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Stimulus-to-matching-stimulus interval influences N1, P2, and P3b in an equiprobable Go/NoGo task

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Keywords
task, p2, n1, nogo, stimulus, go, matching, p3b, interval, influences, equiprobable

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Abstract

Previous research has shown that as the stimulus-to-matching-stimulus interval (including the target-to-target interval, TTI, and nontarget-to-nontarget interval, NNI) increases, the amplitude of the P300 ERP component increases systematically. Here, we extended previous P300 research and explored TTI and NNI effects on the various ERP components elicited in an auditory equiprobable Go/NoGo task. We also examined whether a similar mechanism was underpinning interval effects in early ERP components (e.g., N1). Thirty participants completed a specially-designed variable-ISI equiprobable task whilst their EEG activity was recorded. Component amplitudes were extracted using temporal PCA with unrestricted Varimax rotation. As expected, N1, P2, and P3b amplitudes increased as TTI and NNI increased, however, Processing Negativity (PN) and Slow Wave (SW) did not show the same systematic change with interval increments. To determine the origin of interval effects in sequential processing, a multiple regression analysis was conducted on each ERP component including stimulus type, interval, and all preceding components as predictors. These analyses showed that matching-stimulus interval predicted N1, P3b, and weakly predicted P2, but not PN or SW; SW was determined by P3b only. These results suggest that N1, P3b, and to some extent, P2, are affected by a similar temporal mechanism. However, the dissimilar pattern of results obtained for sequential ERP components indicates that matching-stimulus intervals are not affecting all aspects of stimulus processing. This argues against a global mechanism, such as a pathway-specific refractory effect, and suggests that stimulus processing is occurring in parallel pathways, some of which are not affected by temporal manipulations of matching-stimulus interval.

Keywords: Auditory event-related potentials (ERPs); Target-to-target interval (TTI); Nontarget-to-nontarget interval (NNI); Sequence effects; Interstimulus interval (ISI); Probability; P3(00); P3b; Equiprobable; Go/NoGo; N1; P2; P3b; Slow Wave (SW).

1. Introduction

The stimulus-to-matching-stimulus interval is the time between presentations of a particular stimulus, such as the target-to-target interval (TTI) and nontarget-to-nontarget interval (NNI). Increases in TTI have been shown to systematically enhance P300 amplitude (Croft et al., 2003; Gonsalvez et al., 1999, 2007; Gonsalvez & Polich, 2002; Steiner et al., 2013a), a component of the event-related potential (ERP) thought to be related to "endogenous" aspects of information processing (Donchin et al., 1984), such as decision-making (Johnson & Donchin, 1982; Nieuwenhuis et al., 2005; Verleger, 1997). Similar patterns in nontarget P300 have been reported for increases in NNI (Steiner et al., 2014), but these appear to be contingent on the paradigm used (Steiner et al., 2013b).

Work on the TTI/P300 relationship was motivated by early oddball studies exploring global probability (Duncan-Johnson & Donchin, 1977; Polich & Bondurant, 1997; Polich et al., 1991), stimulus sequence (Hermanutz et al., 1981; Johnson & Donchin, 1982; Leuthold & Sommer, 1993; Sams et al., 1983, 1984; Squires et al., 1976, 1977; Starr et al., 1997; Verleger, 1987), interstimulus interval (ISI; Fitzgerald & Picton, 1981; Miltner et al., 1991; Polich 1990a, b), and temporal probability (Fitzgerald & Picton, 1981). Gonsalvez et al. (1999) argued that all the P300 results from those early studies may have been attributable to changes in TTI, as manipulations of global probability, sequence, and ISI unavoidably alter the temporal probability of the target, and consequently the TTI. A similar argument can be made for changes in nontarget ERP component amplitudes with inadvertent manipulations of NNI.

To date, TTI effects have been explored *directly* for only N1 and the P300 (Gonsalvez et al., 1999, 2007), while studies specifically analysing NNI have considered only P300 (Steiner et al., 2013b, 2014). Data from a recent study in this journal, Steiner et al. (2014), suggested possible TTI/NNI effects in earlier ERP components (N1, P2, N2), however it was

beyond the scope of that study to analyse those effects. Hence, the purpose of the current paper is to explore both TTI and NNI effects in a wider range of ERP components in order to clarify the origin of these effects in sequential processing.

The effect of ISI on the N1 is well documented (Budd et al., 1998; Čeponienė et al., 1998; Coch et al., 2005; Miltner et al., 1991; Polich, 1990b; Polich & Bondurant, 1997; Teder et al., 1993; Woods & Courchesne, 1986; Woods et al., 1980), with studies consistently observing a systematic augmentation in N1 negativity as ISI increases. This pattern of results has been taken as evidence for a relatively long refractory period or recovery cycle of N1 neural generators (Budd et al., 1998; Callaway, 1973; Ritter et al., 1968), which may last up to 10 s, or even 1-2 minutes (Näätänen, 1988). However, Näätänen and Picton (1987) detail several overlapping N1 components that can become selectively adaptive, responding differentially to stimulus repetition. That is, some neurons with wide receptive fields become refractory, where others with greater specificity continue to respond. Thus, any study examining N1 amplitude changes related to temporal factors needs to consider these multiple generators and utilise appropriate measures to separate overlapping components (e.g., principal components analysis; PCA).

These reliable N1-ISI effects contrast with the inconsistent pattern of results reported by studies exploring the effect of stimulus sequence on N1. For example, some studies have found enhancements in N1 amplitude with increases in preceding stimulus sequence length (Hermanutz et al., 1981; Starr et al., 1997; Verleger, 1987), while others have reported a decrease (Thomas et al., 2009), or no change (Kenemans et al., 1991; Polich & Bondurant, 1997). These discrepancies in results cannot be entirely attributed to task differences; for example, Hermanutz et al. (1981) and Polich and Bondurant (1997) reported contrasting patterns of results, but both studies derived ERPs similarly from randomly presented sequences.

Inconsistencies in the N1 response profile are also apparent in TTI studies. Gonsalvez et al. (2007) showed that N1 amplitude increased as TTI increased, however, that study used a single-stimulus task where TTI was confounded with ISI. Further, Gonsalvez et al. (1999) demonstrated that when ISI was controlled, TTI did not emerge as a determinant of N1 amplitude. Discrepancies may have arisen from paradigm differences, and thus a careful investigation of stimulus-to-matching-stimulus interval effects on N1 is warranted.

Evidence from temporal probability, sequence, and ISI studies suggests that P2 may be affected by matching-stimulus intervals similarly to P300. For instance, Fitzgerald and Picton (1981) showed that P2 amplitude increased as temporal probability decreased; Polich and Bondurant (1997), and Polich (1990b) reported longer P2 latencies to longer sequences; Polich (1990b), Miltner et al. (1991), and Woods and Courchesne (1986) found larger P2 amplitudes to longer ISIs in adults; whilst Coch et al. (2005) reported a similar pattern in children. These studies present a congruent pattern of results suggesting that P2 amplitude may increase as TTI/NNI increases.

The theoretical mechanism for a temporally-determined response-pattern as consistent and systematic as the TTI effect on the P300 requires special consideration. Arguably, the most pervasive P300 theory in the literature is the context-updating hypothesis (Donchin & Coles, 1988), where it is argued that the “endogenous” P300 indexes violated expectancies that arise when events differ from a contextual model of the environment. However, there are two issues arising from TTI/NNI data that are irreconcilable with this perspective. First, TTI studies consistently report that in oddball tasks, increases in TTI evoke P300s with larger amplitudes and *shorter* latencies. However, the context-updating hypothesis predicts that events violating expectancies should elicit larger P300s with *longer* latencies due to the additional time required to update the model of the context. Second, the context-updating hypothesis does not make explicit the role P300 plays in sequential processing, and how P300

may be an outcome from earlier "exogenous" processes (indexed by N1, P2 etc.; Donchin et al., 1978). That is, no predictions are possible for TTI/NNI effects in earlier ERP components.

Our previous papers examining TTI (e.g., Gonsalvez et al., 2007), and more recently stimulus-to-matching-stimulus interval effects on the P300 (Steiner et al., 2013b, 2014), have utilised the template-update model (Gonsalvez et al., 2007) as a theoretical framework. Briefly, this model states that TTI/NNI effects on the P300 reflect an immediate memory process involving the decay and update of stimulus templates. Implicit within this model is the assumption that P300 amplitude is unrelated to both unexpected (Donchin & Coles, 1988) and awaited (Verleger, 1988) events, and that the timing of events, rather than the specific "context" is important in determining P300. However, elaboration is required to make sense of similar possible TTI/NNI response profiles in earlier more "mechanistic" ERP components (N1, P2 etc.). Importantly, and as argued in Steiner et al. (2013a), it is the TTI/NNI response-profile that indexes memory-update, not solely ERP component amplitudes, which can reflect a wide array of stimulus characteristics including novelty, intensity etc.

The purpose of the current paper is to explore whether a similar mechanism underpins interval effects in the P300, and (possibly) early ERP components. Interval effects similar to those seen for P300 have been reported previously in N1 (Gonsalvez et al., 2007), perhaps suggesting a similar underlying mechanism (e.g., updating of a memory trace and/or a recovery cycle effect). The ISI/recovery-cycle effects reported for N1 (e.g., Budd et al., 1998) may indicate that a simple refractory process, traceable through sequential ERP components, might be the mechanism underpinning P300 TTI/NNI effects. If this were the case, it would suggest that P300 is not solely the outcome of complex cognitive operations (e.g., a strategic response; Donchin & Coles, 1998; Sommer et al., 1998; Verleger, 1998), but is somewhat "hardwired" to automatic/obligatory processes (Näätänen & Picton, 1987), such

as the recovery cycle of early sensory components. Alternatively, differential patterns of timing effects in sequential ERP components might suggest mechanism(s) other than a general stimulus-pathway refractory effect.

Importantly, it should be noted that a refractory effect is difficult to separate empirically from the updating of memory trace. For instance, Näätänen and Picton (1987) speculate that N1 component 1 may reflect the neural formation of a sensory memory trace of a stimulus, where "a 'neuronal model' of a stimulus may…be represented in the pattern of refractoriness prevailing in the generator mechanism" (p. 414). However, there is no understanding of the mechanism of TTI/NNI effects, and the origin of these effects in sequential processing should be examined. Thus, the current study pursued two aims. We aimed to clarify whether early "exogenous" ERP components, such as N1 and P2, are affected by the stimulus-to-matching-stimulus interval, and whether those effects could be sequentially traced over time to the P300.

Here, we used the same paradigm as Steiner et al. (2014), but tested a different group of subjects, extracted additional ERP components, and performed a wider range of analyses. In line with previous probability (Fitzgerald & Picton, 1981), sequence (Polich & Bondurant, 1997), ISI (Coch et al., 2005; Miltner et al., 1991; Polich, 1990b; Woods & Courchesne, 1986), and TTI/NNI studies (Croft et al., 2003; Gonsalvez et al., 1999, 2007; Gonsalvez & Polich, 2002; Steiner et al., 2013a, b, 2014), we expected the P300 and other ERP components, such as P2, to increase in amplitude as stimulus-to-matching-stimulus interval increased. At first glance, the extant literature does not suggest a consistent N1 response profile (e.g., Hermanutz et al., 1981 vs. Polich & Bondurant, 1997), but the majority of studies indicate that N1 may increase as TTI/NNI increases (Budd et al., 1998; Čeponienė et al.,1998; Coch et al., 2005; Gonsalvez et al., 1999; Hermanutz et al., 1981; Miltner et al., 1991; Polich, 1990b; Polich & Bondurant, 1997; Starr et al., 1997; Teder et al.,

1993;Verleger, 1987; Woods & Courchesne, 1986; Woods et al., 1980), thus allowing the prediction that N1 will increase as stimulus-to-matching-stimulus interval increases. Further, and in line with Steiner et al. (2014), we expected RT to increase then decrease. We also explored the similarity between interval effects in different ERP components, and the origin of these phenomena in sequential processing by using a regression approach. Here, we expected to find that stimulus-to-matching-stimulus interval would positively predict a variety of ERP component amplitudes.

2. Method

2.1 Participants

Thirty students from the University of Wollongong participated in return for course credit (mean age = 19.6, *SD* = 1.8 years; 14 females, 28 right-handed). Prior to commencing the experiment, participants provided informed consent, and were free to withdraw at any time without penalty. Individuals self-reporting neurological or psychiatric illnesses, and/or use of psychotropic medication, were excluded. Self-reports indicated that participants had refrained from psychoactive substances for at least 12 hours and from tea, coffee, alcohol, and cigarettes for at least 2 hours prior to testing. All participants had normal or corrected-to-normal vision and self-reported normal hearing.

2.2 Procedure

A demographic and screening questionnaire was completed by all participants before they were fitted with EEG recording apparatus. Prior to the experiment, participants completed an electrooculogram (EOG)/EEG calibration task (Croft & Barry, 2000). Participants were seated in an air-conditioned room 600-800 mm in front of a 48.3 cm (19”) Dell LCD monitor and instructed to fixate on a 10 × 10 mm grey cross centred on a black background. Acoustic stimuli were delivered binaurally through Sony MDR V700

circumaural stereo headphones, and consisted of 1000 and 1500 Hz 60 dB SPL tones, of 50 ms duration (15 ms rise/fall time).

The experimental task[1] was an uncued equiprobable Go/NoGo task, broken into four different blocks (approximately 4.5 min each), with short rest intervals between blocks to minimise fatigue. Equiprobable targets and nontargets (1000 and 1500 Hz tones, 132 of each across the 4 blocks; counterbalanced across subjects) were presented in a pseudo-random order (fixed across subjects; see Figure 1). Desired TTIs and NNIs were obtained by varying the stimulus onset asynchrony (SOA; no less than 1 s) to create intervals of 1, 2, 5, 10, 15 s, each with 13.2 % probability, and 3, 7, and 12 s with lower probability to be used as fillers (not analysed further). Within each block, matching-stimulus intervals were not significantly correlated with the preceding-stimulus interval (PSI), $r(20) = .310$, $p = .183$, two-tailed (this analysis did not include the first interval as this was necessarily correlated with PSI). To balance possible speed/accuracy trade-offs, participants were instructed to "respond to target stimuli with a button press, as quickly and as accurately as possible". Participants responded with their dominant hand on a Logitech® Precision game controller. Instruction was given to sit as still as possible, but participants were not directly instructed to refrain from blinking (Verleger, 1991). This procedure was approved by the joint South Eastern Sydney/Illawarra Area Health Service and University of Wollongong Health and Medical Human Research Ethics Committee.

Figure 1 about here…

2.3 Materials and Apparatus

EEG data were recorded continuously using a 70 Hz lowpass filter from A2 and 30 scalp sites (Fp1, Fp2, F7, F3, Fz, F4, F8, FT7, FC3, FCz, FC4, FT8, T7, C3, Cz, C4, T8, TP7,

[1] It should be noted that this is the same paradigm as Steiner et al. (2014), and was based on that used in Steiner et al. (2013b), but here we added a manipulation of NNI.

CP3, CPz, CP4, TP8, P7, P3, Pz, P4, P8, O1, Oz, O2) with an electrode cap using tin electrodes. A1 was used as a reference and the cap was grounded by an electrode located midway between Fp1, Fp2 and Fz. Data were acquired using a Neuroscan Synamps 2 digital signal-processing system and Neuroscan 4.3.1 Acquire software, and were recorded DC – 70 Hz. The display and stimulus markers were controlled by a linked stimulus computer using Neurobehavioral Systems Inc. Presentation V 13.0 Build 01.23.09 software.

EOG was recorded using tin cup electrodes placed 2 cm above and below the left eye for vertical movements, and on the outer canthus of each eye for horizontal movements. Impedance was less than 5 kΩ for cap, EOG, and reference electrodes. Scalp and EOG potentials were amplified with a gain of 500 and digitised at a rate of 1000 Hz.

2.4 Data Extraction

Trials containing omission (misses) or commission (false alarms) errors, or response times longer than 800 ms, were excluded. Participant error rates were < 1 % (M = 0.85 %, SD = .88). The EEG data were EOG corrected using the RAAA EOG Correction Program (Croft & Barry, 2000). Single trial ERPs were re-referenced to digitally linked ears and extracted offline using the Neuroscan Edit software, low pass filtered (0.1 – 30 Hz, zero-phase shift, 24 dB/Octave), epoched for -100 ms pre- to 500 ms post-stimulus, and baselined to the pre-stimulus period. Data were manually inspected for additional artefacts, and any contaminated trials were rejected; together with errors and RTs > 800 ms, an average of 1.22 % trials (SD = 1.05) were excluded across subjects. For each stimulus type, averages were computed for each subject for each of the five intervals of major interest.

2.5 Principal Components Analysis

The averaged data (-100 to 500 ms: 600 datapoints) from 30 scalp locations were submitted to a temporal PCA using Dien's ERP PCA toolkit (v. 2.23; Dien, 2010) in MATLAB (The Mathworks, R14SP3). Data for the PCA were half-sampled to 300 time-

points (variables) to reduce computation time. Factors for all conditions were quantified simultaneously (9000 observations: 30 participants × 2 stimulus types × 5 intervals × 30 sites). The PCA used the unstandardised covariance matrix with Kaiser normalisation, and all 300 unrestricted factors underwent Varimax rotation, following Kayser and Tenke (2003). PCA factors were identified as ERP components based on their latency, topography, and polarity of their conspicuous maximum loading, and those explaining > 3 % of the total variance were retained for analysis. The factor scores at the maximum peak of these components were output and entered into subsequent statistical analyses.

2.6 Statistical Analyses

To define the topography for each of the ERP components identified, separate MANOVAs were carried out on the microvolt-scaled factor scores (Dien, 2012) at the 9 inner sites involving the sagittal plane: *Frontal* (F3, Fz, F4), *central* (C3, Cz, C4), and *parietal* (P3, Pz, P4); and the coronal plane: *Left* (F3, C3, P3), *midline* (Fz, Cz, Pz), and *right* (F4, C4, P4). Planned contrasts compared regions sagittal (frontal vs. parietal, and central vs. mean of the frontal and parietal) and coronal planes (left vs. right, and midline vs. mean of the left and right sites). The topographic distribution of component amplitudes can be examined efficiently by utilising these orthogonal planned contrasts. No Bonferroni-type α adjustment was required as a priori contrasts were used, and the number of contrasts did not exceed the degrees of freedom for effect (Tabachnick & Fidell, 1989). Component amplitude was then defined as the mean amplitude across the maximal region (e.g., parietal maximum → mean across P3, Pz, and P4); using a mean across a region defined by multiple sites, rather than a single electrode, reduces the impact of chance variance at a single site.

Separate repeated-measures MANOVAs assessed each component's amplitude for the effects of *Interval* (5 levels: 1, 2, 5, 10, 15 s) and *Stimulus Type* (2 levels: Target vs. Nontarget). Within the interval factor, weighted linear and quadratic trends were assessed.

RT to target stimuli was assessed over the five interval levels with a one-way repeated-measures MANOVA, again with weighted linear and quadratic contrasts. The violations of sphericity assumptions associated with repeated-measures analyses do not affect single degree of freedom contrasts, so Greenhouse-Geisser-type correction was not necessary (O'Brien & Kaiser, 1985). All *F*-tests reported have (1, 29) degrees of freedom unless otherwise noted.

To examine the second aim of this study, regarding the origin of interval effects in sequential processing, the determinants of each ERP component were examined through a series of regressions. Separate stepwise multiple regressions with each component as the dependent variable were conducted; these included factors of interval, stimulus type, and all sequentially preceding ERP component amplitudes. One-way tests were utilised for all analysed predictions.

It should also be noted that, as this paper details results for a number of dependent measures, the frequency of Type I errors increases. However, Howell (1997) argues that this increase in frequency of Type I errors cannot be controlled by adjusting α-levels, because the probability of Type I error remains the same.

3. Results

3.1 Grand Means

Figure 2 (left column) illustrates the grand mean ERPs for targets and nontargets from midline sites. Grand mean ERPs for each of the five intervals of major interest from midline sites are displayed in Figure 3 (targets: left column; nontargets: right column).

Figures 2 and 3 about here…

3.2 PCA Output

Out of the 300 temporal factors extracted, the first eight explained 87.3 % of the total variance. The middle column of Figure 2 displays the sums of these eight temporal

components at the midline sites. Comparison between original data and PCA ERPs indicates a good fit (Figure 2, right column).

The temporal factor loadings (rescaled to μV by multiplying each time point by the standard deviation; Tabachnick & Fidell, 1989) for the eight ERP components are displayed as a function of time in Figure 4. The percentage of variance explained, latency, and factor order for each rotated component is also indicated. Topographic headmaps of the temporal components, averaged across stimulus type and interval, are displayed at the top. These components were tentatively identified in terms of their polarity, latency, temporal sequence, comparison with the raw ERPs, and topography as P1, N1, Processing Negativity (PN; temporally distributed negativity occurring relatively late in the N1 latency range, described in Näätänen & Picton, 1987), P2, N2, P3b (distinct parietal positivity), frontal-P3 (topographically resembling P3a, although surprisingly occurring *after* the P3b), and SW. Of these eight components, factors 6-8 (identified as P1, N2, frontal-P3; dashed lines, grey text Figure 4) did not explain a substantial amount of total variance (< 3% of the total each), and consequently were not considered for further analysis and will not be discussed further. The remaining 5 components (P2, N1, PN, P3b, SW) explained 80.8 % of the total variance.

Figure 4 about here…

The analyses of these five components are reported below in order of component latency. The topography for each component and the corresponding *F*- and *p*-values, and partial effect sizes (η_p^2) are detailed in Table 1; the largest effect size identified the component topography and this region (indicated in italics) was used for analysis. The direction of these effects is written as "<" and ">", and interactions between contrasts as "×". Trends examined as a function of interval are denoted as "linear intervals" or "quadratic intervals". Figures 5-9 illustrate component amplitudes over intervals separately for targets and nontargets, with standard error bars. The dashed line indicates the linear trend across

stimuli, and the line equation is indicated. Unfilled markers at the end of each series indicate mean target and nontarget amplitudes across intervals. Table 2 shows the relative change (%) in component amplitudes c.f. the 1 s matching-stimulus interval level.

Table 1 about here…

3.3 Factor 4: N1

As shown in Table 1 and the top panel of Figure 4, N1 demonstrated a central topography. There was a vertex enhancement, and N1 was marginally more negative in the left. N1 showed a systematic linear increase in negativity as interval increased (across both TTI and NNI; linear intervals: $F = 29.65$, $p < .001$, $\eta_p^2 = .51$; Figure 5); this plateaued from around 10 s (quadratic intervals: $F = 24.64$, $p < .001$, $\eta_p^2 = .46$), this can be seen as a relative change in Table 2. There was no main effect of stimulus type and no interval × stimulus type interaction.

Table 2 and Figure 5 about here…

3.4 Factor 5: PN

PN was maximally negative centrally (Table 1 and Figure 4). There were enhancements in frontal and temporal areas, and a central enhancement that was smallest in the midline. Figure 6 illustrates that there were no significant main effects or interactions involving interval or stimulus type.

Figure 6 about here…

3.5 Factor 3: P2

P2 showed greatest positivity centrally (Table 1, Figure 4). Amplitudes were enhanced in the midline, and this interacted with the central maximum to produce a vertex enhancement. Figure 7 illustrates that P2 amplitude increased over intervals in a linear fashion (linear intervals: $F = 5.99$, $p = .021$, $\eta_p^2 = .17$) at 0.09 μV/s (see Table 2 for

percentage increase in μV over intervals[2]). There was a main effect of stimulus type with greater positivity to nontargets (nontarget > target: $F = 7.08$, $p = .013$, $\eta_p^2 = .20$). There was no interval × stimulus interaction.

Figure 7 about here…

3.6 Factor 2: P3b

P3b was parietally maximal (Table 1 and Figure 4). P3b was also enhanced centrally, at the vertex, and was larger in the midline than in the hemispheres, especially parietally. Across stimuli, P3b increased linearly as interval increased (linear intervals: $F = 29.79$, $p < .001$, $\eta_p^2 = .51$; Figure 8, Table 2), and plateaued around 10 s (quadratic intervals: $F = 11.95$, $p = .002$, $\eta_p^2 = .29$). There was no main effect of stimulus type, or interval × stimulus type interaction.

Figure 8 about here…

3.7 Factor 1: SW

SW demonstrated the typical frontally-negative, parietally-positive topography, with this difference being relatively greater in the left than right, and smallest in the midline (Table 1, Figure 4). There was also a right-central enhancement. To reflect the typical bipolar topography, SW was defined as the relative difference between frontal and parietal sites (i.e., [mean P3, Pz, P4] minus [mean F3, Fz, F4]). The frontally-negative/parietally-positive difference was more positive to targets than nontargets (target > nontarget: $F = 65.72$, $p < .001$, $\eta_p^2 = .69$; Figure 9). There was no main effect or interaction involving interval.

Figure 9 about here…

3.8 RT

[2] It should be noted that as P2 has a near-zero baseline the relative change is grossly exaggerated.

Figure 10 shows that the response-profile of RTs followed a quadratic trend over TTI, with an initial increase followed by a later decrease at longer TTIs (quadratic intervals: F = 30.65, $p < .001$, $\eta_p^2 = .52$).

Figure 10 about here…

3.9 Regression

To test the origin of matching-stimulus interval effects in sequential processing, five separate stepwise multiple regressions were conducted with each ERP component as the dependent variable. Predictors were all preceding ERP components, stimulus type, and interval, but as no analysed ERP components sequentially preceded N1, only factors of stimulus type and interval were included in its analysis. When stimulus type was excluded from the model (i.e., it was nonsignificant), interval (β = -.196) explained 19 % of the variance in N1, $F(1, 298) = 11.54$, $p = .001$. PN and P2 were not significantly predicted by previous ERP components, interval, or stimulus type. But when we relaxed the entry criteria for P2 (α = .075), interval (β = .105) had a weak effect on P2 and explained 10.5 % of its variance, $F(1, 298) = 3.35$, $p = .068$. For P3b, when interval (β = .154) remained as a predictor and the stepwise regression excluded all the other factors (N1, PN, P2, stimulus type), the model reached statistical significance $F(1, 298) = 4.45$, $p = .036$. P3b (β = -.232) accounted for 26 % of the variance in SW, and was the only factor not to be eliminated from the model, $F(1, 298) = 22.40$, $p < .001$.

4. Discussion

This study was carried out to explore two aims. The first was to extend the findings of Steiner et al. (2014) and ascertain whether matching-stimulus interval effects were present in a range of ERP components. The second of these was to highlight whether a single mechanism was responsible for interval effects in other ERP components elicited in this task. Concerning the first aim, we showed that N1, P2, and P3b increased in amplitude as

matching-stimulus interval increased. PN and SW showed no effect of interval, but SW was more positive to targets. RT showed an initial increase followed by a decrease at longer TTIs. In relation to the second aim, interval predicted N1, P3b, and weakly predicted P2, amplitudes, but not PN or SW. SW amplitude was predicted by P3b only. These data suggest a similar (or the same) temporal mechanism is affecting N1, P3b, and to some extent, P2. But the lack of a consistent response-pattern for sequential ERP components (PN) indicates that stimulus-to-matching stimulus intervals are not affecting all stages of the processing sequence, arguing against a single definitive mechanism that operates throughout the processing stream.

The unrestricted temporal PCA facilitated the identification and subsequent analysis of 5 ERP components including N1, PN, P2, P3b, and SW. The vertex N1 identified here is topographically consistent with Näätänen and Picton's (1987) 'nonspecific' component 3, which has a diffuse generator source including motor areas, reticular formation, thalamus, and superior, middle, and inferior frontal gyri. However, Näätänen (1988) notes that the recovery time for this component "is very long, perhaps 1-2 min, and after a discrete stimulus, this component is deeply refractory" (p. 128). Here, N1 amplitude increased up to the 10 s interval (170 % for targets, 130 % for nontargets), before plateauing at the longest TTI/NNI. This pattern of results is more consistent with Näätänen and Picton's (1987) dominant component 1, thought to be generated in the supratemporal plane of the primary auditory cortex (Vaughan & Ritter, 1970), which "appears to recover fully in about 10 s" (Näätänen, 1988, p. 128). It therefore seems likely that the temporal PCA approach employed here has captured a subset of variance shared by multiple N1 components elicited in a similar time-frame (i.e., N1 components 1 and 3 both occur around 100 ms poststimulus). This is consistent with Näätänen's (1988) assertion that an auditory stimulus presented after a

long period of silence elicits a large N1 peak comprised of both nonspecific and supratemporal components.

The increase in N1 over intervals corroborates previous TTI (Gonsalvez et al., 2007), ISI (Budd et al., 1998), and some sequence studies (Hermanutz et al., 1981), and extends this pattern of results to NNI research[3]. Discrepancies with other studies may result from paradigm differences. For instance, Thomas et al. (2009) reported a decrease in N1 amplitude to increases in sequence length, however, that was a *visual* inhibitory-style Go/NoGo task, with response-requirements and N1 generators different to *auditory* oddball/equiprobable tasks. There was no mean difference in N1 amplitude between stimulus types, a finding that differs from some equiprobable (Barry & De Blasio, 2013) and oddball tasks (Fitzgerald & Picton, 1981). However, Näätänen and Picton (1987) highlight that when the timing of stimuli are made unpredictable (eliminating the possibility of selective prior preparation), attention-related changes are not present in the N1, particularly at longer ISIs, which may explain the current pattern of results.

It should also be noted that the increase over intervals (i.e., both TTI and NNI) reported for the PCA N1 component does not seem to be entirely consistent with the raw ERP waveforms. That is, Figure 3 suggests that N1 is influenced by TTI, but not NNI. This discrepancy may be due to the temporal PCA analysis, which distinguishes ERP components by their common temporal variance. As partly outlined above, it is possible that our PCA-N1 reflects a subset of variance shared by all N1 components, and this has contributed to the main effect of interval reported here. Future research could apply a two-step temporal-spatial PCA to try and separate the sources contributing to this complex.

[3] It should also be noted that these temporal effects in N1 are attributed to matching-stimulus interval and not to PSI. For P300, Polich has reliably demonstrated in multiple experiments (e.g., Polich, 1990b; Polich & Bondurant, 1997) that TTI effects are not due to PSI. That is, by comparing P300 derived from one and two-stimulus tasks using a range of ISIs, TTI effects remain unchanged when PSI is varied widely. To the best of our knowledge, the current study is the first to demonstrate similar temporal effects in N1 from matching-stimulus intervals.

The PN was centrally negative with temporal enhancements, a topography consistent with the processing negativity first identified in Näätänen et al. (1978). PN did not show any statistically significant effect of interval, which is congruent with the conceptualisation that PN is an effortful "attentional trace" (Näätänen, 1988), dependent on participant rehearsal, rather than an automatic sensory-memory trace dependent on the physical and temporal characteristics of stimuli (e.g., N1 component 1; Näätänen, 1990). The lack of a PN amplitude difference between stimulus types is also consistent with Alho et al. (1987), who showed that PN can be elicited by both attended and ignored stimuli.

Central P2 increased in positivity as interval increased, a finding broadly consistent with temporal probability (Fitzgerald & Picton, 1981) and ISI (Miltner et al., 1991; Polich, 1990b) studies. Figure 7 shows that the increase in P2 was most apparent after the 2 s TTI/NNI, a pattern in line with Woods and Courchesne's (1986) refractory study, where P2 amplitude had partly recovered by 1500 ms. Compatible with De Blasio and Barry (2013), P2 was more positive to nontargets than targets. That finding supports Crowley and Colrain's (2004) assertion that the P2 marks the withdrawal of attention from a stimulus.

In line with previous TTI (Croft et al., 2003; Gonsalvez et al., 1999, 2007; Gonsalvez & Polich, 2002; Steiner et al., 2013a,b), NNI (Steiner et al., 2014), and nontarget sequence length studies (Duncan-Johnson & Donchin, 1977; Hermanutz et al., 1981; Johnson & Donchin, 1980; Sams et al., 1983; Squires et al., 1977; Starr et al., 1997; Verleger, 1987), P3b amplitude increased as stimulus-to-matching-stimulus interval increased, before reaching a plateau around 10 s. Targets and nontargets showed no difference in the rate of P3b amplitude increase over intervals, and in congruence with Squires et al. (1977), did not differ overall between stimulus types. This relatively large parietal nontarget P3b, together with the similar target/nontarget response profiles over intervals, suggests that targets and nontargets may have been processed similarly. Duncan-Johnson and Donchin (1977) reported a similar

finding for the equiprobable condition in their varied probability study, indicating that similarity in target/nontarget P300 amplitudes may be due to the equiprobable task. Speculatively, equiprobable tasks with highly variable ISIs may facilitate additional processing of nontargets, compared to traditional oddball tasks with highly probable standards (Duncan-Johnson & Donchin, 1977). This corroborates Sawaki and Katayama's (2006) assertion that the target/standard context determines whether nontargets are processed as task-relevant, background, or distracting information.

The bipolar SW did not show an amplitude change over intervals, but was more positive overall to targets than nontargets, a finding largely consistent with previous research (Fitzgerald & Picton, 1981; Hermanutz et al., 1981; Starr et al., 1997). As discussed in Steiner et al. (2014), the definition of SW topography can seriously alter the outcome of an analysis. Here, we followed Dien's (2012) suggestion that the frontal and negative aspects of SW represent only a single component, and optimised our analysis by selecting the frontal/parietal difference.

RT demonstrated a similar response profile to Steiner et al. (2013a, 2014), showing an initial increase, followed by a decrease after the 5 s interval. Unlike P300 measures, which show consistent TTI effects, RT can either increase (Gonsalvez et al., 2007), decrease (Steiner et al., 2013b), or show a combination of those trends (as reported here). This mixture of results is not surprising, given the wide variety of paradigms with which TTI has been explored, and that RT varies greatly with temporal expectancy (e.g., variable foreperiod paradigms; Thomaschke et al., 2011), response readiness (generated by sequential and strategic factors; e.g., Verleger, 1997), task requirements (e.g., oddball vs. choice reaction time), and response facilitation (e.g., cued tasks). Several of these mechanisms may have contributed to the RT response profile reported here. That is, the variable ISI and randomised

stimulus-sequences continually changed response requirements, and this may have affected response readiness by disrupting participant strategies.

Regression analyses demonstrated that interval explained most of the variance in N1 and P3b amplitudes, a finding consistent with Gonsalvez et al. (2007), where N1 was found to correlate with P300 over TTIs. P2 was also predicted by interval, but this effect was weak. This suggests a similar, or perhaps the same mechanism is affecting N1, P3b, and somewhat P2. Over a quarter of the variance in SW was predicted by P3b, confirming the link between P3b and SW reported previously (Barry & Rushby, 2006; Barry & De Blasio, 2013). Importantly, PN was not determined by interval, stimulus type, or previous ERP components, indicating other determinants. For N1 and P3b, the degree of component amplitude change over intervals differed considerably. That is, as interval increased, N1 amplitude increased at 0.16 µV/s, where P3b increased to a greater extent at 0.25 µV/s; approximately a 200 % overall increase. Increases in P2 amplitude are difficult to interpret, as P2 was near-zero at the first interval level, meaning relative increases are exaggerated. Together, the overall regression results indicate that the stimulus-to-matching-stimulus interval effects reported for P300 are not the result of a single mechanism, such as a simple refractory effect progressing throughout the ERP from N1[4], but rather that a similar temporal mechanism is operating on non-sequential components (N1, P2, P3b). The different rates of increase suggest differential sensitivities to this mechanism. This view is compatible with Woods and Courchesne (1986), who demonstrated the dissociation in refractory properties of exogenous and endogenous components of the auditory ERP. TTI/NNI effects throughout the ERP may be evidence of refractoriness in a diffusely connected "system" e.g., memory (which is also connected to perceptual registries; Wagner, 1981) rather than the specific generators of particular

[4] A further stepwise regression, without the interval factor, was conducted to confirm that interval was an independent predictor of P3b. No significant predictors were found, suggesting that the effect of interval on N1 was not translated to P3b.

components (e.g., the network of regions that generate the N1). Furthermore, the lack of interval effects in sequential components (PN) suggests that stimulus processing is occurring in multiple parallel pathways, some of which may be unaffected by temporal changes in stimulus presentations.

The purpose of this study was to follow-up on interval effects Steiner et al. (2014) observed in ERP components other than P300. Here, we showed that as interval increased, N1, P2, and P3b amplitudes increased; PN and SW did not show the same trends. When the determinants of these component amplitudes were examined with multiple regression, N1 and P3b were predicted by interval, P2 was determined by interval to some extent, PN had determinants other than the examined variables, and SW was predicted by P3b. These data indicate that a similar mechanism is operating on the processing stages reflected in N1, P2, and P3b, rather than throughout the entire stimulus processing sequence. Together, this suggests that there is not a simple definitive mechanism (such as a stimulus-pathway refractory period) underpinning interval effects consistently throughout the ERP, but rather that stimuli are processed in several parallel pathways, which are not all affected by matching-stimulus intervals. Future research should focus on determining whether these effects are present at the single-trial level in more typical ERP tasks.

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Figure Legends

Figure 1. An example of the stimulus sequence: targets (T), nontargets (N), and silence (shaded rectangles). Each rectangle illustrates the 1 s SOA. An example of six sequential TTIs (5, 1, 15, 1, 3, and 2 s) and seven NNIs (2, 3, 5, 5, 1, 1, and 5 s) are illustrated above and below, respectively.

Figure 2. Left: Grand mean ERP waveforms from midline sites for targets and nontargets; analysed components are labelled at Fz. Middle: Waveforms constructed from the sum of the eight factors extracted from the PCA. Right: Difference between original data (left) and PCA derived data (middle); any deviations are small.

Figure 3. Average ERPs at midline sites for each of the five matching-stimulus intervals examined (illustrated by the figure key; left column, Cz); targets (left), nontargets (right); components analysed are labelled at Fz.

Figure 4. Top: Headmaps for each of the eight components averaged across all subjects, stimulus types, and intervals. Factor order, latency, and percentage of total variance explained, is indicated below each. Middle: Factor loadings (μV) for the eight components identified. The solid lines indicate factors analysed, dashed lines represent factors explaining < 3 % of the total variance. Bottom: Target and nontarget headmaps averaged across subjects and intervals. Contour lines for very negative components (N1 and PN) are shown in grey to increase their visibility.

Figure 5. Target and nontarget N1 across relevant TTIs/NNIs. A significant across-stimulus type linear trend is apparent, where N1 is increasing in negativity at 0.16 μV/s. The overlapping unfilled markers at the end of the series indicate that there is no overall difference in N1 amplitudes between targets and nontargets.

Figure 6. PN over intervals. No substantial linear trend or stimulus-related effect is apparent.

Figure 7. P2 over TTIs and NNIs. Main effects of interval and stimulus type are apparent.

Figure 8. P3b Increased as matching-stimulus interval increased; this did not differ with stimulus type. The slope coefficient indicates that P3b is increasing at a rate of 0.25 μV/s.

Figure 9. SW amplitudes over TTIs and NNIs with a non-significant linear trend illustrated. Amplitudes were more positive to targets than nontargets.

Figure 10. Mean RT as a function of TTI with standard error bars; the dashed line represents the quadratic trend.