ICA-P3a waveforms crossed the 75% amplitude value. In both two-target and three-target trials, the mean ICA-P3a offset latencies were 331 ms at lag 9 and 383 ms at lag 3, reflected in a main effect of lag, $F(1,39) = 22.9$, $\eta_p^2 = .375$, $p < .001$. However, as shown in Figure 2.3, the mean ICA-P3a offset latencies observed in two-target and three-target trials did not differ significantly, $F < 1$, $p > .6$, nor was an interaction between number of targets and lag observed, $F < 1$, $p > .6$.

Regression of ICA Components

A direct link between T3-locked ICA-P3a amplitude and offset and ICA-P3b latency was explored through multiple linear mixed-effect regression analyses carried out on 160 ICA-P3b values –40 participants, each contributing one value in four cells of the number of targets by lag design– analyzed in the foregoing sections. The choice of both amplitude and offset to quantify ICA-P3a variations was based on the result of a preliminary analysis that revealed a positive correlation ($r$ = .28; $p$ = .038) between these two parameters across participants, as though greater P3a amplitude values were interindividually associated with slightly postponed P3a offset values.

A first regression explored the presence of a possible latent covariation between ICA-P3a and ICA-P3b parameters that were independent on the experimental variables manipulated in this design. The regression considered T3-locked ICA-P3b latency as dependent variable (y), T3-locked ICA-P3a amplitude (x1), and ICA-P3a offset (x2) as independent variables. The resulting linear model was:

Model 1: y = 313 – 18.8(x1) + 0.4(x2)

With a $R^2$ = .55, $t$(x1) = −4.6, and $t$(x2) = 5.8.

Model 1 was compared with the result of a second regression that was carried out on the same data set in which the four levels of the number of targets by lag design were explicitly included as independent factors, setting the least attention-demanding condition (i.e., two-target trials at lag 9; 2T-lag 9) as the baseline for contrasts against each of the other three conditions, that is, two-target trials at lag 3 (2T-lag 3) and three-target trials at lags 3 and 9 (3T-lag 3 and 3T-lag 9, respectively). The resulting linear model was:

Model 2: y = 335 – 4.3(x1) + 0.2(x2) + 37.9(2T-lag3) + 10.8(3T-lag9) + 79.0(3T-lag3)

With a $R^2$ = .71, *t*(x1) = −1.5, *t*(x2) = 2.7, *t*(2T-lag 3) = 3.2, *t*(3T-lag 9) = 1.0, and *t*(3T-lag 3) = 6.3.

Models 1 and 2 were submitted to a likelihood ratio test that indicated a Bayesian information criterion for the model 2 that was significantly smaller than the corresponding Bayesian information criterion for model 1 (1781 vs. 1803; $\chi^2(3) = 38.1$, $p < .001$). The ratio between Bayes factors (Bf) corresponding to model 2 and model (1) was greater than 100, indicating that model 2 explained the relationship between ICA-P3a modulations and ICA-P3b latency shifts of several order of magnitude more precisely than model 1 (Kass & Raftery, 1995).

Separate linear regressions were carried out on the data from each cell of the number of targets by lag design to better qualify the effect of the experimental manipulations on the distribution of P3b latency values, which are graphed in Figure 2.4.

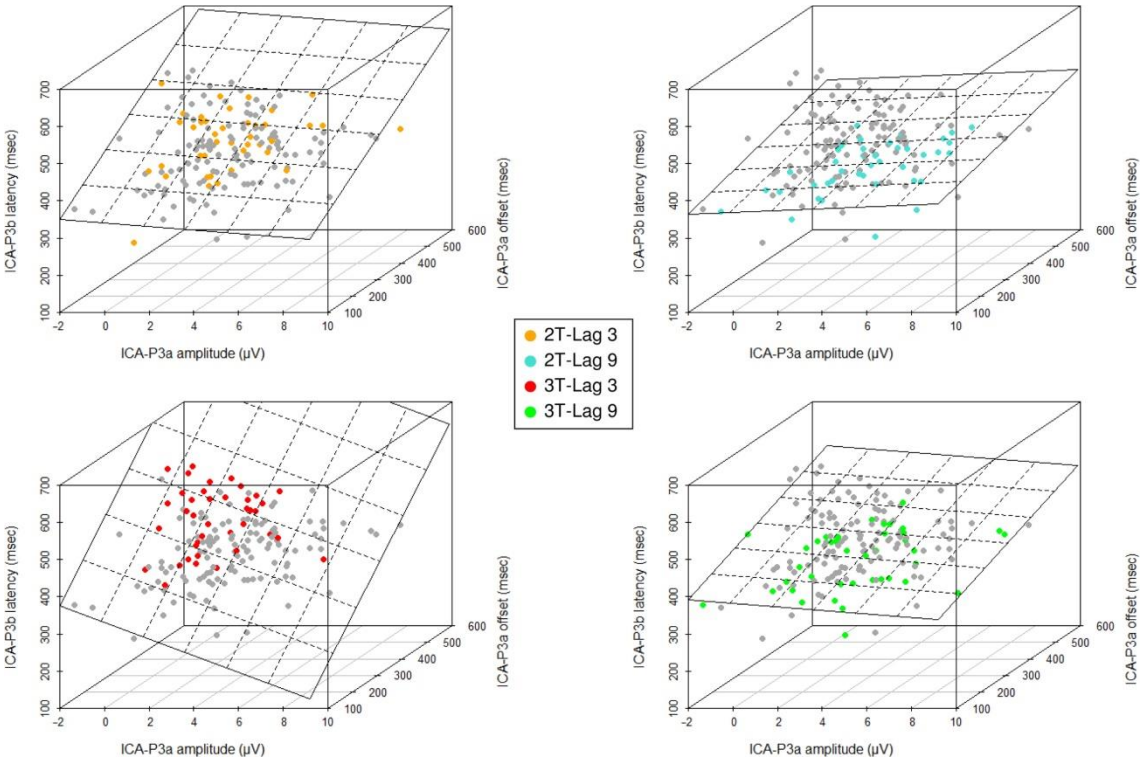


**Figure 2.4.** Results of the regressions on ICA-P3b latency values. ICA-P3b latency values in each scatterplot are reported as a function of the two predictors included in the regression models, ICA-P3a amplitude and ICA-P3a offset. Each scatterplot includes the entire data set of 160 ICA-P3b latency values–40 participants, each providing four ICA-P3b values in the number of targets (two-target vs. three-target trials) by lag (3 vs. 9) design–with the different conditions indicated by the different colors reported in the legend. All remnant gray dots in any given scatterplot correspond to ICA-P3b latency values in all the other three conditions. The plane intersecting the 3-D matrices in each scatterplot is a graphical representation of the linear model tested in each regression. Effects due to experimental manipulations are evident in the form of progressively increasing adherence of ICA-P3b latency values to the respective model (intersecting plane) from the least (2T-lag 9) to the most attention-demanding conditions (3T-lag 3).

At lag 9, in both two-target (cyan) and three-target (green) trials, the respective linear models –visually represented in each panel by the plane intersecting the 3-D distribution of ICA-P3b latency values– were not significant (both Fs < 1, ps > .5). At lag 3, in contrast, the regression analysis on ICA-P3b latency values in two-target (yellow) trials considering T3-locked ICA-P3a amplitude and ICA-P3a offset as predictors indicated a significant impact of ICA-P3a amplitude (45.01, $t(38) = 1.64$, $p < .01$) and P3a offset (0.74, $t(38) = 3.19$, $p < .003$) and a marginally significant trend toward an interaction between ICA-P3a offset and amplitude (−0.12, $t(38) = -1.83$, $p < .07$), with a $R^2 = .23$, $F(3,36) = 6.6$, $p < .03$. The regression analysis on ICA-P3b latency

values in three-target (red) trials considering the same predictors indicated a significant impact of ICA-P3a amplitude (114.0, $t(38) = 1.93$, $p < .05$) and P3a offset (1.33, $t(38) = 3.75$, $p < .001$) and a significant interaction between ICA-P3a offset and amplitude ($-0.34$, $t(38) = -2.16$, $p < .04$), with a $R^2 = .35$, $F(3,36) = 6.6$, $p < .03$. Note that $R^2$ values associated with each regression provide an estimate of the adherence of the cluster of ICA-P3b latency values in each experimental condition to the plane representing the tested model. As Figure 2.4 makes particularly clear, the adherence of ICA-P3b latency values increased from lag 9 to lag 3, the more so in three-target trials relative to two-target trials, as suggested by the increased $R^2$ parameters and ts associated with the interaction between ICA-P3a offset and amplitude.

## 2.1.5 Discussion

To characterize how distinct information processing stages interact during the selection and encoding of visual information distributed across time, an RSVP paradigm with a manipulation of the number of consecutive initial targets to modulate the magnitude of the AB (Dux et al., 2014). The last target in the RSVP streams was unmasked to allow the temporal dynamics of AB interference in the EEG signal to be observed in the absence of confounds from differing levels of accuracy on the final target (e.g., Ptito et al., 2008; Robitaille, Jolicœur, Dell'Acqua, & Sessa, 2007; Vogel & Luck, 2002). Behaviorally, presenting two targets before the final target (three-target trials) reduced accuracy of the final target, although it was unmasked. This finding is generally more compatible with proposals that the AB is a target-induced phenomenon (Dell'Acqua, Jolicœur, Pascali, & Pluchino, 2007; Dell'Acqua, Jolicœur, Luria, & Pluchino, 2009; Dux et al., 2014; Nieuwenstein et al., 2009; Visser, 2007; Wyble et al., 2009; 2011) rather than a form of attentional perturbation induced by distractors (Di

Lollo, Kawahara, Ghorashi, & Enns, 2005; Olivers & Meeter, 2008; Raymond et al., 1992; Taatgen, Juvina, Schipper, Borst, & Martens, 2009).

The electrophysiological measures showed clear anterior P3a and posterior P3b responses to the last target in the RSVP streams, and these were modulated differentially both by lag (3 vs. 9) and the number of preceding targets (1 vs. 2). The absolute magnitude of both ERP components was attenuated at short relative to long lags. However, there were key differences in these reductions with regards to latency and amplitude. Specifically, the P3a was reduced in amplitude, but its latency was unaffected by experimental manipulations. Conversely, the P3b exhibited both a decrease in amplitude and an increase in latency at short relative to long lags. Of import, this P3b latency increase for three- relative to two-target trials at the shorter lag could hardly be because of increased variance in the termination of pretarget(s) processing reflected in P3b jitter at short relative to long lags. In fact, Figure 2.2 reveals consistently higher amplitude values in the two-target relative to the three-target trials at lag 3 in the descending portion of P3b (compare orange and red functions in both graphs) suggesting –paradoxically– that more jitter was affecting two-target trials relative to three-target trials. This pattern was still present after isolating, via ICA analyses, the two components and fractionating the possible spatiotemporal overlap of P3a and P3b responses to the last target. Critically, the ICA reconstruction of P3b (Figure 2.3), at the shorter lag, shows a clear tendency of the P3b response in three-target trials to onset almost 100 ms after the P3b response in two-target trials. This strongly suggests that P3b component jitter is unlikely to be the cause of latency postponement affecting P3b responses elicited by the last target in two-target versus three-target trials. Rather, this finding complements and extends prior proposals referring to P3b as a signature of postponed consolidation of last target in working

memory for delayed report by tying P3b amplitude and latency modulations to effects induced by the number of targets preceding the last target in RSVP sequences.

Crucially, P3a responses consistently preceded P3b responses, and this has been hypothesized to reflect the direction and/or temporal order of activation of the neural structures composing a fronto-temporo-parietal circuit enabling conscious vision of attended objects (Daffner et al., 2003; Debener, Makeig, Delorme, & Engel, 2005; Friedman et al., 2001; Gazzaniga et al., 2000; Polich, 2003; Soltani & Knight, 2000). The results of the regression analyses revealed a parametric link between P3a amplitude and P3b latency. At the longer lag, processing reflected in P3a responses was largely independent of processing occurring later and reflected by P3b responses, as shown by the absence of correlation between these estimates. At the shorter lag, in contrast, the amplitude of P3a responses was correlated with P3b response latency, suggesting that the observed delay in processing of the target within posterior brain areas results from reduced efficacy (i.e., amplitude) of the frontally mediated detection process. In this vein, P3a and P3b responses would be separate but interacting manifestations of two functional stages of processing involved in targets' conscious access: reduced efficacy of attentional recruitment in frontal areas (P3a) and a consequent delay in the processing of the target by posterior areas (P3b). These results corroborate current thinking about the crucial role of the frontal lobe in the control of selective attention and the establishment of conscious representations during perception (e.g., Cohen, Cavanagh, Chun, & Nakayama, 2012; Corbetta, 1998; Desimone & Duncan, 1995; Dell'Acqua et al., 2006). In addition, the present findings complement fMRI results by providing an electrophysiological signature recorded at scalp of the involvement of frontal structures in the AB effect (Kranczioch et al., 2005; Marois et al., 2004).

Compared with the impressive corpus of studies focusing on the centroparietal P3b subcomponent of the P3 complex, the frontocentral P3a subcomponent has been the object of less investigation, and its functional connotation is still a matter of debate. The last decade of studies on P3a has unveiled a surprisingly tight connection between P3a and mental operations involved in attentional control. Indeed, contrary to the original depiction of P3a as a typical response to infrequent, novel, and contextually deviant stimuli (e.g., Friedman et al., 2001), more recent studies have provided evidence of P3a responses to task-relevant information displayed in a variety of cognitive tasks, like feedback signals displayed at the end of trials (e.g., Butterfield & Mangels, 2003), no-go signals in standard go/no-go designs (e.g., Rushworth, Walton, Kennerley, & Bannerman, 2004), and task-relevant stimuli displayed on first trials (e.g., Huettel, Mack, & McCarthy, 2002). In an elegant attempt at providing a unitary functional account of P3a activity encompassing these diverse experimental contexts, Barceló and colleagues (Barceló et al., 2006; Barceló, Periáñez, & Knight, 2002) devised a variant of a task-switch design in which one of four cards of the Wisconsin Card Sorting Test had to be matched with a target card of the same set according to two alternating criteria, either on the basis of the color of the symbols on the cards or on the basis of the symbols' number. On any given trials, the criterion for the classification task was specified by a tonal cue before the onset of the target card, which indicated if the classification scheme had to be maintained for the incoming stimulus (repeat-cue) or changed (switch-cue). Randomly on a proportion of trials, a contextually deviant sound –always novel on each trial– was displayed during the interval between a repeat-cue and the target card. Interestingly, P3a responses of equal amplitude were detected in response to both deviant sounds and switch-cues, a finding strongly suggesting that a primary determinant of P3a activity in task-switch designs is

attentional control demanded to (re)configure the mental set to carry out the classification task appropriately. Further work led these authors to put forth the hypothesis that the bursts of delta power, held to give rise to P3a responses, reflect inhibition of the current mental set to establish a different mental set (Prada, Barceló, Herrmann, & Escera, 2014).

This hypothesis is corroborated by studies on the AB that have shown a correlation between P3a amplitude and the probability of reporting T2 correctly in designs in which a task-switch was required to process T1 and T2. Sergent, Baillet, and Dehaene (2005; see also Marti, Sigman, & Dehaene, 2012) asked participants to distinguish between "XOOX/OXXO" strings displayed as T1 and to identify a T2 number word whose onset was signaled by four surrounding dots. P3a activity was detected for correctly reported T2 stimuli but not for missed items, as though the cause of the failure to report T2 could be ascribed to a failure to switch mental set or selection criteria between the different tasks. Task-switching in RSVP designs is known to exacerbate AB effects (Kawahara, Zuvic, Enns, & Di Lollo, 2003), and this may be so because task-switching is controlled by frontal areas partly overlapping with those underpinning the deployment of top–down attention to target information (e.g., Cutini et al., 2008; Dove, Pollmann, Schubert, Wiggins, & Yves Von Cramon, 2000). However, task-switching between T1 and T2 processing in the AB has been hypothesized to draw on distinct capacity limitations relative to those held to constitute the root cause of the AB (Dale, Dux, & Arnell, 2013; Kelly & Dux, 2011; Visser, Bischof, & Di Lollo, 1999).

One may still argue that P3a responses detected in the present experiment reflect some form of attention control processes directed at visual input, as proposed by Barceló and colleagues. These researchers suggest that task-switching is inextricably

linked with multitarget RSVP designs where selection criterion and task requirements on targets are uniform (i.e., report letters embedded among digits). Targets in this case must undergo a set of mental operations optimized for successful classification whereas a different set of mental operations may be hypothesized to be required to inhibit distractors (e.g., Di Lollo et al., 2005; Olivers & Meeter, 2008; Taatgen et al., 2009). One critical finding that is at odds with this proposal, however, is that that P3a responses described by Barceló's and colleagues, though correlated with behavioral switch costs (Monsell, 2003), were not correlated with trailing target-locked P3b activity, contrary to what was observed here at the shorter intertarget lag.

Perhaps, a better functional connotation of P3a and P3b responses in the present context can be attempted by resorting to psychological theory, whereby the present electrophysiological results appear to corroborate AB models ascribing the phenomenon to inhibition of top–down attention to visual input. Wyble et al. (2011) have proposed that the AB reflects mechanisms involved in parsing the visual continuum into discrete visual episodes and memory representations and provided P3b evidence compatible with this idea (Craston, Wyble, Chennu, & Bowman, 2009). More specifically, the model put forward by Wyble et al. (2009) hinges on the principle that top–down attention allocation to preattentive target sensory representations –or target types– is instrumental to bring activation of these representations suprathreshold. This triggers in turn an encoding mechanism that binds target types to time-coding memory units denoting targets' episodic arrangement-producing target tokens. These tokens are maintained in visual working memory and are available for subsequent conscious report. While target encoding is under way (e.g., for T1 in standard two-target RSVP streams), top–down attention is momentarily inhibited so as to segregate T1 from trailing visual inputs (i.e., T2 or distractors) lagging T1 for longer than 200 ms. This

processing dynamic is held to be at the root of the AB effect (and of the so-called lag 1 or protracted sparing effect; Dell'Acqua, Dux, Wyble, & Jolicœur, 2012).

This model fits naturally in the present electrophysiological picture by assuming that P3a amplitude is a measure of top–down attention allocation efficiency and that P3b amplitude and latency are a combined estimate of working memory encoding processes. In this augmented framework, encoding two previous targets in three-target trials versus one previous target in two-target trials would take up more processing capacity and produce stronger inhibition of top–down attention allocation to the last target. This is captured in the present results by the amplitude attenuation of P3a response to the last target observed at short lag, which was more pronounced in three-target trials than in two-target trials. Furthermore, inhibited top–down attention to target types during the AB would delay encoding of the last target in the RSVP sequence at short relative to long lags. This increased delay would be reflected in the prolonged latency and sizable amplitude reduction of P3b observed at the short lag, which was more evident in three-target trials relative to two-target trials.

When describing their results with reference to P3b suppression during the AB, Vogel et al. (1998) reported a frontal positive ERP component that preceded temporally the posterior P3b, which they labeled P2. The P2 component showed amplitude suppression under the same AB conditions as those associated with P3b suppression, raising thus the possibility that the P2 and the present P3a may be manifestations of the same underlying mechanism. Despite similarities however, the P2 is primarily reactive when attentional selection occurs based on simple features (Luck & Hillyard, 1994a), whereas P3a is a multimodal component elicited by a wide variety of stimuli. Indeed, the P2 found by Vogel et al. was observed under conditions where T2 had to be selected based on color (i.e., T2 was a white stimulus embedded among black

distractors). Similarly, a T2-elicited P2 component has been reported by Pesciarelli et al. (2007), who used to-be-detected red targets interspersed among white distractors. These researchers explored the T2-locked P2 as a function of whether T2 was missed or correctly reported at short lags and found no P2 amplitude/latency difference between these two conditions. Pesciarelli concluded, contrary to Vogel et al. (1998), that the visual P2 was not influenced by the AB. P2 and P3a differ in this respect, because P3a is demonstrably evident following a correctly reported target and absent following a missed target (Sergent et al., 2005). Most importantly, P2 amplitude tends to increase with stimulus repetition, whereas P3a amplitude behaves in the opposite manner, increasing when stimuli are novel and/or infrequent (Curran & Dien, 2003; Misra & Holcomb, 2003; Rugg, 1987). Collectively, these empirical observations reinforce the conclusions that the frontal subcomponent of the P3 complex evident in this study was really a P3a ERP component and not a P2.

To summarize, using a multitarget RSVP design and manipulating intertarget lag, prior findings indicating reduced amplitude and latency postponement of last target-locked P3b activity at short relative to long lags was replicated. This AB effect on P3b was dependent on target load, as these modulations were more pronounced in three-target trials, in which the last target was preceded by two to-be-encoded targets, than in two-target trials, in which the last target was preceded by just one to-be encoded item. An important and novel result was the clear AB effect on P3a responses, which were analogous in terms of amplitude modulations to P3b, but different in terms of latency, as no postponement was observed on the onset of this component. I have proposed that Wyble's et al. (2011) computational account of the AB offers a unitary framework where all the present results can be interpreted collectively. The suppression of P3a amplitude can be taken as evidence of reduced attention

allocation efficiency for detecting the last target during the AB window which, in turn, leads to a prolongation of the time taken to stabilize the sensory trace for generation of the conscious visual episode enabling delayed report of the last target.

## 2.2 Experiment 1b

### 2.2.1 Introduction

As explained in the General Introduction, a very peculiar aspect of the AB is its absence when the two targets are presented consequently (lag 1 sparing phenomenon, Potter, Chun, Banks, & Muckenhoupt, 1998).

Given the richness of the EEG dataset of Experiment 1a, portions of it were left unexplored. In particular, P3a and P3b responses time-locked to the initial target(s), preceding the last one, in two-target and three-target RSVP trials were not compared. Experiment 1a was focused to highlight the attentional dynamics between different attentional episodes (the episode which encode the initial target(s), and the one which encode the last target). In contrast, the analysis here presented was focused on the neurophysiological variation within a single attentional episode. Specifically, two-targets and three-targets trials of Experiment 1a gives the possibility to investigate the ERP activity elicited by a single target (that is the first target in two-targets condition; henceforth, TD condition, suggesting that the first target is followed by a distractor) and two consequent targets (which are the two first targets in the three-targets condition; henceforth, TT condition, suggesting that the first target is followed by a second one). By comparing these two conditions, it is possible to highlight attentional dynamics within a single attentional episode. In other terms, this data can provide evidence about lag 1 sparing phenomenon, and highlight the neural characteristics of this particular pattern of behavioral result happens.

Based on the two main models which described AB and lag 1 sparing phenomena, two different sets of prediction can be hypothesized. On the one hand, a prediction can be derived from Olivers and Meeter's (2008) model: a T1-locked frontal positive response should be immediately followed by a frontal negative component

indexing attentional inhibition elicited by the first target-trailing distractor. The onset timing of this frontal negative component should therefore differ between TD and TT condition, because the first target-trailing distractor is displayed in the position right after the only target in TD condition, and after two consecutive targets in TT condition. Based on evidence showing a magnified AB effect following two consecutive targets in three-target trials relative to the magnitude of the AB elicited by a single target in two-target trials (Dux et al., 2014), the amplitude of the frontal negative activity following the frontal positive response in TT condition should therefore be magnified relative to equivalent activity detected in TD condition. This model does not provide sufficient details regarding P3b activity –other than the shared assumption that P3b reflects target(s) consolidation in visual working memory– to make an exact prediction about this component. However, a cornerstone of the model is that there are no functional impediments to consolidate targets in visual working memory prior to the onset of the first target-trailing distractor. On this premise, there are no P3b modulations resulting from the present test that could be diagnostic of the appropriateness of the model to account for the AB and its ERP correlates.

On the other hand, the model proposed by Wyble et al. (2011) explicitly predicts that, upon detection of T1, tokenization (i.e., consolidation) is immediately activated, whether T1 is trailed by a distractor or by T2, and this prediction has been tested in an ERP study by Craston et al. (2009) showing that two sequential targets in an RSVP elicit a single P3b response. Referred to the present context, a first prediction is therefore that, following a T1-locked frontal positive response, the onset latency of T1-locked P3b should not vary whether T1 is trailed by a distractor or by T2, respectively. A second prediction arises from how the tokenization stage is characterized in the model, that is, as a stage where spatiotemporal information about target occurrence is

bound to information about target identity. This leads to the hypothesis that processing required to generate a single token in working memory is increased when two such tokens must be generated for working memory storage. To note, tokenization in this perspective is strongly akin to the function ascribed to the stage of memory consolidation proposed by Jolicœur and Dell'Acqua (1998), who characterized this stage as operating serially on sequential targets (see Craston et al., 2009; Kihara, Kawahara, & Takeda, 2008, for analogous proposal). On these premises, if tokenization (or consolidation) takes longer in TT condition relative to TD condition, then P3b should offset later in TT trials, when T1 is trailed by T2 and both targets must be consolidated in working memory, rather than in TD trials, when T1 is the only to-be-consolidated target.

## 2.2.2 Method

The dataset here considered was from the Experiment 1a of this thesis. Only a brief recap of the conditions used in this analysis and differences regarding ERP analysis are reported in the next sections.

## 2.2.3 EEG/ERP Recordings and Preprocessing

As illustrated in the lower part of Figure 2.5 and 2.6, the critical analyses were carried out on separate T1-locked ERP waveforms generated in trials at the longer lag only. This was done in order to minimize the overlap between ERP waveforms elicited by T1 in two-target trials and by consecutive T1 and T2 in three-target trials, and ERP waveforms elicited by the last target in the RSVP streams. Henceforth, for ease of exposition, I will refer to two-target trials as TD (to indicate that T1 was

followed by a distractor) and to three-target trials as TT trials (to indicate that T1 was followed by another target, T2). T1-locked ERP waveforms in TD and TT conditions were estimated by averaging EEG epochs recorded on both target-present and target-absent trials (i.e., with and without a final target ending the RSVP streams) associated with the correct report of T1 in TD trials, and T1 and T2 in TT trials. ERPs recorded in no-target trials were subtracted from these ERP waveforms to eliminate EEG oscillations in phase with the rate of presentation of RSVP items.

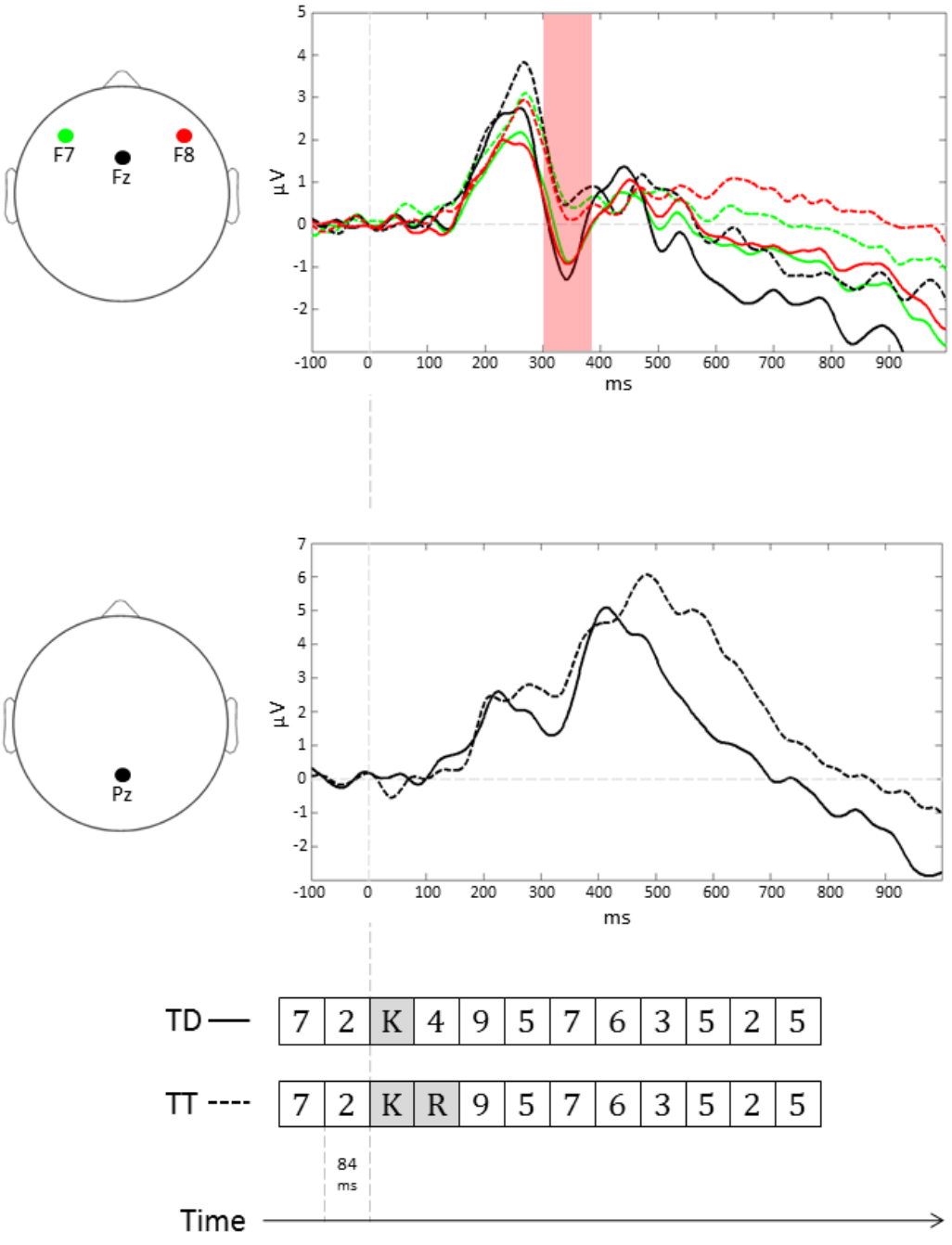


**Figure 2.5.** Lower: Gantt diagram illustrating the conditions of interest compared in the present article. In TT trials, T1 and T2 (highlighted as shaded letters for illustrative purposes) were always consecutive items, whereas T1 was trailed by a distractor in TD trials. Reported here are target-absent trials. Half of the trials were composed of target-present trials, namely, trials in which RSVP streams ended with a further target displayed after 8 distractors (SOA = 756 ms). Upper: T1-locked P3a and P3b components in TT (dashed lines) and TD trials (solid lines) flanked by a color-coded topographical indication of the originating electrode sites. The red shaded area provides information on the time window used for the post-P3a frontal negativity amplitude values calculation.

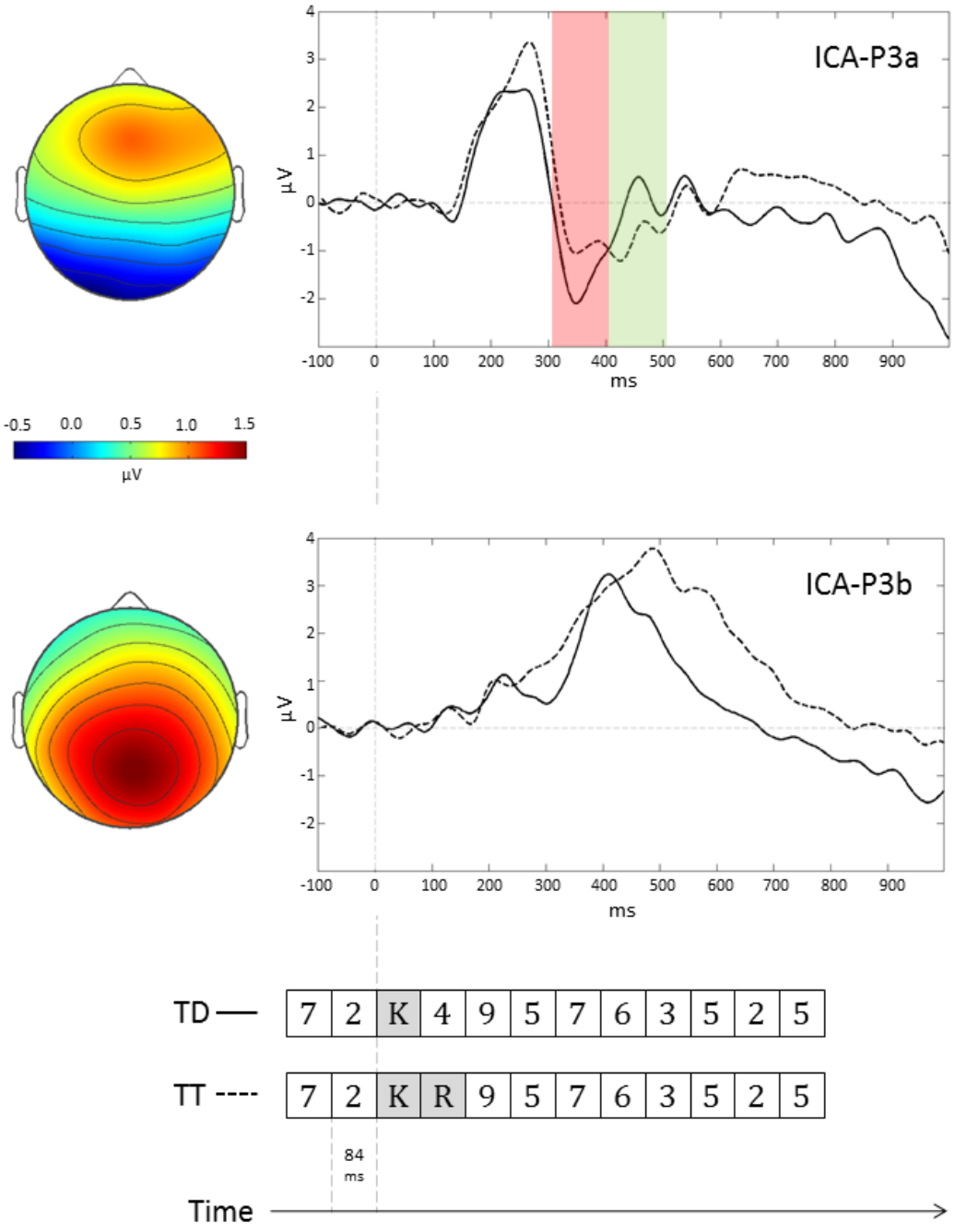


**Figure 2.6.** Lower: Gantt diagram illustrating the conditions of interest compared in the present article. Upper: Results of ICA decomposition of both T1-locked P3a and P3b components in TT (dashed lines) and TD trials (solid lines) flanked by the corresponding scalp plots of peak activity. The red and green shaded areas provide information on the time windows used for amplitude estimation of ICA-decomposed post-P3a frontal negativity in TD trials (red) and TT trials (green).

The mean amplitude of the subtracted T1-locked P3a and P3b components was quantified as the mean value in a 150 ms window centered on the peak of each

grand-averaged ERP. Given the explicit reference of Olivers and Meeter (2008) to the frontal modulations of ERP activity reported by Martens et al. (2006), frontal activity in the P3a time range was analyzed at F7, Fz, and F8 electrodes. The P3b component, as in experiment 1a was analyzed at Pz (Polich, 2003). The mean latency of the subtracted P3a and P3b components at the same recording sites was estimated using the jackknife approach (Kiesel et al., 2008; Ulrich & Miller, 2001), and individual values were derived with the solution proposed by Brisson and Jolicœur (2007; see also Smulders, 2010). Onset latency values were calculated as the time point when the ascending portion of individual jackknife time course reached 75% of the peak amplitude. Offset latency values were calculated as the mean time point when the descending portion of individual jackknife ICA time course crossed the 75% amplitude value. The Greenhouse-Geisser correction for nonsphericity was applied when appropriate.

## 2.2.4 Results

### Behavior

Separate analyses of variance (ANOVAs) were carried out to compare the mean proportion of correct target report in TD and TT trials. Subjects were more accurate in reporting T1 in TD trials (95.4%) than in TT trials (79.2%), $F(1,39) = 104.5$, $\eta_p^2 = .732$, $p < .001$. In TT trials, subjects were more accurate in reporting T2 (93.3%) than T1, $F(1,39) = 100.4$, $\eta_p^2 = .724$, $p < .001$. T1 report in TD trials was also superior to T2 report in TT trials, $F(1,39) = 5.7$, $\eta_p^2 = .130$, $p = .038$. Block order (i.e., whether subjects started the experiment with three-target or two-target trial blocks) did not exert any effect on behavioral performance, max $F < 1$. Furthermore, in 46.1% of TT trials, T1 and T2 were correctly reported albeit in reversed order. In short, as repeatedly

observed in prior investigations, under TT conditions T2 was reported more accurately than T1 and, on a substantial proportion of trials, was reported as the first target.

### ERPs

The artifact screening procedures described above resulted in the exclusion of 0.74% of the segments. For most subjects, less than 1% of the data were excluded. Two subjects had exclusion rates of about 7%. Visual inspection of their ERPs suggested their results were comparable to those of the other subjects, and thus their data were included in the final analyses. The final sample included all 40 participants tested in the experiment. In all the following ERP analyses, block order (i.e., whether subjects started the experiment with three-target or two-target trial blocks) was included in the various ANOVA designs. However, given that block order was never associated with significant main effects or interactions with the other considered factors, max $F < 1$, min $p > .43$, the influence of this factor is not discussed in the forthcoming sections. The most important T1-locked ERP waveforms observed in the present experiment are reported in Figure 2.5.

### P3a

The mean P3a amplitude values observed in TT versus TD trials were 2.95 μV and 2.09 μV at Fz, 2.44 μV and 1.66 μV at F7, and 2.1 μV and 1.68 μV at F8. An ANOVA carried out on individual P3a amplitude values indicated that P3a was of greater amplitude in TT than TD trials, $F(1,38) = 5.8$, $\eta_p^2 = .132$, $p = .02$. Furthermore, P3a amplitude differed across electrode sites, $F(2,76) = 5.2$, $\eta_p^2 = .120$, $p = .007$. False discovery rate (FDR; Benjamini & Hochberg, 1995) corrected t tests indicated that P3a amplitude was greater at Fz relative to both F7, $t(79) = 3.0$, $p = .008$, and F8,

$t(79) = 3.44$, $p < .001$, which did not differ significantly, $t < 1$, $p > .34$. The mean P3a onset latency in TD trials (219 ms) and in TT trials (225 ms) did not differ significantly, $F(1,38) = 1.2$, $p > .4$. The mean P3a offset latency in TD trials (283 ms) and in TT trials (294 ms) also did not differ significantly, $F(1,38) = 2.1$, $p = .22$. No difference in P3a onset/offset latencies was found across F7, Fz, and F8 electrodes, max $F < 1$, min $p > .35$.

Post-P3a frontal negativity

In order to test Olivers and Meeter's (2008) prediction concerning distractor-induced attention inhibition being indexed by a peak of negativity following the initial frontal activation reflected by the T1-locked P3a, ERP activity trailing P3a was explored at each frontal electrode considered in the P3a analyses (i.e., F7, Fz, and F8) in a time window starting 303 ms post-T1 (i.e., a value corresponding to the mean time point at which the descending portion of P3a crossed the baseline in TT and TD trials) and ending at 390 ms post-T1 (highlighted in red in Figure 2.5). The mean amplitude of this component was −.39 μV on TD trials, and .74 on TT trials. An ANOVA revealed that these values differed significantly, $F(1,38) = 14.8$, $\eta_p^2 = .281$, $p < .001$, and were comparable across electrode sites, $F = 1.3$, $p = .21$. As shown in Figure 2.5, separate one-tailed *t* tests indicated that negative ERP activity (i.e., significantly less than 0) was detected in TD trials, $t(119) = -2.1$, $p = .048$, but not in TT trials, where the component was positive.

P3b

The mean amplitude of P3b was 4.03 μV in TD trials, and 5.31 μV in TT trials. The ANOVA showed that these values were significantly different,

$F(1,38) = 14.6$, $\eta_p^2 = .269$, $p < .001$. The P3b onset latency was not different between TD trials (386 ms) and TT trials (393 ms), $F < 1$, $p = .75$. The P3b offset latency was, however, substantially postponed in TT trials (496 ms) relative to TD trials (596 ms), $F(1,38) = 70.3$, $\eta_p^2 = .641$, $p < .001$.

ICA of ERPs

The same EEGLAB routine of Experiment 1 was used to decompose T1-locked ERPs through ICA (Delorme & Makeig, 2004). As in the previous experiment, this was done to provide a more faithful depiction of the ERP results by decomposing the various components explored through standard analyses into maximally spatiotemporally independent signals available in the channel data, and minimize the influence of their potential overlap/summation on the interpretation of the above findings. One hypothesis in particular that had to be ruled out is that the post-P3a frontal negativity (absent) in TT trials may have been camouflaged by spatiotemporal superimposition with a surge of positive activity trailing P3a, which could unpredictably have been more intense and/or anticipated in TT versus TD trials.

Individual ERPs in TT and TD trials were first analyzed using singular value decomposition to determine the dimensionality of the signal subspace containing most of the relevant event-related activity. A scree plot of the singular values showed a clear break after the first three components, leading to retain the first four dimensions, which accounted for 51.8% of the variance. The ICA analysis was thus restricted to this subspace of the signal space using an initial principal component analysis (PCA). The ICA decomposition isolated two components of the P3 family, namely, an earlier anterior component (ICA-P3a) and a later posterior component (ICA-P3b). The grand-

averaged time courses and relative topographies for these two components in TD and TT trials are illustrated in Figure 2.6.

ICA: P3a

The mean amplitude of ICA-P3a was significantly greater in TT trials (2.45 μV) than in TD trials (1.89 μV), $F(1,38) = 5.46$, $\eta_p^2 = .123$, $p = .027$. The mean onset latency of ICA-P3a in TD trials (201 ms) and TT trials (223 ms) did not differ significantly, $F(1,38) = 1.9$, $p = .36$. The mean offset latency of ICA-P3a was 272 ms in TD trials and 289 ms in TT trials. These values were statistically different, $F(1,38) = 4.31$, $\eta_p^2 = .101$, $p = .036$.

ICA: Post-P3a frontal negativity

As Figure 2.6 suggests, my speculations that post-P3a negative activity may have been influenced by spatiotemporal overlap with contrasting positive activity that varied between TT and TD trials was correct. Contrary to the results observed for the standard ERP analyses, the ICA decomposition revealed that negative ERP activity trailed P3a in both TD and TT trials.

An ANOVA carried out on latency values indicated that the onset latency of ICA-decomposed post-P3a frontal negativity did not differ between TD trials (322 ms) and TT trials (327 ms), $F(1,38) = 1.6$, $p = .22$. However, inspection of Figure 2.6 suggests that TT and TD trials elicited T1-locked ERP time courses seemingly compatible with the prediction that post-P3a frontal activity in TD trials should be anticipated relative to equivalent activity in TT trials (i.e., in which the distractor is postponed by 84 ms). In a 310–510 ms time window (i.e., from the mean time point at which the descending portion of the P3a component in TD trials crossed the baseline to

the final convergence of TT and TD waveforms), the post-P3a negative deflection in TD trials is in fact more skewed toward an earlier peak in TD trials, and more symmetrical around a later peak in TT trials. The crucial test on the relative amplitude of the post-P3a negative component in TD and TT trials –guided by assuming the postponement of post-P3a activity in TT trials versus TD trials, predicted on the basis of Olivers and Meeter's (2008) model– was performed by splitting the 310–510 ms time window in half, and by comparing the amplitude of post-P3a negative activity recorded in a 310–410 ms time window for TD trials with the amplitude of post-P3a negative component recorded in a 410–510 ms time window for TT trials. Two preliminary one-tailed *t* tests confirmed that the recorded activity was indeed negative (i.e., significantly less than 0) in both TD trials, $t(39) = -4.9$, $p < .001$, and TT trials, $t(39) = -2.3$, $p = .018$. Contrary to the predicted magnification of the amplitude of the post-P3a negative component, a subsequent analysis revealed that the amplitude of post-P3a negative activity was greater in TD trials (−1.45 μV; 310–410 ms) than in TT trials (−.74 μV; 410–510 ms), $F(1,38) = 5.31$, $\eta_p^2 = .144$, $p = .027$. A final analysis was conducted to compare the overall amplitude of post-P3a negative activity in the entire 310–510 ms time window. The result of this analysis revealed that post-P3a negativity amplitude in TT trials (−.731 μV) was basically identical to post-P3a negativity amplitude in TD trials (−.733 μV), $F(1,38) = .05$, $p = .99$.

ICA: P3b

The mean amplitude of the ICA-P3b was significantly greater in TT trials (3.29 μV) than in TD trials (2.48 μV), $F(1,38) = 13.34$, $\eta_p^2 = .255$, $p < .001$. The mean onset latency of ICA-P3b was not different between TD trials (382 ms) and TT trials (397 ms), $F(1,38) = 2.0$, $p = .22$. However, the mean offset latency of the ICA-P3b was

substantially longer in TT trials (492 ms) than in TD trials (600 ms), $F(1,38) = 68.4$, $\eta_p^2 = .642$, $p < .001$. This 108 ms difference between ICA-P3b offset latencies was significantly longer than the 17 ms difference between ICA-P3a offset latencies in TD versus TT trials, $F(1,76) = 52.8$, $\eta_p^2 = .410$, $p < .001$.

## 2.2.5 Discussion

This experiment was aimed at investigating attentional dynamics within a single attentional episode of a single target vs. two consecutive targets embedded within an RSVP. Standard analyses and an ICA reconstruction of the spatiotemporal T1-locked ERP patterns were consistent in revealing that the difference between TT and TD trials was reflected primarily in modulations of two subcomponents of the P3 complex. Specifically, both analytical approaches produced results indicating a frontocentral T1-locked P3a waveform of larger amplitude in TT trials than in TD trials. This P3a amplitude increase elicited by consecutive targets was accompanied by a 17 ms postponement of the corresponding P3a offset latency. Hints of negative activity trailing P3a were found only in TD trials using a standard ERP approach, and generally more marked in TD versus TT trials using the ICA approach. A centroparietal P3b was also observed to be of greater amplitude in TT trials relative to TD trials, with a postponement of P3b offset latency in TT trials that was, however, one order of magnitude more substantial than that for P3a, amounting to 108 ms. There is reasonable agreement on the role of the dorso- and ventrolateral prefrontal cortices in the generation of P3a (e.g., Ranganath & Rainer, 2003). Indeed, these current results are in broad agreement with evidence indicating the involvement of the frontoparietal network in enabling attentional selection of task-relevant information, both when displayed simultaneously with arrays of spatially distributed distracting information (Corbetta,

1998; Todd & Marois, 2004; Xu & Chun, 2006; Yantis et al., 2002) and when embedded in a spatially overlapping, but temporally distributed, sequence of distracting events (Dell'Acqua et al., 2006; Husain, Shapiro, Martin, & Kennard, 1997; Joseph, Chun, & Nakayama, 1997; Lagroix, Grubert, Spalek, Di Lollo, & Eimer, 2015; Marcantoni et al., 2003; Marois, Chun, & Gore, 2000). There is also good agreement that more posterior regions, including the temporoparietal junction and inferotemporal cortices, are likely involved in the generation of P3b (Polich, 2003, 2007).

Specific predictions about the possible ERP modulations in the present design were derived from two current neurocomputational models of temporal selective attention. Predictions from Olivers and Meeter (2008) and Wyble et al. (2011) concerning the time course of attention deployment to the first target(s) encountered in RSVP were confirmed by the amplitude increase of T1-locked P3a when T1 was trailed by another target relative to when T1 was displayed as a single target in RSVP. According to Olivers and Meeter (2008), attention deployment to RSVP targets is necessary to transfer these stimuli into visual working memory. As detailed in the introduction, this model predicts that attentional deployment to T1 in TD trials would be curtailed by the inhibitory response elicited by the distractor trailing T1, which would attenuate the T1-locked P3a response. This would not occur in TT trials given the presence of T2 trailing T1, which would provide more time for the P3a response to grow further, as was in fact observed. According to Wyble et al. (2011; see Figure 6, p. 493), attention is deployed to RSVP targets to enhance their sensory traces so as to enable them to activate corresponding "types," namely, nodes in conceptual short-term memory (Chun & Potter, 1995; Potter, 1976). Types in turn can be encoded as tokens, that is, reportable items, once they are bound to physical features promoting episodic distinctiveness. In this model, the summation of attentional responses to T1 and T2

would be the cause of the increased P3a amplitude in TT trials compared to TD trials. In line with Experiment 1a, the offset latency difference of P3a between TT and TD trials was minimal. This suggests that processing of two consecutive targets at stages prior to memory encoding overlap considerably, dovetailing with earlier reports using faster RSVP presentation rates than typically employed. For example, Potter et al. (2005) displayed two synchronous RSVP sequences of nonwords, one above and one below a central fixation point at 20 Hz, each embedding one target word, T1 and T2. T1 and T2 were names of semantically related real-world concepts on half of the trials, and unrelated concepts on the other half of trials. Critically, at SOA ranging from 0 to 120 ms, a semantically related T2 primed T1, thus supporting the idea that, when presented in close temporal proximity, type nodes were simultaneously active in conceptual short-term memory.

One may wonder why T2 in TT trials, whose onset coincided temporally with the bulk of attention accumulation indexed by P3a, was reported less correctly than T1 in TD trials. The AB models used to generate the predictions tested in the present study provide different explanations for this often-observed effect. Both accounts postulate that encoding two consecutive targets incurs some form of intertarget interference. The models differ, however, relative to the locus of this interference. Olivers and Meeter (2008) propose a visual working memory locus, wherein encoded targets compete for maintenance and recall (see also Raymond et al., 1992, for an analogous proposal). Wyble et al. (2011) posit mutual inhibition of concurrently active types, and this is reflected in slightly lower report accuracy for consecutive targets relative to when targets are displayed in RSVP separated by intervals outlasting the AB window (cf. Dell'Acqua, Dux, Wyble, & Jolicœur, 2012, for supporting evidence).

The P3a found in this data could corresponds to the transient enhancement of frontal selection positivity (FSP) found by Martens et al. (2006; see also Potts, 2004; Smid et al., 1999) and measured in a 180–350 ms window post-T1 onset. Martens et al. (2006) concluded that FSP reflected attention control over target(s) selection, which is germane to the present idea of the function indexed by P3a. Thus, a small difference aside in the topography of the FSP component, whose peak was found at F7/F8 by Martens et al. (2006), the overlap of temporal parameters and proposed functional connotations of FSP and P3a suggests that the recruitment of the frontal brain regions for attention-guided selection in RSVP is reflected in rapid increments of frontal positive activity upon detection of T1.

Limited evidence was found for attention inhibition induced by the lag 1 distractor as predicted by Olivers and Meeter (2008). Based on the boost and bounce architecture, a negative component with a time course corresponding to that of the AB should have been observed following P3a at frontal electrode sites. This negative component, reported by Martens et al. (2006) in a post-FSP/P3a T1-locked time window, was parametrically investigated by Niedeggen, Hesselmann, Sahraie, Milders, & Blakemore (2004), who proposed that this component could index the activation of an "attention-gating mechanism" temporarily halting processing of visual information trailing a leading, attention-demanding visual event. In this perspective, given the temporal shift in onset of the distractor trailing one single target or two consecutive targets in TD and TT trials, respectively, a negative component with a postponed latency was expected in TT trials relative to TD trials. The results produced using the standard and ICA approaches do not appear in line with this prediction. No latency variations compatible with the hypothesis that post-P3a negative activity was elicited by the first target(s)-trailing distractor were detected when the negative component

emerged following the ICA decomposition of the multivariate spatiotemporal distribution of the T1-locked ERP signal. Furthermore, when the overall amplitude of post-P3a negative component was explored in a 310–510 ms time window, the results indicated an equivalence between TD and TT trials, which is incongruent with behavioral findings reflecting a much more pronounced AB in TT versus TD trials when tested with the behavioral variant of the present design (i.e., by masking the last target and monitoring its correct report; Dux et al., 2014). So, although a precise functional characterization of the post-P3a negative activity is beyond the scope of the present investigation and certainly worth further experimental inspection, the present ERP results appear to generally run counter to the idea of a distractor-induced nature of the AB.

The ICA-P3b results were clear-cut: The onset of the P3b did not differ in TD versus TT trials, and its duration was longer in TT trials than in TD trials. These results suggest that encoding two targets took longer than encoding one target (Dell'Acqua et al., 2012; Dell'Acqua et al., 2009; Dux, Asplund, & Marois, 2008; Jolicœur & Dell'Acqua, 1998), and converge with proposals about the target-locked essence of the AB effect (see Dux & Marois, 2009; Martens & Wyble, 2010, for extensive surveys and comparisons of models of temporal attention hinging on this principle). Collectively, the P3b findings appear to be congruent with predictions based on Wyble et al. (2011).

Two aspects of the P3b response time course deserve particular consideration. The P3b responses recorded in both TT and TD trials showed comparable onsets but clearly different offsets, that is, the P3b offset latency was postponed in TT relative to TD trials. In other words, the P3b response was unimodal, much like the P3b response to consecutive targets reported by Craston et al. (2009). These authors

interpreted the unimodal P3b response elicited by two consecutive targets as evidence for the targets' integration into a single attention episode (see also Kessler et al., 2005, for analogous evidence produced using magnetoencephalography, MEG). The present results complement and extend those of Craston et al. (2009) by establishing a direct link between the amount of information that is ultimately encoded in visual working memory and the temporal extension of an attention episode.

The second aspect emerges from the comparison between P3b duration (difference between offset and onset latencies) across TD and TT trials. The 108 ms difference in P3b duration for TT trials relative to TD trials suggests it takes, on average, 108 ms more to encode two targets compared with one target. This may seem surprising given the apparently relatively long time required for the P3b to reach its peak amplitude. However, the peak of the P3b presumably reflects encoding as well as all processes taking place prior to encoding. The difference between TT and TD conditions presumably subtracts out some of these differences, leaving a closer estimate of the mean encoding duration. Interestingly, Jolicœur and Dell'Acqua (1998), using dual-task methods and computer simulation, arrived at an estimate of about 169 ms of additional time to encode two letters (suggesting an encoding cost of about 84 ms per item, which is not far from the present estimate of 108 ms; see their Table 2 and Experiment 7). Encoding into working memory, or short-term consolidation, appears to be a slow process with high variance but for which the cost of additional items hovers around 108 ms, a value that converges nicely with the estimate reported by Craston et al. (2009) of 100 ms estimated from the T1-locked P3b offset in lag 1 trials compared to that in lag 8 trials.

To summarize, rapid visual information processing for items that appear within the same temporal attention window was assessed by using EEG. Contrasting

two prominent computational models of the AB, P3 complex could be fractionated into distinct components, which were affected differently by whether an attentional window contained two targets or a target and a distractor. Specifically, whereas only frontal P3a amplitude was influenced by increased target load, both amplitude and latency of the parietal P3b were increased by target load. The results suggest that within temporal attention windows there are two stages of information processing subserved by distinct neural substrates. Selection appears to occur close-to-concurrently for multiple targets and draws on frontal regions of the brain. This then leads to encoding of this information in a serial manner that prominently taps the temporoparietal lobes.

## 2.3. Conclusion: Chapter 2

In the current Chapter temporal dynamics of attention in an AB task was highlighted. As previously reported by other studies (e.g., Gross et al., 2004), frontal and posterior brain modulations occur during the AB effect. Specifically, in Experiment 1a, the P3a component (which reflects the detection of relevant information) presented a decreased amplitude at short relative to long lags. P3b component was both decreased in amplitude and postponed in latency under analogous conditions. Collectively, the results suggest that AB delays target encoding in working memory, although the detection mechanism is not slow down but only decreased in efficacy.

Moreover, in Experiment 1b, the activity elicited by a single target or two consecutive targets was investigated. the P3a was increased when two targets were presented relative to a single target. P3b component was also increased in amplitude but its time course was longer when two targets compared to one were presented. These results provided evidence for the involvement of frontal brain region in the selection of information presented in close succession, and of posterior brain regions in the serial of targets in visual working memory.

The evidence of Experiment 1a will be now used as the benchmark to compare the same activations in a quite similar task which also includes visual spatial deployment of attention. Indeed, in the next Chapter, the last target of RSVPs will be lateralized. The predicted outcome is two folded: (i) a similar pattern of Experiment 1a regarding P3a and P3b modulations is expected, reflecting a common mechanism of visual attention in the RSVP; (ii) a modulation of N2pc, which reflect the posterior engagement of visual attention is expected, showing that visual spatial attention is disrupted by the same task dynamics which affect the typical AB effect.

# Chapter 3

Part of the content presented in this chapter has been described in the following published article:

Losier, T., Lefebvre, C., Doro, M., Dell'Acqua, R., & Jolicœur, P. (2017). Backward masking interrupts spatial attention, slows downstream processing, and limits conscious perception. *Consciousness and Cognition*. https://doi.org/10.1016/j.concog.2017.04.005

# 3.1 Experiment 2

## 3.1.1 Introduction

The findings of Experiment 1A showed how the processes underlying AB dynamics are modulated through lags and by the target(s) preceding the last one of an RSVP. Results show that the decreased amplitude and the offset postponement of the P3a within the AB time window, which reflect the detection of the target, explain the onset delay and the decrease amplitude of the P3b, which mirror the target encoding.

Some studies highlighted that there is a link between temporal and spatial deployment of attention (i.e., Dell'Acqua et al., 2006). In order to explore more in detail the functional link between temporal and spatial attention, Experiments 2 combined together Experiment 1a with a design inspired by the study of Dell'Acqua and colleagues (2006). Specifically, the activity elicited by a lateralized target presented in the end of an RSVP was recorded. The target could be separated by a short or long lag of one or two target(s). Moreover, a between subjects manipulation was the absence or the presence of a masking after the last target (see Method for details). As anticipated in the General Introduction, a lateralized target elicit a lateralized activity in the N2 time window, called N2pc (Luck & Hillyard, 1994a). The aim of this experiment was to highlight whether the N2pc, which is an earlier component compared to the P3a and P3b, is modulated by the AB attentional dynamics or by masking. Vogel et al. (1998) found no effects of AB on the visual P1 component, suggesting a locus of interference somewhere after early sensory encoding: based on this evidence, the N2pc could be a good candidate as the first marker of attentional disruption. In other terms, I want to test whether visual spatial attention is modulated by the same AB dynamics which modulate the P3 complex.

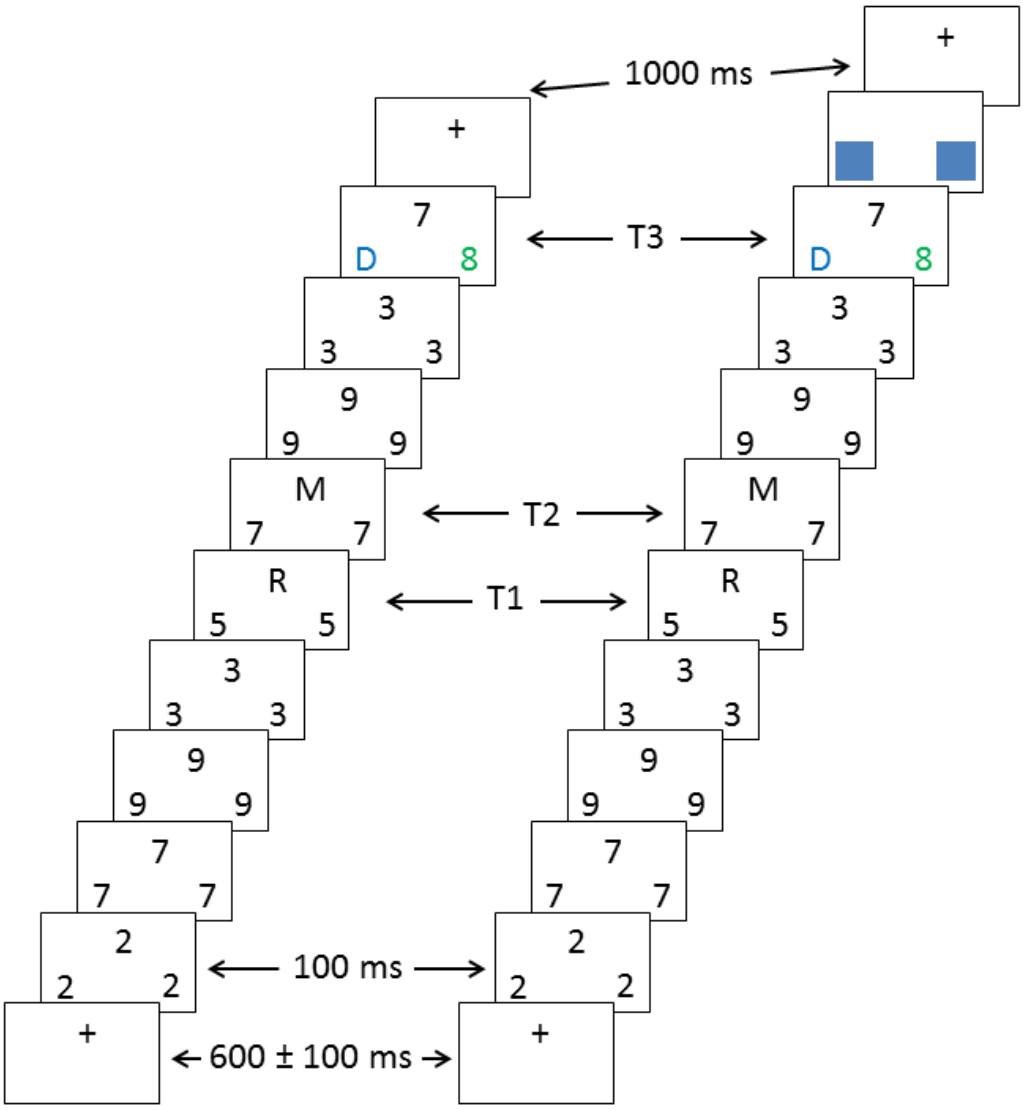


**Figure 3.1.** Example of an RSVP presentation of Experiment 2. In this example, three targets were presented. In the RSVP on the left part of the picture no mask was presented after the last target, differently from the panel on right. As for Experiment 1a, the RSVP could contain two consecutive targets and then a third one (at a short or long lag), or two targets separated by a short or long lag.

As in Experiment 1a, three or two targets were presented in each RSVP. At the end of each RSVP the central position, previously occupied by distractors and target(s) were occupied by a fixation cross. At the left and right of the fixation cross a green and a blue stimuli appeared: subjects' task was to report at the end of each trial the identity of the midline presented target(s) and of the target-colored letter (blue or green, counterbalanced) at the end of the RSVP. Moreover, the last target of the RSVP was followed by a mask for a group of subjects.

In summary, the impact of AB and masking was studied in spatial deployment of attention. Moreover, attentional ERP components related to detection and encoding of a target are investigated to replicate the findings of Experiment 1a.

Regarding the deployment of attention, a decreased amplitude of the N2pc is expected when the last target is presented at short lag compared to a long one. Moreover, an analogous effect related to the number of target(s) which precede the last one is expected. Namely, a short lag could decrease the amplitude of the N2pc. Finally, visual masking is expected to decrease or suppress the N2pc as well.

## 3.1.2 Method

Participants

The participants were eighty-five undergraduate students at Université de Montréal. Fifty-one were originally assigned to the no-mask group but eleven were excluded from analysis for various technical reasons (see the Electrophysiological Recording and Data Analysis section for details). Forty participants (30 females) between the ages of 19 and 33 (mean age = 21.71, SD = 2.3) were therefore kept for further analysis. Thirty-nine participants were assigned to the mask-present group. Thirty-four participants were kept for analyses (see the Electrophysiological Recording

and Data Analysis section for details) (25 females) and they were between the ages of 18 and 26 (mean age = 21.45, SD = 1.9). All reported normal or corrected to normal vision, no history of neurological disorders, and all showed normal performance on the Ishihara color test. They received 20 $Can for their voluntary participation in the study after providing written informed consent.

### Stimuli

An example of the stimuli and sequence of events on each trial is illustrated in Figure 3.1. Filler frames in the RSVP sequence consisted of 3 identical digit distractors (between 2 and 9) in a triple RSVP stream. Trials could contain one, two or three targets that consisted of uppercase letters from the English alphabet (excluding B, I, O, Z, and Q, to avoid confusion with digits). The characters were light gray in Courier New font on a black background. Stimuli were 1° of visual angle in height, at a distance of 57 cm from the screen of a CRT monitor (maintained using a chin rest). In each frame, one stimulus was at fixation whereas the other two were displaced to the left or right by 3° and down by 1° of visual angle. Each item of the RSVP was presented for 100 ms, with no inter-stimulus interval. Three-targets and two-targets condition are basically composed as in Experiment 1a: in the first case two consecutive target letters were presented in the central RSVP, in the second case only one letter was presented. The first target was randomly in the 6th, 7th, 8th, or 9th frame. In three-target trials, the second target was in the frame immediately following the first target. Finally, in order to elicit a deployment of attention that could be tracked by monitoring N2pc, the position of the last target was in the right or left visual field of the final frame; 3 or 8 frames following the previous target. For participants in the masking group, last target frame was followed by a five by five blue and green checkerboard of 1° of visual angle.

Digits and letters were semi-randomly assigned during the sequence with no two same digits repeated from one frame to the next. Targets for a particular trial were always different letters from each other. For the last frame, participants were instructed to pay attention to a target color (blue or green; counterbalanced). The last target, whether a distractor or target, was in the target color (e.g., blue) while the middle digit was light gray and the digit on the other side was in the distractor color (e.g., green). Thus, the last frame containing characters could be a target (when a letter was printed as the target color) or a distractor (when a letter was printed as the distractor color). The stimulus on the other side of fixation was always a distractor (digit). This manipulation allowed participants to know when and where to deploy attention when the last frame consisted only of distractors. The luminance of the three colors used (blue, green, and light gray) was adjusted to be approximately equiluminant using a Minolta CS100 chromameter (3.30 cd/m2).

Procedure

Participants were seated in a dimly-lit electrically-shielded room and initiated each trial by pressing the space bar. A 100 ms jittered delay of 600 ms preceded the onset of the RSVP sequence. The task was to maintain fixation at the center of the screen and to encode letters presented at that location, and to encode a blue (for half of the subjects, green for the other half) letter in the final frame, for participants in the no-mask group, and in the frame just before the mask for those in the mask group. After the end of the sequence, they were to maintain fixation on a fixation cross for another 1 s, after which they entered all the letter(s) they saw on a standard keyboard after the fixation cross disappeared and a question mark appeared. The delay between the end of the RSVP sequence and the response period ensured that muscle activity and

ocular artifacts from eye movements towards the keyboard did not overlap with ERPs of interest.

The experiment included 672 experimental trials divided into 21 blocks of 32 trials each, preceded by a practice block of 16 trials. There were four within-subject experimental variables (factors) each with two levels, lag between the penultimate and the last target (3 or 8), trial type (2 or 3 stimuli to attend in the RSVP), presence or absence of the last target, and side-of-presentation of the last target (left or right visual field), yielding 16 combinations. And, there was one between-subjects factor: presence or absence of a mask after the last target (Figure 3.1).

### 3.1.3 EEG/ERP Recordings and Preprocessing

EEG was recorded using a BioSemi Active Two system and an elastic cap with 64 Ag/Ag–Cl electrodes positioned according to the International 10/10 system. The sampling rate was 512 Hz and the signal was referenced to the average of left and right mastoids after the recording. A high-pass filter of 0.1 Hz and a low-pass filter of 30 Hz were applied offline. The horizontal electro-oculogram (HEOG) was obtained using the subtraction of activity from a pair of electrodes situated on the left and right eye outer canthi, which was used to monitor eye movement. Vertical eye movements and blinks (VEOG) were measured by subtracting data of an electrode situated below the left eye from the data above the left eye (Fp1). VEOG and HEOG channels were filtered with a 10 Hz low-pass filter and a 0.1 Hz high-pass filter to facilitate trial-by-trial ocular artifact rejection. An Independent Component Analysis (ICA) was performed to remove blink artifacts. Components related to ocular artifacts were selected by comparing the components identified by the ICA to the EOG signal waveforms and by examining the topography and time course of the components using

the method described by Drisdelle, Aubin, & Jolicœur (2017). Any remaining fluctuation higher than 50 μV within a 150 ms period of the VEOG signal was labeled as a blink that was missed by the ICA procedure (although it could simply be noise from another artifact). Trials containing such fluctuations were removed from the data set. Similarly, segments with an HEOG difference of more than 35 μV over a 300 ms period were considered eye movements and were removed. Data from any channel exceeding ±100 μV during a trial segment was interpolated, up to a maximum of 7 channels in any given trial. Trials with more than 7 channels exceeding this range were rejected. Participants that had more than 40% of trials rejected based on these criteria were excluded from further analysis. This resulted in the exclusion of data from 11 participants in the mask-absent group and 2 participants in the mask-present group. Additionally, three participants in the mask-present group were rejected because they correctly reported T3 in less than 2% of trials were it was present no matter the lag. The other participants correctly reported T3 67% of the time. For these participants, an average of 7.65% of trials were rejected. EEG was segmented based on the onset of S3, with a 100 ms pre-stimulus baseline and a 1000 ms post-stimulus-onset period.

In order to isolate last target-locked P3a and P3b from the overlapping activity caused by the previous stimuli in the RSVP stream, trials where the last target was absent were used. Following the same rational employed in Experiment 1a, by subtracting target absent from target present conditions, it was possible to isolate the P3a and P3b activity related to the processing of the last target. This technique (or close variant) was used in previous related work (e.g., Ptito et al., 2008; Vogel & Luck, 2002). The P3b was measured at electrode Pz and the P3a was measured at Fz. The last target-locked N2pc was measured at PO7/PO8 (where N2pc activity usually reaches its peak), and computed by subtracting electrical activity measured at an electrode

ipsilateral to the attended stimulus from activity measured at a corresponding contralateral electrode. Only the last target was in a lateral location on the screen, ensuring that the stimulus of interest (last target) was the only one eliciting the N2pc component. Since the N2pc component collapse together both the scalp hemispheres, in this experiment was not possible to deconstruct the ERP trough ICA as for the previous experiments.

## 3.1.4 Results

Behavior

Based on the experimental hypothesis, only trials in which the last stimulus was a target were considered for analyses, and the mean proportion of correct report for each target was contingent on the correct report of preceding targets (see Table 3.1). ANOVAs were performed on the mean proportion of correct report for each target as a function of the trial type (two-targets and three-targets trials) and lag (3 vs. 8) as within-subject factors and presence of the mask (mask-present trials vs. mask-absent trials) as a between-subject factor.

| ***Mask Absent*** | *Trial Type* | *Lag* | |
|---|---|---|---|
| *Target* | | *3* | *8* |
| *p*(T1) | Two-targets | .96 | .96 |
| | Three-targets | .82 | .83 |
| *p*(T2\|T1) | Two-targets | .97 | .97 |
| | Three-targets | .88 | .90 |
| *p*(T3\|T1^T2) | Three-targets | .93 | .69 |
| ***Mask Present*** | *Trial Type* | *Lag* | |
| *Target* | | *3* | *8* |
| *p*(T1) | Two-targets | .97 | .96 |
| | Three-targets | .84 | .84 |
| *p*(T2\|T1) | Two-targets | .66 | .78 |
| | Three-targets | .88 | .87 |
| *p*(T3\|T1^T2) | Three-targets | .48 | .75 |

**Table 3.1.** Mean probability of correct report of each target in each condition.
Values in the table are contingent on the correct report of preceding target(s) (e.g., T2|T1 indicates the probability of T2 correct response when T1 is report correctly.

On average, subjects were more accurate in reporting T2 in two-targets trials than in reporting T1 in three-target trials, $F(1,72) = 252.64$, $\eta_p^2 = .642$, $p < 0.001$. That is, report of the first target in the stream was better when there were only two targets shown overall than when there were three. There was no significant difference between lags, however, $F(1,72) = 0.14$, $p = 0.709$, or between mask-present and mask-absent trials $F(1,72) = 0.40$, $p = 0.531$, on accuracy of report of the first target in the stream (T1, in T1-present trials; T2 in T1-absent trials).

An ANOVA was carried out to compare the mean proportion of correct report for T3 (conditional on correct report of the preceding target(s)), as a function of

lag, mask presence, and trial type (two targets and three-targets trials). Last target accuracy was lower at lag 3 than at lag 8, $F(1,72) = 113.46$, $\eta_p^2 = .764$, $p < 0.001$. Last target accuracy was lower for T1-present trials than for T1-absent trials, $F(1,72) = 108.18$, $\eta_p^2 = .420$, $p < 0.001$. T3 accuracy was lower when the last target was followed by a mask compared with the no-mask condition, $F(1,72) = 74.99$, $\eta_p^2 = .768$, $p < 0.001$. Additionally, a significant three-way interaction between these factors was found, $F(1,72) = 30.21$, $\eta_p^2 = .245$, $p < 0.001$. FDR (Benjamini & Hochberg, 1995) corrected *t* tests with indicated that there was no lag effect for two-targets trials when no mask was present, $t(39) = 0.73$, $p = 0.471$, while reliable lag effects were found for two-targets trials when a mask was present, $t(33) = 5.89$, $\eta_p^2 = .648$, $p < 0.001$. Lag effects, though of different magnitude, were detected both when the mask was present, $t(33) = 10.99$, $p < 0.001$, and when the mask was absent, $t(33) = 3.01$, $p = 0.005$. Results indicated that, when the masking is absent, an AB effect was only found for three-target trials, according to the results found in Experiment 1a, and converging with prior studies (Giesbrecht & Di Lollo, 1998; Jannati et al., 2011, 2012; Sessa et al., 2007). As expected, in mask-present trials the AB deficit (lower accuracy for lag 3 trials vs. lag 8) was more pronounced when more targets were presented (three-targets vs two-targets trials) $t(33) = 7.37$, $p < 0.001$.

ERP

P3b

Figure 3.2 shows the grand average P3b difference waves (last target-present minus last target-absent) for the various trial types at Pz, while Table 3.2 lists the mean amplitudes of these waveforms. Amplitudes were obtained by measuring the mean amplitude in a 150 ms window centered on the peak of the waveform in mean

grand averages for each condition. Peak amplitudes were reached at the following time points for mask-absent trials conditions: two-targets trials, lag 3 = 507 ms; two-targets trials, lag 8 = 457 ms; three-target trials, lag 3 = 566 ms; and three-targets trials, lag 8 = 490 ms. For mask-present trials, mean peak amplitudes were reached at the following time points: two-targets trials, lag 3 = 537 ms; two-targets trials, lag 8 = 523 ms; three-targets trials, lag 3 = 701 ms; and three-targets trials, lag 8 = 597 ms. The amplitudes for each subject were submitted to an ANOVA that considered lag (3 vs. 8) and trial type (two-targets trials vs. three-targets trials) as within-subject factors, and masking (mask-present vs. mask-absent trials) as a between-subject factor. The mean amplitude of the P3b component was significantly smaller at lag 3 than lag 8, $F(1,72) = 34.12$, $\eta_p^2 = .367$, $p < 0.001$. The P3b had a smaller amplitude for mask-present then mask-absent trials, $F(1,72) = 17.91$, $\eta_p^2 = .521$, $p < 0.001$. However, there was no significant amplitude difference between two-targets and three-targets trials, $F(1,72) = 0.40$, $p = 0.52$. There were no significant interactions in the analysis, all $p$s > 0.09.

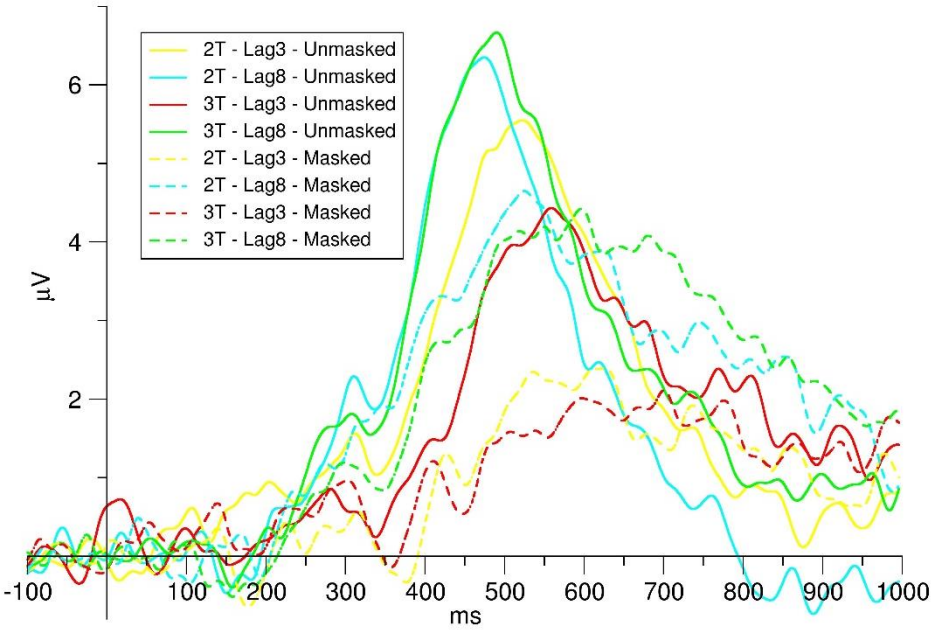


**Figure 3.2.** P3b waveforms. Grand average ERP difference waves (last target-present minus last target-absent trials) showing the P3b component at Pz electrode site for lags 3 and 8, two-target and three-target trials, for the mask-absent group and the mask-present group.

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | 5.18 | 5.52 |
| Three-targets | 4.23 | 5.92 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | 2.34 | 4.11 |
| Three-targets | 2.32 | 4.14 |

**Table 3.2.** P3b amplitude. Mean P3b amplitudes (μV) (last target-present minus last target-absent trials).

Mean P3b latencies, estimated using a jackknife approach (Kiesel et al., 2008; Ulrich & Miller, 2001) with individually derived values using the solution proposed by Brisson and Jolicœur (2007) and Smulders (2010), were also compared (see Table 3.3). Latency values were calculated as the time-point when individual jackknife waveforms reached 50% of the area under the curve (for values above 0 μV) in a 190–990 ms window from the onset of the last target. The same ANOVA model as described above was used. P3b latency was significantly delayed for lag 3 trials compared to lag 8 trials, $F(1,72) = 47.86$, $\eta_p^2 = .427$, $p < 0.001$. P3b latency was also longer for three-targets trials compared to two-targets trials, $F(1,72) = 29.02$, $\eta_p^2 = .318$, $p < 0.001$. Furthermore, P3b latency was longer for mask-present trials compared to mask-absent trials, $F(1,72) = 29.64$, $\eta_p^2 = .531$, $p < 0.001$. There were no significant interactions between these factors, all *p*s > 0.2. As expected, the P3b results converge with Experiment 1 results, and further them indicating that encoding of a target is less efficient and delayed in the presence of a mask.

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | 530 | 486 |
| Three-targets | 593 | 519 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | 627 | 589 |
| Three-targets | 667 | 628 |

**Table 3.3.** P3b amplitude. Mean P3b latency (ms) (last target-present minus last target-absent trials).

P3a

Figure 3.3 shows the grand average P3a difference waves (last target-present minus last target-absent) at Fz. Mean amplitudes estimated using a window between 270 and 320 ms are listed in Table 3.4. The amplitude was larger for lag 8 trials compared to lag 3 trials, $F(1,72) = 7.65$, $\eta_p^2 = .471$, $p = 0.007$, the difference between two-targets and three-targets trials did not reach significance, $F(1,72) = 2.66$, $p = 0.108$. There was a marginally significant interaction between lag and trial type, $F(1,72) = 3.61$, $\eta_p^2 = .347$, $p = 0.061$. For lag 3, more targets (T1-present) meant a smaller amplitude whereas the opposite was found for lag 8. Finally, P3a amplitude was larger for mask-absent trials than for mask-present trials, $F(1,72) = 7.11$, $\eta_p^2 = .418$, $p = 0.009$. There were no other significant effects in the analysis, all $p$s > 0.102. Results indicate that masking makes engagement of attention less efficient during the AB.

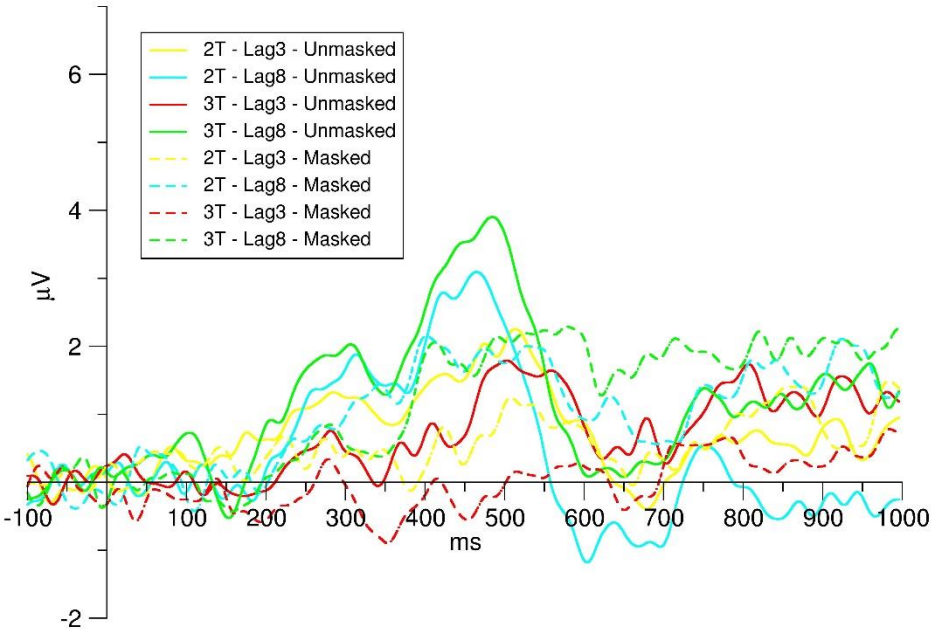


**Figure 3.3.** P3a waveforms Grand average ERP difference waves (last target-present minus last target-absent trials) showing the P3a component at Fz electrode site for lags 3 and 8, two-targets and three-targets trials, for the mask-absent group and the mask-present group.

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | 1.02 | 1.11 |
| Three-targets | 0.49 | 1.65 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | 0.51 | 0.97 |
| Three-targets | –0.16 | 0.46 |

**Table 3.4.** P3a amplitude. Mean P3b amplitudes (µV) (last target-present minus last target-absent trials).

N2pc

Figure 3.4 displays the grand average contralateral minus ipsilateral waveforms for T3-present trials for two-targets and three-targets trials, for each lag, and each group (mask-absent vs. mask-present). N2pc onset was about the same for the two masking conditions, but the peak amplitude and duration of N2pc was clearly different across masking conditions. For this reason, the window used to estimate the mean amplitude of N2pc was different for the two groups (200–300 ms for the mask-absent group, and 170–270 ms for the mask-present group; which was a 100 ms window around the approximate peak in the grand average difference waves). The mean amplitudes for each participant for each condition were submitted to an ANOVA with the same model as for the P3 analyses. The overall means are shown in Table 3.5.

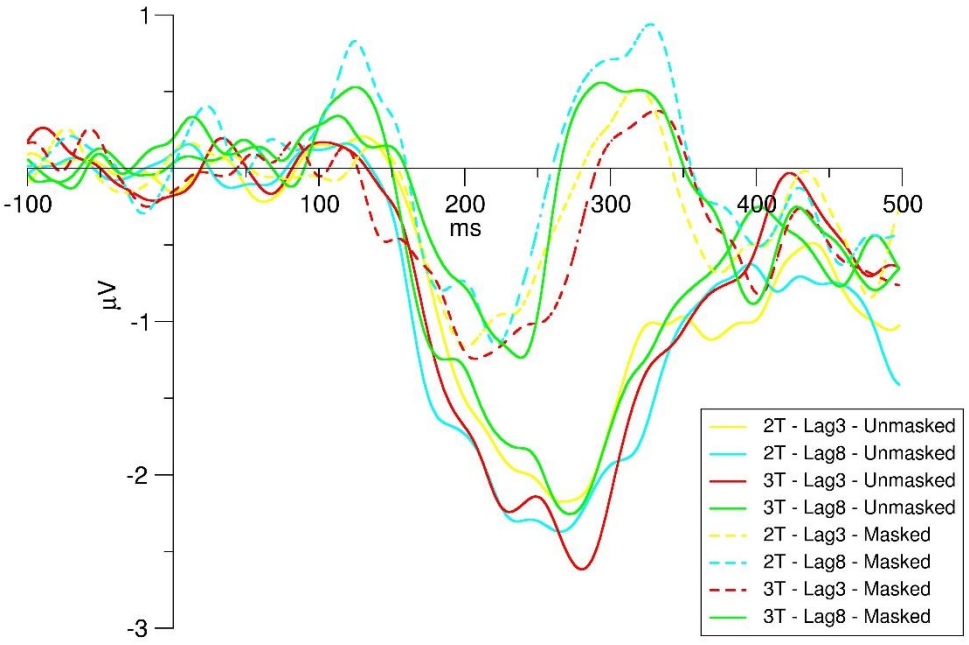


**Figure 3.4.** N2pc waveforms. Grand average ERP difference waves (contralateral minus ipsilateral) showing the N2pc component at PO7/PO8 electrode sites for lags 3 and 8, two-targets and three-targets trials, for the mask-absent group and the mask-present group (for three-targets trials only).

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | –2.23 | –1.88 |
| Three-targets | –1.94 | –1.58 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | –1.01 | –0.77 |
| Three-targets | –0.83 | –0.66 |

**Table 3.5.** N2pc amplitude. Mean N2pc lateralized amplitude (μV) (contralateral minus ipsilateral) for lags 3 and 8, T1-absent and T1-present trials, as well as for trials with a mask compared to trials without a mask.

Although an N2pc for T3-absent trials was found, given the presence of the selection in the last stimulus frame, preliminary analyses showed that N2pc was much smaller for T3-absent trials than for T3-present trials, presumably because attention could be quickly disengaged when the last stimulus contained a distractor (T3-absent) rather than a target (T3-present). The much-reduced N2pc for T3-absent trials made it difficult to examine differences in N2pc as a function of other task variables. For that reason, following analyses were focused on T3-present trials. N2pc amplitude did not vary significantly between lags, $F(1,72) = 1.78$, $p = 0.186$, or between three-targets and two-targets trials, $F(1,72) = 1.72$, $p = 0.193$. However, as is apparent upon inspection of Figure 3.4, N2pc amplitude was larger for mask-absent relative to mask-present trials, $F(1,72) = 15.28$, $\eta_p^2 = .414$, $p < 0.001$. The analysis of N2pc amplitudes did not detect other significant effects, all $p$s > 0.18.

Visual inspection of Figure 3.4 also revealed marked differences in the offset of N2pc for mask-present and mask-absent trials. An ANOVA on jackknife latency estimates (see Table 3.6) confirmed this, $F(1,72) = 14.81$, $\eta_p^2 = .187$, $p < 0.001$.

However, N2pc offset latency did not vary between lags, $F(1,72) = 0.001$, $p = 0.975$, nor across two-target vs. three-target trials, $F(1,72) = 0.14$, $p = 0.709$. There were no significant interactions between these factors, min $p > 0.20$.

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | 307 | 304 |
| Three-targets | 305 | 325 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | 263 | 252 |
| Three-targets | 243 | 238 |

**Table 3.6.** N2pc latency. Mean N2pc latency (ms) (contralateral minus ipsilateral) for lags 3 and 8, two-targets and three-targets trials, as well as for trials with a mask or trials without a mask.

N2pb

Given the absence of AB-related effects on the N2pc, I tried to assess whether any bilateral modulation in the N2 time range was present. In this case, the N2pb data was the average of the contralateral and the ipsilateral ERPs used to estimate the N2pc (in the latter case, the ipsilateral ERP was subtracted from the contralateral one). For this reason, by submitting the N2pb data to an ANOVA which consider as predictors the same variables of the other analysis (trial type, lag, masking). In this way, it was possible to investigate also the bilateral modulations of the ERP.

The mean amplitudes for each condition are shown in Table 3.7. N2pb was larger (more negative) at lag 8 than at lag 3, $F(1,72) = 10.06$, $\eta_p^2 = .301$, $p = .002$. N2pb

was also more negative for three-targets trials than for two-targets trials, $F(1,72) = 141.65$, $\eta_p^2 = .210$, $p < .001$. Furthermore, these factors interacted $F(1,72) = 8.03$, $\eta_p^2 = .249$, $p = .006$. To further understand this interaction, the amplitude of the N2pb across two-targets and three-targets trials was considered in separates *t*-tests, finding that only trials at lag 8 and showed a significantly larger amplitude for three-targets trials $t(73) = 8.74$, $p < .001$. At lag 3, the amplitude was also larger in three-targets trials $t(73) = 9.53$, $p < .001$. Although the main effect of mask was not significant, $F < 1$, it interacted with lag, $F(1,72) = 5.21$, $\eta_p^2 = .373$, $p = .025$, and an independent sample *t*-test revealed that the decrease in N2pb amplitude from lag 3 to lag 8 was larger when the last target was followed by a mask than when it was not masked $t(73) = 3.03$, $p = .003$.

| ***Mask Absent*** | *Lag* | |
|---|---|---|
| *Trial Type* | *3* | *8* |
| Two-targets | 1.70 | 0.89 |
| Three-targets | –0.69 | –0.50 |
| ***Mask Present*** | *Lag* | |
| *Trial Type* | *3* | *8* |
| Two-targets | 2.30 | 0.15 |
| Three-targets | 0.46 | –1.24 |

**Table 3.7.** N2pb amplitude, for lags 3 and 8, two-targets and three-targets trials, for trials with and without a mask presented at the end of the RSVP.

### 3.1.5 Discussion

In these two experiments, the role of masking in the spatial deployment of attention, attentional engagement, and encoding of representations in working memory during the AB were investigated. To do so, an AB paradigm containing letters (targets) among distractors (digits) was employed. Each RSVP could be composed by two or three stimuli to attend, and the lag between the last stimulus and the previous(es) could be 2 or 7 distractors. Finally, a between subjects experimental manipulation consisted in the absence or presence of a masking after the last stimulus of the RSVP. All the ERP analysis has been time-locked to the onset of the last stimulus. Together, these manipulations produced a rich set of experimental conditions, enabling an examination of the impact of masking and processing load on the AB, both on behavior and on ERP isolated from concurrent measurements of the EEG during the task.

Behaviorally, when the last target was masked, an AB effect (i.e., a reduction in the accuracy of report of the last target at lag 3 vs. lag 8) was observed both in two-targets and three-targets trials, and this effect was larger for three-targets trials. When the last target was not masked, two-targets trials no longer showed a behavioral AB effect while traces of an AB effect were still detected in three-targets trials. The absence of an observable behavioral AB effect for mask-absent trials might be a consequence of the high accuracies, which might have led to a ceiling effect. It is therefore difficult to interpret the absence of AB deficit under such conditions. An experiment by Nieuwenstein et al. (2009) demonstrated that an AB deficit can be generated even when distractors are replaced by blank intervals. This suggests that masking might not be necessary in order to observe AB deficits. Moreover, the accuracy in reporting the last target was lower in three-targets trial compared to two-target trials. These results replicated the findings of Experiment 1a, extending the evidence in

relationship with a lateralized visual information. Importantly, accuracy was significantly impacted by the presence of a backward mask, making it harder for participants to report targets when a mask followed the last target, and masking magnified the AB effect, as expected from previous research.

The electrophysiological measures showed a smaller P3b amplitude for short lag trials as was previously reported in Experiment 1a (and in other studies: Vogel et al., 1998; Vogel & Luck, 2002). This component was however not completely suppressed when the mask was present as was reported by Vogel and Luck (2002). This allowed us to observe a lag effect on latency of P3b in both the mask-absent and mask-present.

The onset of the P3b was later for a target presented at lag 3 compared to lag 8. As reported in the Experiment 1a, increasing the number of initial targets (T1-present compared with T1-absent) also delayed the P3b. These finding complement previous research suggesting that the P3b reflects processing in a capacity-limited mechanism, which is either delayed, and/or slowed, under some conditions. Importantly for this experiment, using a backward mask, a similar pattern as in Vogel and Luck's (2002) study, was observed. Namely, a significant decrease in amplitude of the P3b in the short lag condition. It is possible that this attenuation in amplitude was not pronounced as the one found by Vogel and Luck (2002) because of the use of a mask consisting of a small checkerboard, which perhaps approached masking by noise rather than by pattern. This result, however, suggests that the type of backward mask (at least between pattern and noise masks, at the target-mask SOA that was used) may not affect encoding in working memory differently, both of then making memory encoding a less efficient process. The backward mask delayed the P3b by 70–100 ms (depending on experimental conditions). It is very likely that this effect could be observed for other

types of masks, as long as the strength of masking did not completely suppress the P3b, as occurred in the Vogel and Luck (2002) study. The present findings help to clarify the role of masking in the AB paradigm and refine the understanding of capacity limitations underlying the AB. Masking causes a delay of the P3b, suggesting that processing masked targets is less efficient than processing targets that are not masked. Processing a masked stimulus, under AB load, is slowed, or perhaps even postponed, which can be observed in delayed latency of relevant ERP components, as well as increased response times when the paradigm involves speeded responses (Jolicœur & Dell'Acqua, 1998).

The impact of masking on the frontal engagement of selective attention in the AB was investigated as well. The effects on the P3a found in the Experiment 1 were replicated successfully. Namely, P3a elicited in longer lag trials is larger in amplitude compared to shorter lag. Importantly, both for mask-present and mask-absent trials, lag effect on the P3a amplitude elicited by the last target was more pronounced in three-targets trials compared to two-targets trials). In three-targets masked trials, when the last target is presented at lag 3, there was a particularly large lag effect leading to a complete suppression of the P3a. Total encoding load, therefore, does affect selective engagement of attention on a subsequent target by exacerbating the lag effects during the AB. Of particular interest was the significant effect of the presence of the mask in suppressing the amplitude of the P3a response. As suggested in previous literature, the P3a represents attentional engagement (Barceló et al., 2006), and these results would be consistent with models suggesting that AB reflects greater selection difficulty under some conditions. In the episodic simultaneous type, serial token model (Wyble et al., 2009) for instance, during encoding of the first target, attention is inhibited so as to avoid distractor interference. This inhibition would impede the selection of the blinked target (see also Wyble et al., 2011).

The effect of masking on the N2pc during the AB was investigated. In contrast with several earlier studies, a lag effect on N2pc amplitude was absent in these two experiments. There was also no effect of the number targets presented in the RSVP (i.e., no difference in the N2pc elicited by the last target in two-targets vs. three-targets trials), despite clear effects of this manipulation on accuracy, and on P3b latency as well as a trend, that did not quite reach significance, towards a trial type effect for P3a amplitude, reproducing patterns found in the Experiment 1a. These results were unexpected, and there is no clear-cut explanation for the apparent discrepancies with previous works (Akyürek et al., 2010; Jolicœur et al., 2006; Pomerleau, Fortier-Gauthier, Corriveau, McDonald, et al., 2014). Although many aspects of these experiments were similar to previous ones, one difference that was possibly responsible for this is the fact that the present experiment used three RSVP streams in order to mask the last lateralized target. The literature on the AB shows that dividing attention by adding an irrelevant task could reduce the AB (Olivers & Nieuwenhuis, 2005). Perhaps the three RSVP design created a divided attention situation since the side RSVP streams required attention but where irrelevant until the very end. This possibly reduced the AB impact on attentional deployment. Given the speed of presentation of the stimuli in the RSVP streams, it is possible that participants adopted a strategy of initially attempting to encode the stimuli on both side, before zeroing in on the target side (based on the color cue). Perhaps the most striking result regarding the N2pc was the impressively large effects of masking on N2pc amplitude and duration. The trailing mask appeared to limits the duration of useful processing of the target, reflected in a shorter N2pc duration and lower overall amplitude, compared to the no-mask condition. The attenuation and early termination of the N2pc is consistent with the hypothesis that masking curtailed the attentional deployment on the last target, slowing and attenuating

downstream processing (reflected in poor performance and attenuated and delayed P3 responses). Considering this, processing of a masked last target would produce a representation that may be more vulnerable to interference and it is therefore not surprising that the accuracy in reporting such target is lower during the AB.

Of paramount importance, an examination of the N2pb component showed that, differently from what was expected, bilateral posterior modulations in the N2 time domain was associated to the trial type (two-targets vs three-targets trials), the lag, and the presence or absence of a masking as well. Since these modulations were bilateral (i.e., identical both for the contralateral and the ipsilateral portion of the subtracted ERP) they were covered in the subtracted N2pc.

N2pb has been involved in previous work examining visual-spatial processing (e.g., Schubö, Schröger, & Meinecke, 2004). This result showed for the first time that reducing the lag to previous targets to be encoded in the AB paradigm decreases the N2pb amplitude. This modulation is in line with the decrease in amplitude of both P3a and P3b component. In other words, temporal dynamics of visual spatial attention suffer from a limited capacity mechanism as the frontal detection and the encoding. Interestingly, while reducing lag decreased N2pb (making it less negative, or, more positive), the amplitude of P3a and P3b was reduced with a shorter lag (i.e., P3b became less positive). This is an important finding because it suggests the reduced N2pb cannot be explained by volume conduction of the effects on P3a and P3b.

The effects of lag on N2pb likely reflect some impairment of processing, maybe stimulus categorization, during the AB. To my knowledge, no other AB study investigated the N2pb, and the present results suggest that further studies examining the N2pb are likely to be useful. Interestingly, N2pb locked to the last target was significantly larger (more negative) when there were more leading targets (three-targets

condition). This finding suggests that N2pb reflects total processing load, and increasing the amount of information to be processed increases N2pb. As for the lag effect, the N2pb modulations seems to be not related to the volume conduction of the other components.

Importantly, although the early attentional component of processing was apparently shortened by the mask, later downstream processing was probably lengthened, assuming the representation of the masked target was not completely eliminated. It is interesting to consider these results in relation to the role of the distractor following the first target plays in the AB. Raymond et al. (1992) found a larger AB when T1 (they only had two-target trials) was followed by an immediate distractor, suggesting a lengthening of capacity-demanding processing of T1 (e.g., Jolicœur, 1999b). Brisson et al. (2010) found that masking T1 reduced the amplitude of the P3b, as it did for masking T2 (their last target). According to the authors these findings show that masking T1 could reduce processing efficiency, leading to greater cost for the last target. Maybe the N2pc and P3a results obtained by masking the last target would also be similar to that for a masked first target. As seen in the present work, masking the last target had a big impact on its processing but perhaps masking the first target also does and this effect might carry-over to the last target and accentuate downstream masking effects. While some authors give a central role to masking in the AB, some suggest that masking might not be necessary in order to observe AB deficits. This would explain how an AB can be measured when distractors are replaced by blank intervals (Nieuwenstein et al., 2009). It is however undeniable that masking played an important role in behavioral deficits in this experiment. It has been suggested in several studies (e.g., Ouimet & Jolicœur, 2007; Vogel & Luck, 2002) that the AB is a consequence of limited-capacity of the underlying mechanisms leading to the AB.

## 3.2. Conclusion: Chapter 3

In the experiment presented in this Chapter the influence of the AB on visual spatial attention was investigated. As previously reported (e.g., Dell'Acqua et al., 2006), it was already known that there is a suppression of the N2pc –a component which reflect the deployment of attention in space– when a lateral target is presented in a critical position within an RSVP. As hypothesized, the same pattern of Experiment 1a of the P3a and P3b elicited by the last target of an RSVP emerged. Specifically, the P3a component (detection) was decreased in amplitude and the P3b (encoding) was both decreased and postponed in latency at short relative to long lags. This suggests that, regardless the position of a target, mechanisms which drive temporal dynamics of attention are the same for both foveal and parafoveal information.

Differently from what expected, no N2pc modulations related to the AB experimental manipulations were found. Of interest, visual spatial attention is disrupted by the AB over both the brain hemispheres. Indeed, N2pb component modulates related to number of target presented within the RSVP and also the lag between the last target and the previous(es). This means that the cognitive disruption due to the AB exert a general effect on the visual spatial function domain, and not a specific effect related to the contralateral (that is supposed to be related to target processing, e.g., Eimer, 1996) or ipsilateral (that could be a marker of distractor suppression, e.g., Hickey et al., 2009) portion of the N2pc.

Overall, this evidence highlighted a common base between foveal and parafoveal attention. A limit of this argument is that this common base is presently supported someway indirectly, namely, through an AB task. This point raises the necessity to understand the origin of this evidence: the two experiments presented in the next Chapter try to answer to this question by using a visual search task. Differently

from usual visual search tasks, here a target could appear also on the midline (and not only in lateral positions). The reason of this manipulation is to compare contralateral and ipsilateral activity of the N2pc (elicited by lateral targets) with the bilateral activity of the N2pb (elicited by a target presented on the midline). In this last case, no differences between the two scalp hemispheres emerge, and this activity (that as for the N2pc is the average between two parieto-occipital electrodes such as P7 and P8, or PO7 and PO8) can be compared to the contralateral and ipsilateral portion of the N2pc. In other words, N2pb activity is now used as a baseline for the contralateral and ipsilateral portion of the N2pc; so, the foveal attention served as a baseline for parafoveal attention. Based on my knowledge on this topic, there are no previous evidence which yield to make a prediction regarding the comparison of N2pb and contralateral and ipsilateral portion of the N2pc. Nevertheless, data could provide important evidence related to this issue.

# Chapter 4

## 4.1 Experiment 3a

### 4.1.1 Introduction

Experiment 2 showed that visual attention, when measured separately over the bilateral parieto-occipital scalp portion, is prone to AB-related modulations which are similar to the ERP components previously studied using a standard RSVP (when the stimuli are all presented in the center of the screen). This is in line with a hypothesis tested in my thesis, namely, foveal and parafoveal visual attention are guided by the same mechanisms.

As explained in the General Introduction, a lateral stimulus presented among distractors elicit a lateralized component called N2pc, namely a posterior N2 component which is more negative over the parieto-occipital portion of the scalp contralateral to the target presentation (Luck & Hillyard, 1994a; b). More than two decades of literature suggested that the contralateral portion of this component reflect the selection of the target (i.e., Eimer, 1996; Mazza & Caramazza, 2011) and the ipsilateral portion reflect the suppression of the irrelevant information (i.e., Hickey et al., 2009).

The aim of the experiments presented in this chapter is an investigation of the lateralization of posterior ERP in the N2 time range by following a different strategy, namely, to compare the difference between the ERP activation of a lateral target and a target presented on the midline. As mentioned previously, a target presented in the midline elicit a bilateral N2 component called N2pb (Luck & Hillyard, 1994a; Simson, Vaughan, & Walter, 1976). This component can be considered as the average

of the ERP elicited by two lateralized electrodes, namely, the same two electrodes considered to compute the N2pc (P7 and P8 or PO7 and PO8). The contralateral and ipsilateral portion of the N2pc are also an average of two electrical signals: the average activity of a left electrode (when a target is presented on the right visual hemifield) and a right electrode (when a target is presented on the left visual hemifield) is a contralateral signal. By following the same rationale, the same average of electrodes' activity can be also an ipsilateral signal. By comparing the activity of N2pb (when the target is on the midline), and the contralateral and ipsilateral portion of the N2pc (when the target is presented on the lateral portions of the visual field) it will be possible to test further what there is in common between lateralized and non-lateralized deployment of attention.

To my knowledge, it is hard to do predict a precise outcome. One could hypothesize that, compare to a midline presented target, a lateral one should elicit both a more negative contralateral activity and a more positive ipsilateral activity. This would be in line with the Nt (target negativity) and Pd (distractor positivity) components proposed by Hickey and colleagues (2009): the more negative contralateral activity would reflect the lateral target selection, and the most pronounced ipsilateral activity would reflect the distractor suppression. This scenario cannot be priory excluded, although it would be a result that hardly converge with the fact that parafoveal attention are prone to the same AB behavioral failure and ERP patterns of foveal RSVP presentations. In other terms, since the ERP modulations found in Experiment 2 are bilateral, and since a single RSVP does not present distractors at the same time of T2 (when T2 is within the AB time window), the attentional disruption related to the AB for lateral target is only related to target and not distractors. If the commonality between foveal and parafoveal AB effect is only related to the target processing, it is more

probable that the outcome of the experiments that follow will show similar ERPs for midline target (N2pb) and target-related activity of a lateralized to-be-detected target (contralateral portion of the N2pc).

## 4.1.2 Methods

### Subjects

Eighteen students at the University of Padua (14 females) participated in the experiments after giving informed consent. Their mean age was 23.1 years (SD = 3.2) and all subjects had normal or corrected-to-normal visual acuity. Three subjects were removed from the final sample because of low overall accuracy (less than 50%, one subject) or high percentage of EEG epochs discarded due to eyes blinks and EEG artifacts (more than 40%, two subjects).

### Stimuli and procedure

Equiluminant stimuli (8.9 cd/$m^2$) were presented on a 19-inch CRT monitor at a viewing distance of 57 cm on a black background. Twelve circles (.5° radius) were presented spaced around an imaginary circle (3.5° radius) centered on a fixation cross, eleven of them were grey (RGB = 140,140,140) and the remain one was blue (RGB = 0,110,255) or green (RGB = 52,137,0; counterbalanced). A bar was presented inside each circle; it was .8° long and was tilted 45° clockwise (“slash”) or counterclockwise (“backslash”) relative to vertical. Each trial began with a grey fixation cross (RGB = 140,140,140) displayed during the whole trial, and after a random time interval between 900 and 1100 ms the visual search array was presented for 150 ms. The task was to report, as fast as possible while keeping errors to a minimum, whether the bar inside the target circle was a slash or a backslash by pressing the C key or the M key

(counterbalanced) with the index fingers, on a keyboard placed in front of them. After the response, the fixation cross disappeared and accuracy feedback ("OK" or "WRONG") was presented in the center of the screen. After 20 trials of practice, each subject performed 600 experimental trials, in which the target presence was equally distributed on left, midline, or right side of the visual search array. An example of a trial is illustrated in Figure 4.1.

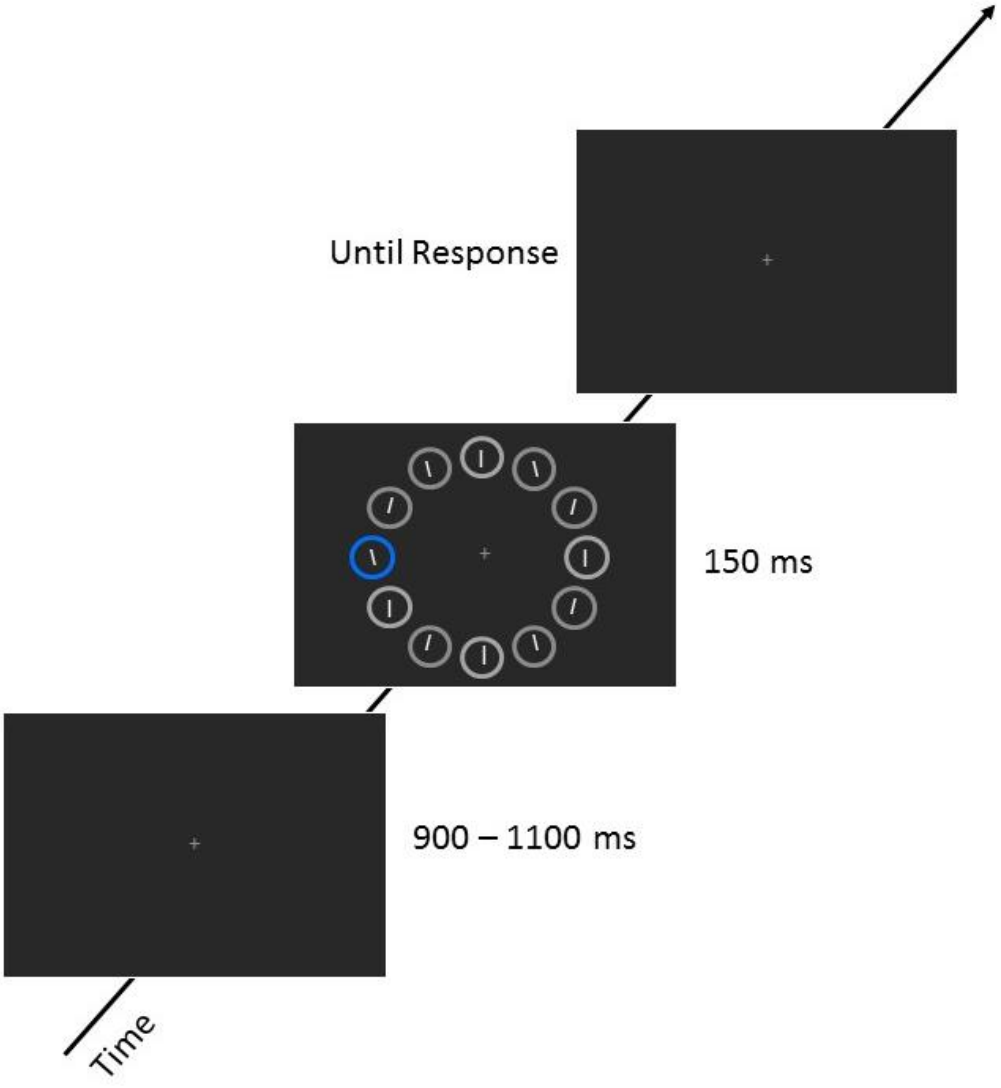


**Figure 4.1.** Example stimulus in Experiment 3a. After a random jittered interval of 900 – 1100 ms, the visual search array appears for 150 ms.

### 4.1.3 EEG Recordings and Analysis

EEG activity was recorded continuously from 28 active electrodes positioned according to the International 10/10 system (Fp1, Fp2, Fz, F3, F4, F7, F8, FCz, C3, C4, Cz, CP1, CP2, CP5, CP6, P3, P4, Pz, O1, O2, Oz, T7, T8, TP9, PO9, PO10, P7, and P8 sites) placed on an elastic ActiCap (Brain Products, München, Germany), referenced to the left earlobe. An electrode was also used to record activity at the right earlobe. HEOG activity was recorded from electrodes positioned on the outer canthi of both eyes. VEOG activity was recorded from two electrodes, above (Fp1) and below the left eye. Impedance at each electrode site was maintained below 10 KΩ. EEG, HEOG, and VEOG activities were amplified, filtered using a band-pass of 0.016–80 Hz, digitized at a sampling rate of 500 Hz, and referenced offline to the average of the left and right earlobes. Independent components analysis (ICA, Delorme & Makeig, 2004; Jung et al., 2000) was used to identify blink and saccade components in the continuous EEG recordings and remove them from the data (Drisdelle et al., 2017). The corrected EEG was high-pass filtered at 0.1 Hz and low-pass filtered at 20 Hz and then segmented into 700 ms epochs starting 100 ms before the onset of the last character in the RSVP stream and ending 600 ms after and baseline-corrected using the mean activity in the interval [−100, 0] ms. To ensure no residual artifacts remained on the EOG channels, each segment was examined in the interval [−100, 600] ms relative to the onset of the visual search array for voltage deviations greater than 80 μV in any period of 150 ms for the VEOG difference waveform or a deviation greater than 45 μV in any 300 ms period for the HEOG difference waveform. Segments with residual ocular artifacts were removed from the data set. EEG channels were flagged when the signal exceeded ±100 μV anywhere in the analysis segment. If a segment had seven or fewer flagged data channels, these channels were interpolated using a spherical spline

interpolation algorithm in EEGLAB (Delorme & Makeig, 2004), for that segment, otherwise the segment was rejected.

The ERPs of P7 and P8 electrodes were split across conditions (lateral target vs. midline target) and averaged to produce three ERPs: the contralateral and ipsilateral responses for lateral targets and the average response for midline targets. These ERPs will henceforth be referred as levels of the 'target position' variable. The mean amplitude of these three N2 ERP components were quantified as the mean value in a 200–300 ms time window from the onset of the search array.

## 4.1.4 Results

Behavior

An ANOVA was performed on the mean proportions of correct response for each presentation side (left = .85, midline = .89, and right = .86) showing a significant difference among the three performances, $F(2,28) = 7.11$, $p = .003$, $\eta_p^2 = .337$. Post-hoc FDR (Benjamini & Hochberg, 1995) corrected *t*-tests showed that the only significant differences were between the accuracy in reporting a target presented on the midline against one in the left visual hemifield ($t(14) = 3.5$, $p = .001$) or in the right ($t(14) = 3.0$, $p = .007$). No significant difference was found between the accuracy in reporting a target in left compared to one in the right ($t(14) = .5$, $p = .88$).

The same analysis was conducted on the mean reaction times (RT; left = 615 ms, midline = 607 ms, and right = 645 ms), finding no significant differences among them, $F(2,28) = .53$, $p = .6$.

ERPs

Figure 4.2 shows the ERP waveforms for the three main conditions (ipsilateral, contralateral, midline). Statistical analysis shown that the averaged P7/P8 activity was different across the target position levels ($F(2,28) = 23.12$, $\eta p2 = .623$, $p < .001$), which has been examined in greater detail with pairwise FDR (Benjamini & Hochberg, 1995) corrected *t*-tests: no significant difference was found between Contralateral (.21 µV) versus Midline (.15 µV) activity: $p = .98$. However, the Contralateral (.21 µV) voltage was more negative compared with the Ipsilateral (1.29 µV) voltage: $t(12) = –6,18$, $p < .001$; and the Midline (.15 µV) voltage was also more negative than the Ipsilateral (1.29 µV) voltage: $t(12) = –5.55$, $p < .001$. significant differences emerged, as suggested in Figure 4.2.

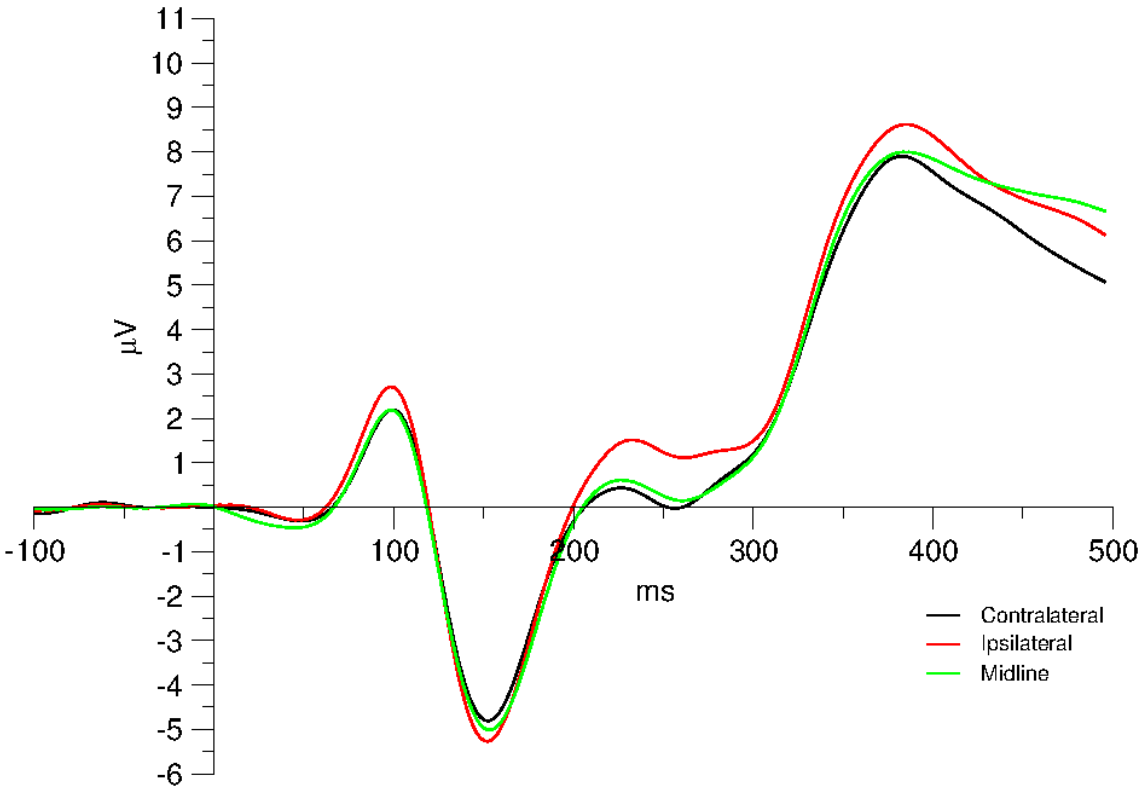


**Figure 4.2.** Results from Experiment 3a. Grand average waveforms for the three levels of target position (ipsilateral, contralateral, midline ERP).

## 4.1.5 Discussion

To further investigate how visual attention is deployed within the visual space, this experiment compared the parieto-occipital scalp activity in the N2 time range during a visual search task. The average of bilateral electrical activity elicited by a midline presented target and the contralateral and ipsilateral activity elicited by a lateral target were compared. Results show a complete similarity between the contralateral activity elicited by a lateral target and the bilateral activity elicited by a midline target. This could mean that while we attend to an object presented in front of us, activity in both the hemispheres shifts negatively, compared to cases in which the relevant information is lateral, in which case only the contralateral hemisphere shows a negative shift, suggesting a dominance of contralateral processing. The bilateral processing related to a midline target could explain the higher accuracy for midline targets compared with lateral targets found in the behavioral data.

The electrophysiological data, moreover, seems to be in contrast with the hypothesis for which the ipsilateral component of the N2pc is a marker of distractor suppression (Hickey et al. 2009), because of its absence when the target is presented on the midline. If the distractor-related activity in this condition would be covered by other signal (namely, the target-related bilateral ERP), there should be a difference between the contralateral and the midline signal, because –differently from the former– the latter should reflect bilaterally both distractors- and target- related activity.

Luck & Hillyard (1994a) reported an interesting result regarding N2pb and N2pc: namely, the authors found that the N2pb amplitude is sensitive to target presentation probability whereas N2pc is not. This is probably one of the first evidence which suggested that the attentional mechanisms reflected by these components are different. Probably due to this, also the opinion for which foveal and parafoveal visual

attention is mediated by difference cognitive functions raised. Nevertheless, there is an important caveat regarding the results by Luck & Hillyard (1994a), namely, the authors consider the N2pc as a unique subtracted value. As in Experiment 2 (Chapter 3) of these thesis, the subtraction of the contralateral and ipsilateral subcomponent of the N2pc could have hidden a bilateral modulation related to the target presentation probability. Although the reason for which many studies considered the subtracted N2pc instead of separate contralateral and ipsilateral subcomponent is probably related to convenience: indeed, by using two separates component would double the ERP plotted and also add an additional independent variable in statistics (namely, contralateral vs. ipsilateral). On the one hand one could argue that the subtracted N2pc is sufficient to detect when the difference between the contralateral and ipsilateral changes among conditions. On the other hand, this solution prevents to assess whether the condition-related ERP modulation is associate to the contralateral or ipsilateral subcomponent and, if they reflect two different brain mechanisms, it prevents to understand which brain mechanism change in relation to an experimental manipulation.

By considering the midline-related activity as a baseline for the parafoveal deployment of visual attention, it seems that only the ipsilateral portion of the N2pc contributes to unbalance the posterior hemispherical electrical activities. Nevertheless, this experiment does not give the possibility to compare the bilateral activity elicited by a target presented on the midline with any kind of baseline condition, in which there is no shifts of attention or focusing on the salient information.

To investigate this issue, the current experimental design was modified in order to obtain conditions in which there is no focusing of a target, although there is a visual search activity of a comparable difficulty to a target present condition.

## 4.2 Experiment 3b

### 4.2.1 Introduction

The second experiment of this chapter was implemented to further the results of Experiment 3a. Namely, the experimental design was modified in order to obtain a control condition of the previous study. The changes allowed to create three control conditions, with three different level of difficulty. Differently from Experiment 3a, due to the greater number of conditions, a multiple frame procedure (MFP, Aubin & Jolicœur, 2016; Drisdelle et al., 2017; Pomerleau, Fortier-Gauthier, Corriveau, Dell'Acqua, & Jolicœur, 2014) was employed. By using the MFP, instead of a single visual search array per each trial, six consecutive visual search arrays (also called frames or subtrials) are presented. At the end of the six frames, participants are asked to report in how many visual search arrays the target was present. Since the number of responses decrease (namely, a single response instead of six), through the MFP is possible to increase the number of trials in the same amount of experimental session's time, and afford a greater number of experimental conditions.

Although the presence of twelve placeholder as in Experiment 3a, another critical manipulation was the use of a heterogeneous visual search. Namely, when a target was presented, other three distractor circles were printed in three different colors. This was done because, when the target color was absent, four different distractor colors were presented, and subjects still had to search in the visual array. Moreover, another target absent condition consist in the presence of four same color distractors, which suggests a weaker activation of visual search mechanisms due to the ease of the task. Finally, a condition in which only twelve grey placeholders were presented was the easiest target absent condition. The rationale behind these manipulations is to obtain different brain activations strictly related to the simple visual search (and not to the

target attentional capture). In this way, different conditions can be used as the reference of the bilateral activation elicited by midline targets, that is the main aim of the current experiment.

## 4.2.2 Methods

### Subjects

Nineteen students at Université de Montréal (11 females) participated in the experiments after giving informed consent. Their mean age was 24.4 years (SD = 3.6) and all subjects had normal or corrected-to-normal visual acuity. Four subjects were removed from the final sample because of low overall accuracy (less than 50%, one subject) or a high percentage of EEG epochs discarded due to eyes blinks and EEG artifacts (more than 40%, three subjects).

### Stimuli and procedure

Equiluminant stimuli (9.2 cd/$m^2$) were presented on a 17-inch CRT monitor at a viewing distance of 57 cm on a black background. Twelve circles (.5° radius) were presented spaced around an imaginary circle (3.5° radius) centered on a fixation cross, and their color could change among the conditions. When presented, the target could appear only in one of four possible positions (randomly chosen among 12 or 6 o' clock, midline positions, and 3 or 9 o' clock, lateral positions). The other eight circles were grey (RGB = 140,140,140).

The target was defined at the beginning of the session as the color of a circle (blue, RGB = 0,110,255; green, RGB = 53,134,0; fuchsia, RGB = 245,0,110; violet, RGB = 195,59,239; dark orange, RGB = 206,104,0) and the orientation of a bar (a slash or backslash as in the Experiment 1) presented inside of it. When the target color was

absent, the stimuli at the four positions (i.e., 12, 3, 6, 9 o'clock) could be in four different non-target colors (i.e., heterogeneous condition), or all in the same non-target color (i.e., homogeneous condition), or all grey (i.e., all-distractors condition). Another possible case consisted in the presence of a circle in the target color, but the bar inside was not oriented as a target. Every trial started with a fixation cross presented in the center of the screen and remained visible during the whole trial, which was followed by six displays (each lasting 150 ms) and separated from each other by 900 to 1100 ms (randomly jittered interval in which only the fixation cross was visible). Each of these six displays was a search array, which is also called "frame" in the context of the Multiple Frame Procedure (MFP, see Aubin & Jolicœur, 2016; Drisdelle et al., 2017; Pomerleau, Fortier-Gauthier, Corriveau, Dell'Acqua, & Jolicœur, 2014). Overall, 238 trials were presented (1428 frames): 204 frames for each distractor condition, as well for the homogeneous and heterogeneous ones. When the target circle was presented, it was in one of the four possible positions 204 times each, and in half of them the bar inside had the target orientation. The task was to count the total number of targets presented in the six frames of each trial and to report this number at the end of the trial, without speed pressure. To do this, they used the left and right arrow keys of a keyboard placed in front of them, respectively decreasing and increasing a number from 0 to 6, representing their response, which was confirmed and entered by pressing the down arrow key. After the response, accuracy feedback was provided by presenting two digits separated by a slash: the digit on left was the subjects' response, and the one on right was the correct response.

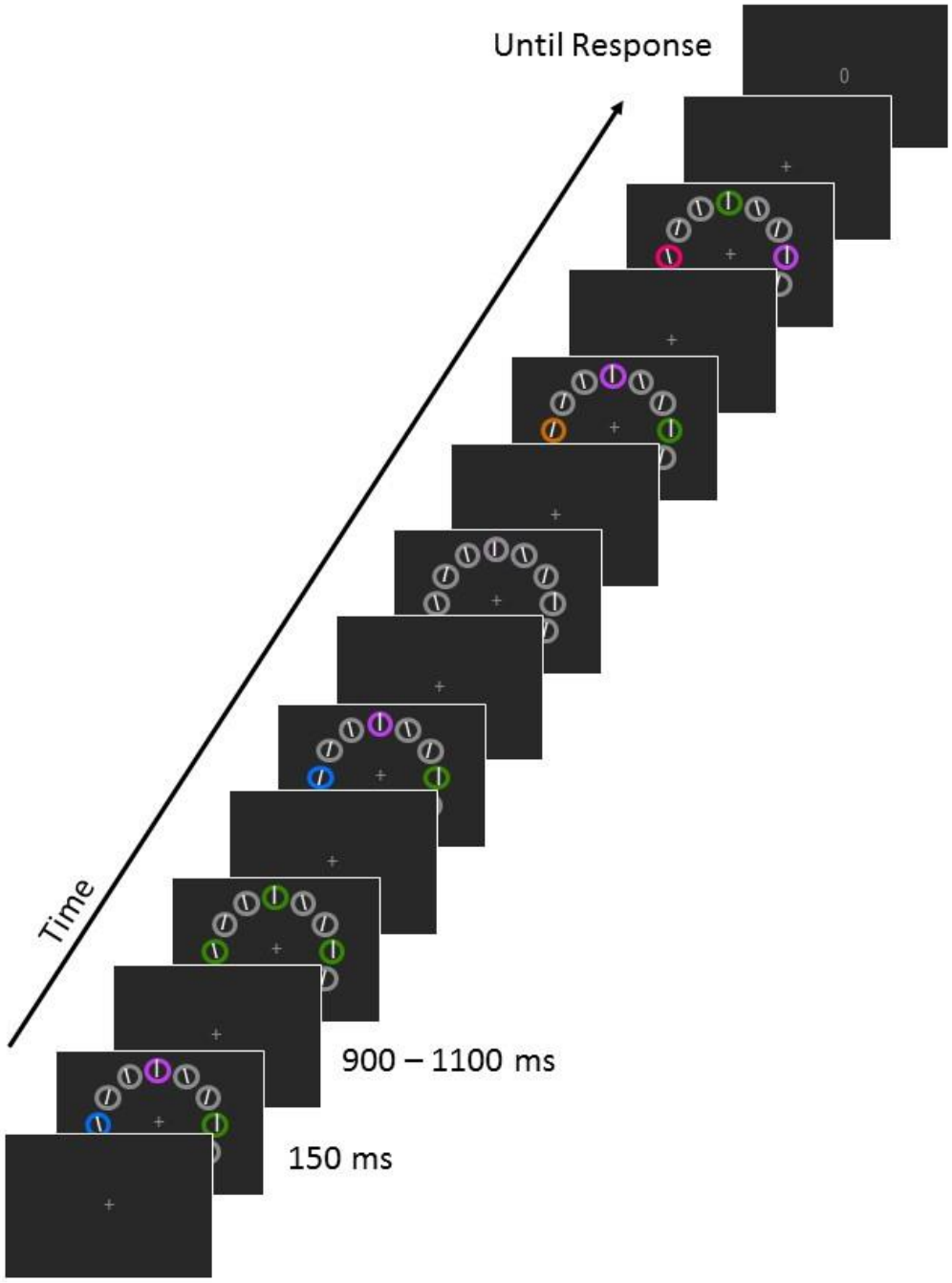


**Figure 4.3.** Display sequence (6 frames) in one trial of Experiment 3b. After a time interval random jittered between 900 and 1100 ms, the trial started. Each visual search array lasted for 150 ms, and each of them were separated by another 900 – 1100 ms jittered time interval.

## 4.2.3 EEG/ERP Recordings and Analysis

EEG was recorded using a BioSemi Active Two system and an elastic cap with 64 Ag/Ag–Cl electrodes positioned according to the International 10/10 system (Fp1, Fpz, Fp2, AF7, AF3, AFz, AF4, AF8, F7, F5, F4, F1, Fz, F2, F4, F6, F8, FT7, FC5, FC3, FC1, FCz, FC2, FC4, FC6, FT8, T7, C5, C3, C1, Cz, C2, C4, C6, T8, TP7, CP5, CP3, CP1, CPz, CP2, CP4, CP6, TP8, P9, P7, P5, P3, P1, Pz, P2, P4, P6, P8, P10, PO7, PO3, POz, PO4, PO8, O1, Oz, O2, Iz). The sampling rate was 512 Hz and the signal was referenced to the average of left and right mastoids after the recording. All the other preprocessing and analysis procedures are the same used for the Experiment 3a. The ERP activity of the three additional conditions (heterogeneous, homogeneous, distractors) were also the average of P7 and P8 electrodes. This choice was due to the lack of lateral shift of attention in these cases. Additionally, due to an unexpected result in the N1 time range, the mean amplitude of this component was estimated in a 135-175 ms time window.

## 4.2.4 Results

Behavioral

Differently from Experiment 3a, since the use of an MFP paradigm, it was not possible to isolate the accuracy or the reaction time of every single condition. Nevertheless, the overall mean proportion of correct responses was .91, comparable with the mean accuracy of the previous experiment.

ERP

N2 component

An analysis of the averaged activity of P7 and P8 electrodes was conducted in line with the Experiment 1, when possible. Namely, since the design was not completely balanced, an ANOVA 2 x 2 x 2 (side x target color presence x target bar presence) has been conducted only for a part of data, which excluded the distractors, homogeneous, and heterogeneous conditions. No effect related to the bar orientation (target vs. non-target) was found (F = .19, $p$ = .67). For this reason, measurements related to the bar orientation was collapsed for each of the four possible target positions for further analysis and graphs. A main effect of the target position was found ($F$(2,28) = 8.66, $\eta_p^2$ = .382, $p$ = .001. Separated pairwise FDR (Benjamini & Hochberg, 1995) corrected $t$ tests replicate the results of Experiment 3a, namely, there was no significant difference between Contralateral (–.93 μV) and Midline (–1.02 μV) conditions for mean amplitudes across target positions ($t$(15) = –.29, $p$ = .95); however the differences between Contralateral (–.93 μV) and Ipsilateral (–.02 μV) ($t$(14) = –3.44, $p$ = .001) and between Midline (–1.02 μV) and Ipsilateral (–.02 μV) $t$(14) = –3.74, $p$ = .001, conditions were significant.

In order to test differences among the other three conditions (Heterogeneous, Homogeneous, Distractors) a second one-way ANOVA revealed a difference between the amplitudes in the N2 time range, $F$(2,28) = 6.15, $\eta_p^2$ = .305, $p$ = .006. Pairwise Tukey-corrected $t$-tests showed that a significant difference was present between Distractors (.36 μV) and Heterogeneous (–.43 μV) condition: $t$(14) = 3.42, $p$ = .002, and between Distractors (.36 μV) and Homogeneous (–.19 μV) condition, $t$(14) = 2.37, $p$ = .04. No significant difference was found between the Homogeneous (–.19 μV) and Heterogeneous (–.43 μV) conditions ($p$ = .54).

Finally, a series of *t*-tests assessed all the other possible differences that were not investigated before because of the lack of orthogonality, only the significant comparisons are reported in detail. Mean amplitude in the N2 time window in the Heterogeneous condition was significantly different only from the Midline target position ($t(14) = 2.36$, $p = .03$). The Homogeneous condition was significantly different from the Midline target position ($t(14) = 2.42$, $p = .03$). Finally, the Distractors condition differed from both the Midline target position ($t(14) = 3.73$, $p = .002$) and the Contralateral target position ($t(14) = 3.12$, $p = .007$).

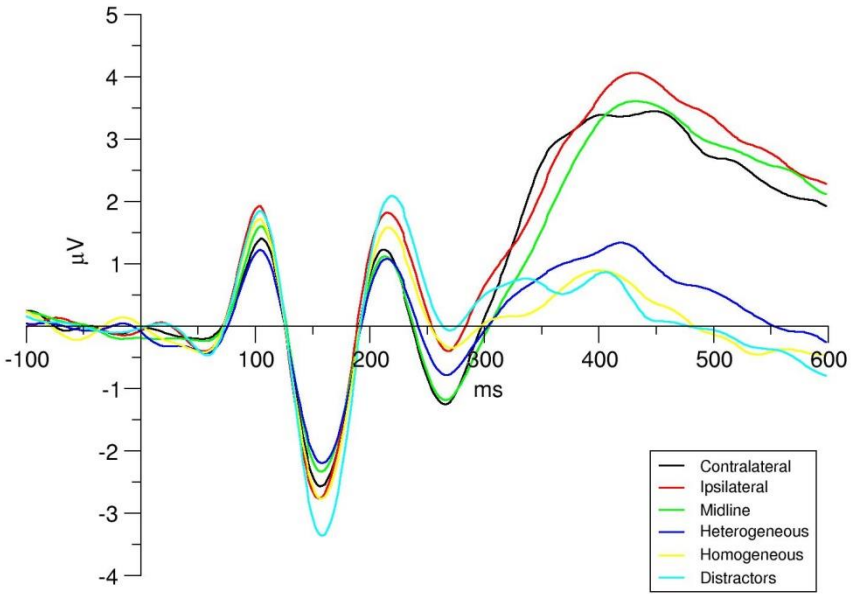


**Figure 4.4.** Grand average waveforms for the principal conditions from Experiment 2 at P7/P8 electrode sites.

N1 component

As can be seen in Figure 4.4, the grand average waveforms suggested possible differences among conditions within the N1 time range. These differences were analyzed using similar analyses to those performed for the N2 time range. An ANOVA

that considered all the conditions but the Distractors, Homogeneous, and Heterogeneous conditions showed that there were no differences in the averaged P7/P8 amplitudes related to the target position ($p$ = .3) nor the presence of the target ($p$ = .49). Nevertheless, a second ANOVA revealed a difference between the Heterogeneous, Homogeneous, and Distractor conditions, $F(2,28) = 15.15$, $\eta_p^2 = .520$, $p < .001$. FDR (Benjamini & Hochberg, 1995) corrected $t$ tests showed that a significant difference was present between Distractor (–3.69 µV) and Heterogeneous (–2.58 µV) conditions: $t(14) = –5.48$, $p < .001$, and between Homogeneous (–3.21 µV) and Heterogeneous (–2.58 µV) conditions: $t(14) = –3.16$, $p = .004$. A marginally significant difference was found between Homogeneous (–3.21 µV) and Distractor (–3.69 µV) conditions: $t(14) = 2.32$, $p = .053$.

Relationship between N1 and N2 modulations

The results in the N1 time range found in the distractors and heterogeneous conditions could reflect differential effects of discrimination processes (Vogel & Luck, 2000) associated with these conditions. However, this differences within this time range was not expected.

Since the aim of this study was to search for condition-related differences in the N2 time range, the possibility that the effects in the N1 time range carry forward to the N2 time range need to be ruled out. To do this, N1 and N2 activities elicited by the conditions which show an unexpected pattern of modulation were (namely, homogeneous, heterogeneous, and all-distractor conditions). The presence of a correlation between the amplitudes in the two time ranges would be a marker of a N2 biased by the N1 processes. Differently, in none of these three conditions a significant and/or reliable correlation was found (homogeneous: $r = .21$, $p = .44$; heterogeneous: $r$

= .22, $p$ = .42; all-distractors: $r$ = .18, $p$ = .52). This suggests that, in this task, there is no functional relation between the N1 and N2 time-range cognitive mechanisms. Reasonably, it seems that the presence or the absence of a target color (contralateral, ipsilateral, midline conditions) compared to the conditions in which the target color is absent (distractors, heterogeneous, homogeneous conditions) elicit a different discrimination process in the N1 time range.

### 4.2.5 Discussion

This experiment was done both as a control of Experiment 3a and to extend its result. In general, the aim of this chapter was to compare the activity elicited by a lateral target to a target presented on the midline. Experiment 3a showed that the contralateral portion of the N2pc is equal to the bilateral N2 elicited by a target presented on the midline. Conversely, the ipsilateral portion of the N2pc is positively shifted. These results are confirmed in the present experiment, although the differences on the experimental design between the two experiments. Namely, in Experiment 3a subjects were asked to search for a target circle (defined by a color), and then to discriminate whether the bar inside the target circle was vertical or tilted. In the current experiment, subject had to detect the presence of a specific circle (still defined by color as in Experiment 3a) and then the presence of a specific bar inside (i.e., oriented in a specific way). Additional conditions showed that, although the absence of a target, a bilateral negative shift still occur when the participant search within the visual array. Interestingly, the harder the search, the greater the negativity. As mentioned in previous sections, 200 ms after the presentation of a target occipital areas increase their firing rate and the activation of high-level structures allow the conscious access to the visual representation (Fahrenfort et al., 2007; Lamme & Roelfsema, 2000). The results of the

current experiment strongly suggest that the visual areas increase their activity also when no target is presented, but relative to the simple search mechanisms. Of interest, the lack of significant difference between homogeneous and heterogeneous condition, which suggests that the parieto-occipital attentional processes enrolled while searching are similar regardless the difficulty of the distractor color(s) discrimination. Compared to the distractors condition, heterogeneous and homogeneous ones are significantly different. This indicates that the complete absence of salient information (i.e., color(s)), requires less amount cognitive resources. Of note, there is no significant differences between distractors condition and the ipsilateral portion of the N2pc elicited by a lateral target. Since a bilateral positive shift was not expectable in distractors condition, the N2 waveform pattern of the component ipsilateral to a lateral target cannot be interpreted as a positive shift, as proposed by (Hickey et al., 2009).

In general, the findings of this chapter strongly suggest that visual search elicits a negativity in the N2 time range, regardless the presence or the absence of a target. Moreover, the reason for which humans are better in foveal compared to parafoveal visual attention could be related to the bilateral posterior brain activity involved in the first compared to the mostly contralateral activity engage in the second. Overall, this evidence raise a more general question regarding what N2pc reflects, since the evidence here presented seems to be in contrast with a view which postulate two separate mechanisms for the contralateral and ipsilateral portion of the N2pc, as well two separate mechanisms for foveal and parafoveal attention.

## 4.3. Conclusion: Chapter 4

The two experiments presented in this Chapter showed that the contralateral activity elicited by a lateral target is exactly the same of the bilateral activity elicited by a target presented on the midline. This suggests that a lateral target is mostly processed by the contralateral parieto-occipital brain hemisphere. Still, when a target was presented laterally, the ipsilateral activity was more positive compared to the contralateral, as already showed in literature (e.g., Luck & Hillyard, 1994a). In Experiment 3b three additional conditions serves as a baseline for the bilateral activity elicited by a midline presented target. First, results of Experiment 3b confirm the results of Experiment 3a. Moreover, regardless the presence or the absence of a target, the simple presentation of a visual search array elicits an N2 ERP response. Of interest, a fine-grained modulation of target-absent conditions elicited a bilateral N2 component which is more negative in relation to the difficulty of the search. Nevertheless, because of the high degree of similarity between the target present conditions, the best control condition for the midline presented target was the heterogeneous condition, where four distractors were printed in four different non-target colors. The activity elicited in this condition was significantly different (more positive) from the activity of the midline condition (which was actually the condition for which a baseline was necessary), suggesting that a target presented on the midline exert a bilateral negative shift. Moreover, the absence of significant difference between the ipsilateral activity elicited by a lateral target and the distractor (control) condition can suggest that the ipsilateral portion of the N2pc could reflect a simple absence (or partial absence) of search activity in the hemifield opposite to the target presentation, which lead to an unbalancing between the two posterior hemispheres.

# Chapter 5 – General Discussion

The aim of this thesis was to explore which mechanisms are in common between the deployment of attention in time and space.

This has been investigated by using the EEG in two ways: first (Chapter 2 and 3), the temporal attentional dynamics of the AB (Raymond et al., 1992) was study for both foveal and parafoveal presented information; second (Chapter 4), dynamics related to the deployment of attention in space were studied through a visual search task.

More specifically, a first investigation (Chapter 2) regarded the AB phenomenon, an impairment in reporting the second of two targets embedded among distractors when it appeared in close temporal succession by the first target. As suggested by previous researches, AB effect interest mainly a fronto-parietal brain network (Choi et al., 2012; Kranczioch et al., 2005; Marcantoni et al., 2003; Marois et al., 2004; Marois & Ivanoff, 2005; Slagter et al., 2012). At the electrophysiological level, the P3b –an ERP usually localized at midparietal scalp sites– has been shown to be the marker of consolidation of information in short-term memory (Akyürek et al., 2010). Additionally to this component, the P3a –a midfrontal component peaking before the P3b– could be considered as a marker of detection of salient information (e.g., Barceló, Escera, Corral, & Periáñez, 2006; Cycowicz & Friedman, 1998; Koechlin, Ody, & Kouneiher, 2003; Polich, 2007). Most of literature regarding the AB showed that consolidation of targets in this paradigm is influenced, although less is known about their detection.

Moreover, AB decreases in long-range phase synchronization in the beta and gamma band, encompassing the frontoparietal attention network have been reported

by Gross et al. (2004; see also Kranczioch, Debener, Maye, and Engel, 2007, Nakatani, Ito, Nikolaev, Gong, and Leeuwen, 2005). In line with this evidence, ERP presented in Experiment 1a showed a frontal (P3a) and a posterior (P3b) activations locked to the last target of RSVP streams. More precisely, the detection mechanism (P3a) presented a decreased amplitude at short relative to long lags, and a slight offset latency postponement. Similarly, the consolidation mechanism (P3b) still present a decrease amplitude at short relative to long lags, and also an onset latency postponement. Of interest, P3a modulations (amplitude and offset latency) are good predictors of the P3b onset latency: this evidence highlights the relevance of the fronto-parietal brain network, showing that the harder the AB task, the more decreased in amplitude and offset-postponed the P3a, and the more postponed the onset of the P3b. These data further the knowledge regarding the deployment of attention in time, by showing that there is a link between the reduction of the detection of information reflected by the P3a and the postponement of the time necessary to start the consolidation, as previously hypothesized by Nieuwenhuis and colleagues (2005).

Once isolated two attentional markers of the AB (Chapter 2), in Chapter 3 a combination of the AB task of Chapter 2 and an AB task with lateral targets (e.g., Dell'Acqua et al., 2006) was employed. In this case, the last target of the RSVP could only appear in a lateral position. As for Experiment 1a (Chapter 2), P3a and P3b components showed AB-related modulations in amplitude and latency, suggesting that the attentional perturbation caused by the AB exert its effect regardless the spatial position of the target, confirming what already suggested by Jolicoeur et al. (2006). Moreover, differently from the paradigm of Chapter 2, in this case the last target also elicited –as expected– an N2pc, that is a negativity over the posterior scalp sites which is greater over the scalp hemisphere contralateral to the target compared to the

ipsilateral. Contrary to what expected, no AB-related modulations were found on the N2pc. Nevertheless, by analyzing the bilateral (i.e., N2pb, average of contralateral and ipsilateral activity) waveforms in the N2 time range, AB-related amplitude modulations were found. This finding indicates that the AB exert an influence not only in general mechanism of attention (i.e., fronto-parietal network). Indeed, also in the specific visual spatial domain, N2 modulations strongly suggest that –bilaterally– the parieto-occipital areas of the brain are prone to the AB effect. At the methodological level, this means that, at least in the AB case, to consider just the voltage difference between hemisphere (i.e., the N2pc) is not necessarily the best way to investigate the deployment of visual spatial attention.

Because of the evidence presented in Chapter 3 regarding the deployment of visual spatial attention and its relationship with the N2 ERP component, two visual search experiments were conducted. By comparing N2 time range activity elicited by a target presented on the midline and on lateral portion of the visual field, it has been possible to show that dynamics underlying foveal and parafoveal attention are more similar than what thought until now. Specifically, the N2 activation contralateral to a lateral target is exactly the same of the bilateral activation elicited by a midline target. Moreover, the presence of trials with no target presented (Experiment 3b) highlighted a fine-grained bilateral N2 component modulation related to the difficulty of the visual search task.

Collectively, all this evidence emphasizes a clear functional overlap of the mechanisms which drive human attention in the visual space. This overlap is highlighted both indirectly –through an investigation of the AB dynamics in foveal and parafoveal attention– and directly –through a comparison of foveal and parafoveal marker of attention–. To my knowledge, only a theoretical model attempted to provide

an opinion regarding the interplay of attention in visual search and attentional blink (Raffone, Srinivasan, & van Leeuwen, 2014; but see Wyble, Bowman, & Nieuwenstein, 2015), although what suggested by the authors did not help in making any prediction at the brain or behavioral level.

A different way to further test the evidence reported in Chapter 4, regarding the comparison between activity elicited by midline and activity elicited by lateral targets, would be to employ the paradigm proposed by Eimer and Grubert (2014). In that study, subjects were presented with two consecutive visual search arrays each with two stimuli (one of the left and one of the right of the fixation). The target was defined by its color, and the task was to report whether the targets of the two visual arrays belonged to the same category (both digits, or both letters) at the end of the trial. When the target of the second presentation was on the opposite side compared with the target of the first presentation, the authors found two identical N2pc components both for the first and the second target with opposite polarities, suggesting that it is possible to quickly shift attention to new visual objects. In a successive experiment (Grubert, Fahrenfort, Olivers, & Eimer, 2017) with a similar design, task-relevant item was still identified by color. Subjects' task was to report the identities of the task-relevant items, when they are letters, at the end of the trial. When the task-relevant item in the second visual array was a digit, no N2pc was elicited. This result shows that the second of two consecutive visual search arrays does not automatically capture attention (differently from what was previously found with the presentation of a single visual search array, Kiss et al., 2008). Based on this evidence, a future study could verify the results of Experiment 3b (Chapter 4). Indeed, Experiment 3b employed target absent conditions in which I assumed that no shift of attention was expected, and so –in my view– those would be proper cases to compare the activity elicited by a target presented on the

midline. In this future proposal, by presenting three items (at left, right, and midline positions) it will be possible to observe activity elicited by a lateral or midline unattended target (i.e., when it appears in the second visual search array and contains a digit, which is not a task-relevant item). An example of the design is represented in figure 5.1.

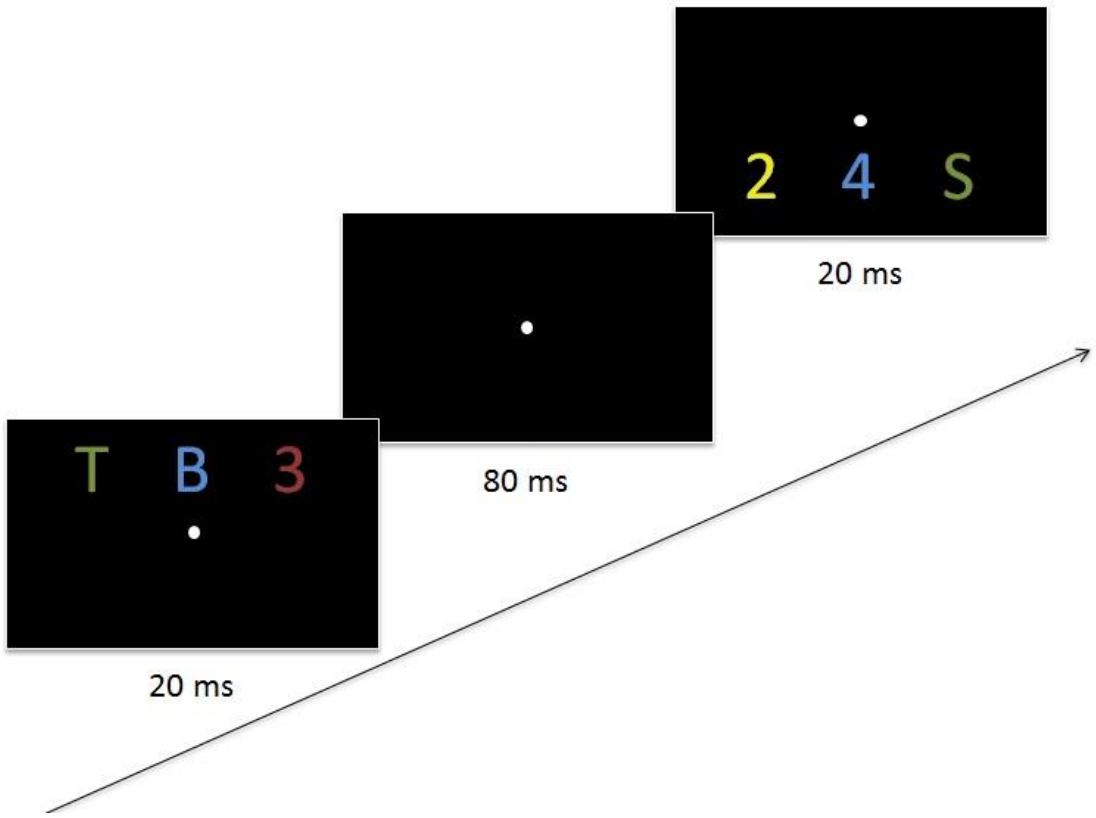


**Figure 5.1.** Example of a trial of a future experiment. Subjects are asked to report the identity of blue letters. When the blue item is not a letter (like in the second array), no target must be detected. The activity locked to the second array will show activity associated with an unattended target presented at the midline, which will be the control condition for the midline attended condition presented in the first array.

As for Chapter 4 experiments, a midline task-relevant target should elicit bilateral activity similar to the contralateral activity elicited by a lateral target. In task-irrelevant midline target conditions, the bilateral activity should be more similar to the ipsilateral activity elicited by lateral target conditions.

All the evidence presented in this thesis raises other issues. First, why a lateral target elicits an electrical unbalancing between the contralateral and ipsilateral scalp hemisphere? If foveal and parafoveal attention share the same attentional mechanism, and since the processes reflected by the N2 ERP component are attention-related (and not perceptual), the N2pc should be a simple accidental consequence of an experimental design with lateral information. This perspective needs to be tested in future.

Moreover, it would be necessary to investigate if other attentional dynamics (and not only temporal dynamics investigated by the AB) exist both in foveal and parafoveal attention. In other terms, by using a paradigm which can be employed both with midline and lateral presented information, and finding different modulations of behavioral or electrophysiological data related to the experimental manipulations, the debate for which foveal and parafoveal attention shares the same attentional mechanisms would remain open.

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# Acknowledgements

First, I would like to thank my advisor, Prof. Roberto Dell'Acqua, for his support and competence along these three years. I am also grateful to my Canadian advisor Prof. Pierre Jolicœur, which actually supervised my research also when I was in Italy through a long-distance supervision.

I want to express my thankfulness also to other people (professors, post docs, colleagues) who took care of my studies in many different ways: Paola Sessa, Simone Cutini, Federica Meconi, Sabrina Brigadoi, Arianna Schiano, Marco Lunghi, Brandi Lee Drisdelle, Christine Lefebvre.