

# ERPs and the evoked cardiac response to auditory stimuli: Intensity and cognitive load effects

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The evoked cardiac response (ECR) may be described as the sum of two independent response components: an initial HR deceleration (ECR1), and a slightly later acceleration (ECR2), hypothesized to reflect stimulus registration and cognitive processing load, respectively. This study investigated processing load effects in the ECR and the event-related potential (ERP). Stimulus intensity was varied within subjects, and cognitive load was varied between subjects, in a counting/no counting task with a long interstimulus interval. The ECR showed a significant effect of counting, but not intensity. ERPs showed the expected obligatory processing effects in the N1, and substantial effects of cognitive load in the Late Positive Complex. Both ERP components varied with intensity. These novel data offer support for ANS-CNS similarities in reflecting some aspects of stimulus processing, but further work is needed to understand the possible contribution of ERP subcomponents to these effects.

Key words: evoked cardiac response, event-related potential, N1, P3, late positive complex

The phasic evoked cardiac response (ECR) elicited by an innocuous stimulus has a complex, often multiphasic, form thought to be affected by both stimulus and situational factors (Barry 1987). In its simplest form, the response is a brief heart rate deceleration referred to as ECR1, which has been linked with stimulus detection or registration (Barry 1987, 1996, 2006, 2009). Increasing stimulus significance, by requiring the subject to respond to the stimuli, results in an additional acceleratory component (ECR2), leading to a biphasic compound response (ECR1 + ECR2). Barry (1984c) demonstrated that even the simplest of cognitive tasks, e.g. counting the stimuli, results in a significant increase in the acceleratory ECR2 over trials. Subsequent research has identified the acceleratory ECR2 as a marker of cognitive load or mental performance (e.g. Barry and Tremayne 1987, Kaiser et al. 1996, 2001). Thus, the biphasic ECR is taken as the sum of two independent response components, thought to reflect different aspects of information processing.

Subtraction of responses (ECR1 from the biphasic ECR1 + ECR2), under conditions varying stimulus significance, is necessary to estimate the ECR2. The advantage of studying the ECR under such conditions is that it shows two different response components, which appear under different circumstances: the first an obligatory ‘transient detection’ response produced to all stimuli, and the second a reflection of ‘cognitive load’, an additional response indicating further processing of stimuli with some significance. Similarly, ERPs allow investigation of the sequential aspects of stimulus processing, using the timing and amplitude of components to elucidate the processing required by any given stimulus. Two components consistently linked to the processes thought to be represented by the ECR are the N1 complex and Late Positive Complex (LPC).

The N1 is a negative component occurring approximately 100 ms post-stimulus onset, and is usually largest in the fronto-central region when elicited by auditory stimuli (Vaughan and Ritter 1970). However, research involving the auditory N1 has indicated that it does not reflect a single underlying cerebral process and should thus not be considered as a unitary phe-

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nomenon (Vaughan and Ritter 1970). Generally, the N1 is thought to represent the initial extraction of information from sensory analysis of the stimulus (Näätänen and Picton 1987), or the excitation relating to the allocation of a channel for information processing out of the primary cortex (Hansen and Hillyard 1980).

The later P3, as it was originally identified, is a pronounced positivity over parietal areas approximately 300 ms after the presentation of an infrequently occurring stimulus (Sutton et al. 1965). However, rather than a single entity, the P3 component has been shown to represent a complex response, with the balance of components/processes differing with experimental design (see Rushby et al. 2005 for a review). Vaughan and Ritter (1970) proposed a change in nomenclature, introducing the more suitable 'late positive complex' (LPC) label used here. Subsequently the LPC has been referred to as the P3b, in addition to the P3 or P300, and has been associated with orienting, attention, stimulus evaluation and memory (e.g. Courchesne et al. 1975, Squires et al. 1975a,b). The amplitude of the LPC has also been demonstrated to be enhanced with increased stimulus intensity (e.g. Picton and Hillyard 1974, Polich et al. 1996, Rushby et al. 2004) and significance (e.g. Donchin and Coles 1988, Squires et al. 1975a,b, 1977).

The P3a is an early fronto-central positivity elicited by infrequent stimuli differing along a single dimension (e.g. frequency) from standard stimuli (Snyder and Hillyard 1976, Squires et al. 1977). This subcomponent is thought to be enhanced with reduced stimulus probability (Johnson 1993), and may also reflect an involuntary switching of attention (Näätänen et al. 1992), response inhibition (Goldstein et al. 2002), and an involuntary OR to unexpected or novel stimuli (Courchesne et al. 1975, Squires et al. 1975a,b). More recent research has indicated that task demands may determine P3a topography (Polich 2007, Wronka et al. 2008).

Previous research has investigated similarities between the cardiac response and indices of central processing (e.g. Kaiser et al. 1996, 1999, Lyytinen et al. 1992, Simons et al. 1998, Zimmer et al. 1991). However, they have been complicated by the inclusion of stimuli not required to affect change in either the cardiac or ERP response. There is a paucity of data which examines these measures in simple single-stimulus conditions, without the added complexities of superfluous

stimuli, clinical populations, or active tasks. If there are similarities between the eliciting conditions for the ECR and the above ERP components, single stimulus conditions should yield clearer effects in the two systems, and facilitate their comparison. Thus, the novelty of this study was the examination in normals of the N1 complex and LPC under simple passive, single-stimulus conditions known to reliably produce differences in the ECR. It was expected that similarities may be drawn between the eliciting conditions of these two sets of measures, and that such findings may be used as a basis for clarifying relationships between ANS and CNS measures during stimulus processing.

Twenty (10 male) university students (aged 19–29,  $M = 21.4$  years), participated in the experiment. The procedure was explained and written consent obtained in accordance with a protocol approved by the joint Illawarra Area Health Service/University of Wollongong Human Research Ethics Committee, in line with the Declaration of Helsinki (WMO 1996). Participants with a history of hearing problems, seizures, psychiatric illness or head injuries, and those who were currently taking psychoactive drugs or had consumed caffeine within 2 hours prior to the test session, were not included.

Participants were instructed to fixate on a central cross displayed on a computer monitor in front of them. Stimuli consisted of 1000 Hz tones with a duration of 1000 ms (15 ms rise/fall times) at 50 or 80 dB SPL intensity, transduced binaurally through stereo headphones. All participants completed two blocks and were alternately assigned to a Count or No Count condition. Subjects were presented with 10 stimuli/block, plus 0–5 extra tones (not analyzed) to prevent identification of a fixed target number. Each block presented a single tone intensity, followed by the alternate intensity in the second block, counterbalanced between subjects within each condition. Tones were presented using a randomly variable ISI of 45–75 s (mean block length ~12 min). In the Count condition, subjects (silently) counted the number of tones presented in the block, and reported the total at the completion of each block. Only subjects who reported the correct number of tones were used in the study ( $n=20$ ). Subjects in the No Count condition were given no instruction in relation to the tones, and were asked simply to relax and fixate on the centralized cross on the computer monitor.

A digital signal-processing hardware and software package from Associative Measurement (AMLAB II),

in conjunction with an IBM compatible computer, was used for data acquisition and storage. HR was recorded using a pair of pre-jelled disposable Ag/AgCl electrodes positioned at mid-sternum and over the third rib on the left mid-axillary line. The signal was recorded as continuous EKG, amplified  $\times 10\,000$ , and sampled by a 16 bit A/D converter at 512 Hz. EKG was analyzed using a locally produced R-wave peak detection program to compute R-R intervals in ms. Measures of cardiac activity were calculated in terms of mean values of HR for 0.5 s intervals relative to event onset (Velden and Wölk 1987), with each epoch of data commencing 5.5 s before stimulus onset and ending 10.5 s after stimulus onset. Only the first ten responses from each block were analyzed.

EEG was recorded from 19 scalp sites (Fp1, Fp2, F7, F3, Fz, F4, F8, T3, C3, Cz, C4, T4, T5, P3, Pz, P4, T6, O1, and O2) using an electrode cap referenced to linked ear lobes and grounded by a cap electrode located midway between Fz and Fpz. Vertical eye movement (vEOG) was measured using tin cup electrodes placed 1 cm above and below the left eye. Horizontal eye movement (hEOG) was monitored from electrodes placed 1 cm beyond the outer canthus of each eye. All electrode impedances were below 5 k $\Omega$ , and care was taken to match the ear impedances. Signals were amplified (EEG  $\times 20\,000$ , EOG  $\times 5\,000$ ) with a bandpass down 3 dB at 0.03 and 30 Hz. The continuous EEG data were analyzed using Neuroscan software (version 4.3; Compumedics, Abbotsford, Australia). The ERP epoch ranged from 1 000 ms pre- to 1 000 ms post-stimulus and was baselined to 100 ms pre-stimulus, and digitally low-pass filtered down 48 dB at 25 Hz. Epochs were subjected to artefact correction, where vEOG was subtracted from the EEG using a regression algorithm in the time domain (Semlitsch et al. 1986). Baseline-to-peak amplitudes were calculated for the N1 complex (defined as the maximum negativity in the 120–150 ms time range following stimulus onset) and the LPC (the maximum positivity 285–365 ms following stimulus onset). The peak markers were confirmed using visual inspection, with manual adjustment if necessary.

The ECR was analyzed using an ANOVA examining response trends in the 5 s following stimulus onset relative to the pre-stimulus HR value. The analysis examined Count (Count/No Count) as a between-subjects factor, and Intensity (Soft/Loud) and Time (shape of the response) as within-subject factors in the design.

Simple (linear, quadratic, cubic) trends over time were used to define response effects. Generally, a brief phasic cardiac response is indicated by a quadratic trend over a short time period and/or a cubic trend if the response is not symmetrical in the time period. This cubic trend may be supplemented/replaced by a linear trend if the response is incomplete in the time period.

ERP analyses were restricted to the sites F3, Fz, F4, C3, Cz, C4, P3, Pz, and P4, in a  $3 \times 3$  (sagittal  $\times$  lateral) matrix. Amplitude and latency measures were subjected to an ANOVA with Count (Count/No Count) as a between-subjects factor, and Intensity (Soft/Loud), Sagittal (Frontal/Central/Parietal), and Lateral (Left/Midline/Right) as within-subjects factors. A planned contrast on the Count factor compared the mean of the Count condition with the mean of the No Count condition, and a contrast for Intensity considered whether the tone was loud or soft within this context. Orthogonal planned contrasts for the Sagittal factor compared frontal activity with parietal, and the mean of these with activity at central sites. For the Lateral factor, contrasts compared left with right hemisphere activation, and the mean of the hemispheres with the midline. These contrasts are optimal for elucidating topographic effects within the sites studied.

As the contrasts for both measures were planned and there were no more of them than the degrees of freedom for effect, no Bonferroni-type adjustment to  $\alpha$  was necessary (Tabachnick and Fidell 1996). Also, Greenhouse-Geisser type correction was not necessary because single degree of freedom contrasts are not affected by the violations of symmetry assumptions common in repeated-measures analyses of physiological data (O'Brien and Kaiser 1985). Where there were main effects of Count or Intensity, ERP data were also submitted to vector scaling (McCarthy and Wood 1985), and only condition  $\times$  topography interactions that remained significant after this procedure are reported. All contrasts reported have (1, 18) degrees of freedom.

The grand mean ECR averaged across Count and No Count conditions is shown in Fig. 1A. There is a brief initial deceleration, followed by an acceleration recovering towards baseline at around 3 s, together indicated by significant linear ( $F=7.34$ ,  $P<0.05$ ), quadratic ( $F=5.62$ ,  $P<0.05$ ) and cubic ( $F=37.08$ ,  $P<0.001$ ) trends. This biphasic response was consistent with the cardiac responses observed in previous research investigating the effects of situational requirements (e.g.

Barry 1984b,c). As found in previous research (e.g. Barry 1977a, 1978, Barry and James 1981) no significant main effects of Intensity, or interactions with Time, were observed (Fig. 1B). Figure 1C shows the ECRs for the Count vs. No Count conditions. For the No Count condition, a simple deceleratory ECR1 can be seen, which gradually returns towards baseline during the 5 s period following stimulus onset. This simple deceleration is consistent with previous research associating the early cardiac response with stimulus registration and transient detection (Barry 1977a,b, 1978, 1984a, 1987). In contrast, the Count condition shows an additional acceleration following the initial deceleratory ECR1 in the same time period. Also shown on the figure is the hypothetical ECR2, the difference between responses in the Count and No Count conditions. The ECR2 shows a rapid acceleration immediately following stimulus onset, which slowly returns toward baseline late in the 0–5 s period. This is reflective of previous research associating the acceleration with more complex cognitive processing (Barry 1996, 2006). Significant differences in the response profiles of the two conditions were indicated by differences in the linear ( $F=7.00$ ,  $P<0.05$ ) and quadratic ( $F=5.45$ ,  $P<0.05$ ) trends during the time period, leading to a significant main effect ( $F=7.99$ ,  $P<0.01$ ), with greater overall acceleration for the Count condition relative to No Count. No Count  $\times$  Intensity interactions were observed.

Grand mean ERPs for responses to Loud and Soft stimuli are shown for the three midline sites in Fig. 2A. Intensity effects are evident in both the N1 and LPC (see Fig. 2A, note particularly the difference wave). Figure 2B shows grand mean ERPs along the midline for Count and No Count conditions. A difference wave illustrates the enhanced LPC observed with the increased cognitive demand of the Count condition.

The N1 amplitude (mean latency 139.5 ms, SD 6.3 ms) showed a strong fronto-central maximum (frontal  $>$  parietal:  $F=7.37$ ,  $P<0.05$ ; central  $>$  frontal/parietal:  $F=17.22$ ,  $P<0.001$ ), and also a midline  $>$  hemispheres effect ( $F=26.21$ ,  $P<0.001$ ). A Sagittal  $\times$  Lateral interaction indicated that the amplitude difference at midline sites relative to the hemispheres was greater centrally than in frontal and parietal regions ( $F=24.25$ ,  $P<0.001$ ). This topography is consistent with the traditional topography commonly observed in a variety of auditory paradigms (Vaughan and Ritter 1970, Picton et al. 1974), and specifically compatible with both the early

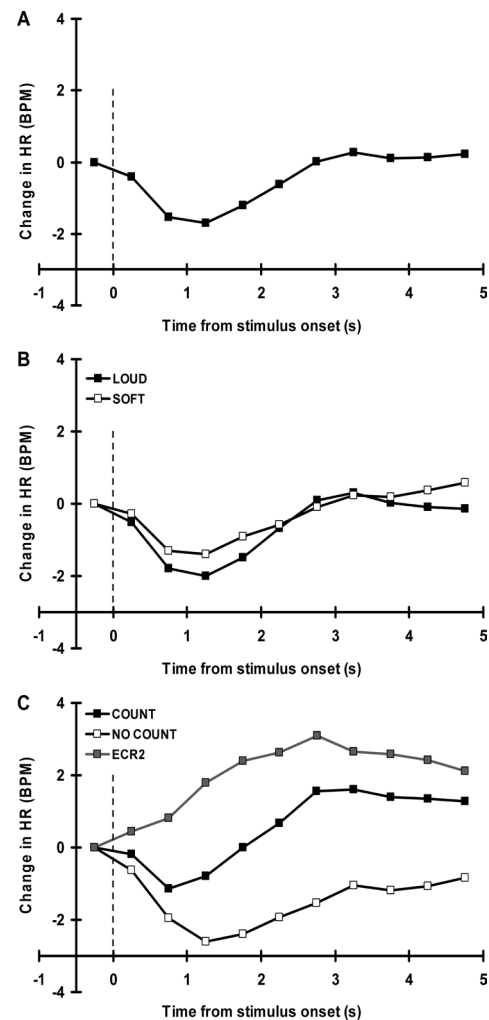


Fig. 1. Grand mean evoked cardiac responses at 0.5 s intervals from stimulus onset. Panel A shows the ECR averaged across Count and No Count conditions. Panel B shows the mean ECR for responses to Loud vs. Soft stimuli. Panel C shows the mean ECR responses for Count vs. No Count conditions and the hypothetical ECR2, included to isolate the effect on the ECR of the instruction to Count. Y-axes represent relative change in HR measured in beats per minute (BPM).

temporal and vertex subcomponents of the N1, thought to reflect physical stimulus properties, in addition to transient detection (Näätänen and Picton 1987).

There was an enhancement of the midline  $>$  hemispheres effect for Loud vs. Soft stimuli ( $F=21.35$ ,  $P<0.001$ ). Additional Intensity  $\times$  Sagittal  $\times$  Lateral interactions revealed that, for responses to Loud stimuli, the difference between the midline and hemispheres was greater in frontal regions, whereas for responses to Soft stimuli this difference was greater in

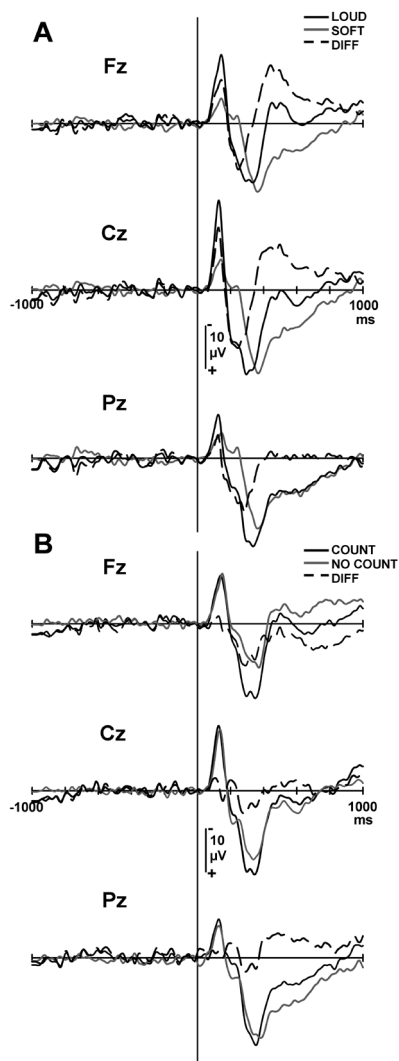


Fig. 2. Grand mean ERPs as functions of intensity and cognitive load. Calibration is relative to 100 ms pre-stimulus baseline. Vertical axes represent stimulus onset, amplitude in  $\mu\text{V}$  and time in ms are marked at Cz. (A) Soft vs. Loud stimuli; (B) Count and No Count stimuli, at Fz, Cz and Pz. The difference (DIFF) between grand mean responses is also included in each panel.

parietal regions ( $F=4.99$ ,  $P<0.05$ ). Further, the vertex effect described in the overall topography for the N1 was greater for responses to Loud than Soft stimuli ( $F=4.99$ ,  $P<0.05$ ). These topographic changes contributed to an overall main effect of Intensity ( $F=28.00$ ,  $P<0.001$ ), with greater overall activation for responses to Loud vs. Soft stimuli, as suggested by the difference wave in Fig. 2A. These effects suggest that subcomponents in the N1 complex are differentially sensitive to stimulus intensity. While not initially expected, inten-

sity differences in the N1 component have been reported previously. On a general level, decreased stimulus intensities have been linked to decreased N1 amplitudes (Beagley and Knight 1967, Picton et al. 1977). More specifically, subcomponents of the N1 have been linked with identification of physical properties of the stimulus, in addition to the detection of the stimulus itself (Näätänen and Picton 1987). The supratemporal subcomponent of the N1 (Component 1), identified by Näätänen and Picton (1987), is described as fronto-central and changes with intensity. However, the vertex subcomponent (Component 3), with timing similar to the supratemporal subcomponent, is "most easily recorded in response to auditory stimuli presented at intensities of greater than 60 dB SPL and at ISIs of greater than 4–5 s" (Näätänen and Picton 1987, p. 412). Given that the Loud tone in this study was 80 dB and the Soft tone was 50 dB, it is possible that an additional subcomponent was produced by Loud stimuli, consistent with Component 3, which may underlie these topographic effects, especially the Intensity  $\times$  Sagittal  $\times$  Lateral interaction.

In terms of cognitive load, no N1 complex differences were observed between Count and No Count conditions (see Fig. 2B, particularly the difference wave). This finding is as expected, and is supportive of the general notion of the N1 as an index of stimulus registration or stimulus detection (e.g. Näätänen 1986, 1990, Squires et al. 1973, 1975a).

The LPC (mean latency 347.1 ms, SD 2.9 ms) showed a parietal maximum ( $F=29.16$ ,  $P<0.001$ ), and also a midline  $>$  hemispheres effect ( $F=95.86$ ,  $P<0.001$ ), and is compatible with an extensive body of research in a range of paradigms implicating this complex in processes such as attention, orienting and stimulus evaluation (e.g. Courchesne et al. 1975, Squires et al. 1975a,b).

An Intensity  $\times$  Sagittal interaction approached significance, where the parietal  $>$  frontal effect was enhanced for responses to Loud vs. Soft stimuli ( $F=3.80$ ,  $P=0.067$ ), note this in the difference wave in Fig. 2B. Further, there was a significant Intensity  $\times$  Sagittal  $\times$  Lateral interaction ( $F=5.73$ ,  $P<0.05$ ), with a midline  $>$  hemispheres difference larger centrally relative to frontal and parietal regions for Loud stimuli, and the reverse for Soft stimuli. LPC amplitude has been demonstrated in previous research to be enhanced with increased stimulus intensity (e.g. Picton and Hillyard 1974 Polich et al. 1996, Rushby et al. 2004),

and research has linked intensity specifically with the P3a (Johnson 1993). Thus, it is possible that the topographic effects with intensity in the LPC here are due to the enhancement of the P3a subcomponent in responses to the Loud stimuli.

In terms of Cognitive Load, the parietal > frontal effect in the overall component topography was greater for responses in the No Count condition ( $F=14.67$ ,  $P<0.001$ ), with a reduced frontal LPC in this condition relative to the Count condition. This difference was due to a marked increase in frontal activity in the Count condition, indicating additional processing reflected in a frontal P3 subcomponent, which may be identified tentatively as the P3a. Research has indicated that task demands are an important factor influencing P3a amplitude (Donchin et al. 1997, Gaeta et al. 2003). Thus, if task demand is increased by requiring the subject to attend to and count the stimulus, then the P3a may be elicited or enhanced, relative to conditions without stimulus significance, such as the No Count condition. Importantly, the overall amplitude of the LPC was greater in the Count than No Count condition, consistent with previous research suggesting that the LPC is enhanced with increased stimulus significance (Donchin and Coles 1988, Squires et al. 1975a,b, 1977), and specifically the processing involved in counting tasks (e.g. Picton et al. 1974, Squires et al. 1973, 1977). The existence of multiple subcomponents of the LPC may also elucidate the Count  $\times$  Intensity  $\times$  Sagittal interaction ( $F=7.62$ ,  $P<0.05$ ), which indicated that in the No Count condition the parietal > frontal effect was greater for Loud vs. Soft stimuli; in the Count condition, this effect was not present. It would appear that for Soft stimuli, evidence of the expected parietal P3b was observed, associated with attentional processing and increased significance, which was enhanced for Count vs. No Count conditions. However, for responses to Loud stimuli, an additional frontal P3a subcomponent was apparent, which was enhanced, along with the P3b, for the Count vs. No Count condition. Together, these results indicate the differential contribution of multiple subcomponents of the LPC to the processes underlying stimulus detection, discrimination, and factors such as significance and task difficulty. More specifically, these results suggest a greater understanding of these processes may be gained through the identification of the individual subcomponents of the LPC in future research.

While the intensity differences observed in the N1 complex diverge from the findings in the ECR, this does not necessarily indicate that the two measures are reflective of separate processes. Rather, it suggests the importance of delineating the subcomponents of the N1 complex and the individual processes these may represent. Based on the results of the present study, it would appear that some subcomponent(s) of the N1 complex (such as Component 2) are similar to early autonomic indices of stimulus detection, such as that observed in the ECR under No Count conditions, while others are more readily influenced by the manipulation of stimulus parameters (e.g. Components 1 and 3). Importantly, there are similarities between the response profile expected of ECR1 and observed for the N1 in Count vs. No Count conditions, indicating that this ERP complex does not reflect the executive processes associated with cognitive load (see Figs 1C and 2B).

Similarly, intensity differences were not reflected in the ECR, but were observed in the LPC (most likely P3b). The results obtained suggest that several subcomponents are contributing to the overall LPC topography. Thus it is possible that some subcomponents of the LPC, and the conditions under which they are elicited, are more similar to the ECR than others. In terms of Count, the ERC and the LPC (specifically P3a), were similar in both showing an additional/enhanced response under increased cognitive load. Figure 1C shows the hypothetical ECR2 as an additional large acceleratory response, and similarly a distinct difference between conditions occurs in the time range of the LPC, as illustrated by the difference wave in Fig. 2B (most notable at Fz). These findings reinforce the notion that these later measures are related to more complex aspects of stimulus processing than the earlier deceleratory ECR1 and N1 complex, which appear to be more closely reflective of the initial aspects of the processing sequence, such as stimulus detection, as exemplified in the No Count condition.

This study has provided some new insights into similarities and differences between the evoked cardiac response and event-related potentials, using basic manipulations thought to affect the conditions under which these components are elicited, without the complexities found in previous research. Future research may benefit from examining this relationship in a within-subjects context, and utilizing somewhat-shorter ISIs. Identification of subcomponents and a larger-

scale within-subjects design will increase the power to allow statistical correlates to be examined in future work. Importantly, some evidence has been provided to suggest that pairs of components in these measures (ECR1 and N1; ECR2 and P3) show similarities in relation to some aspects of stimulus processing. However, the findings also suggest that further evaluation is needed to understand exactly how these two measures interact in reflecting aspects of the stimulus processing sequence, and specifically the differential contribution of subcomponents to the ERP responses associated with these processes.

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