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Research Article

Effects of Cardiac and Respiratory Phases on Auditory Evoked Potentials

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Highlights

- Auditory N1 and P2 amplitudes were compared across cardiac and respiratory phases.
- No differences in N1 amplitudes were found between cardiac and respiratory phases.
- P2 amplitude was greater during diastole than systole.
- P2 amplitude was greater during exhalation than inhalation only for ignored tones.
- Visceral afferent signals can influence auditory processing.

Abstract

Brain-body interactions play a crucial role in the perceptual and cognitive processing of external stimuli. Previous research has examined how cardiac phases (systole, diastole) and respiratory phases (inhalation, exhalation) influence various psychological functions, though findings on their impact on auditory processing remain inconsistent. This study investigated whether cardiac and respiratory phases affect auditory ERP components, specifically N1 and P2. To control for cardiac-related artifacts, pure tones (70 dB) and silent stimuli (0 dB) were presented in alternating, randomized intervals, and ERP difference waveforms were computed by subtracting waveforms elicited by silent stimuli from those elicited by tones. Two experiments were conducted with different participants: watching a video while ignoring the tones (Experiment 1) or pressing a button as quickly as possible in response to the tones while watching the video (Experiment 2). Results showed no significant differences in N1 amplitude between cardiac or respiratory phases. P2 amplitude was significantly larger at diastole than systole, although the effect size was small ($d_z = 0.26$). For respiratory phases, P2 amplitude was greater during exhalation than inhalation when participants ignored the tones ($d_z = 0.35$), but this effect disappeared when they attended to the tones. These findings suggest that visceral afferent signals may influence auditory processing by modulating attentional resource allocation across different cardiac and respiratory phases.

Keywords

Cardiac Phase, Respiratory Phase, Event-related Potential, Auditory Processing, N1, P2

1. Introduction

Research has established that brain-body interactions are critical in perceptual and cognitive processing of external events. The brain continuously processes external information (e.g., visual or auditory stimuli) and internal bodily signals, including cardiac activity and respiration (for reviews, see Engelen et al., 2023; Parviainen et al., 2022). This suggests that the perception or cognition of external information may be influenced by dynamically changing internal physiological states. Recently, an increasing number of studies have been investigating the effect of cardiac and respiratory phases on the processing of external events.

The cardiac cycle is divided into systole and diastole. During systole, the heart contracts, and stretch-responsive arterial baroreceptors transmit information about the heartbeat to the brainstem. In contrast, these baroreceptors are inactive during diastole, and afferent signals from the heart to the brain are mainly absent. Several studies have examined whether cardiac phases modulate the perceptual processing of external stimuli. The enhancements of certain functions—such as emotional processing, voluntary movement initiation, and risky decision-making—have been observed during systole (Garfinkel et al., 2014; Kimura et al., 2023; Kunzendorf et al., 2019). However, the perception in auditory and somatosensory domains and the integration of audio-tactile stimuli tend to be attenuated during systole (Al et al., 2020; Motyka et al., 2019; Saltafossi et al., 2023; Schulz et al., 2009; for a review, see Skora et al., 2022). These findings suggest that cardiac afferent signals may act as noise, dampening perception.

Studies investigating the modulation of auditory processing by cardiac phases have produced mixed results. Schulz et al. (2009) reported that startle eyeblink responses and intensity ratings of auditory stimuli were lower during systole than

diastole. However, other researchers, such as Delfini and Campos (1972) and Velden and Juris (1975), reported that cardiac phases did not affect auditory signal detection. ERP studies have also explored whether cardiac phases influence auditory processing. For example, Schulz et al. (2020) reported that the N1 amplitude, elicited by pure or startle tones, was smaller during systole than diastole, suggesting attenuated auditory processing during systole.

However, Li et al. (2024) found no significant effects of cardiac phases on auditory processing. They investigated whether auditory deviance detection, measured by mismatch negativity (MMN), was modulated by cardiac phases. Auditory MMN is typically observed at the frontocentral site in response to infrequent sounds within a repetitive sequence of sounds 100–250 ms after tone onset (Garrido et al., 2009; Näätänen et al., 2007, 2012). Li et al. (2024) showed no significant differences between systole and diastole. Furthermore, the authors examined intensity-dependent N1 and P2 amplitudes but found no differences between systole and diastole. They attributed the slight increase in N1 and P2 amplitudes to suboptimal stimulus parameters, such as the intensity of the standard tones (80 dB and 60 dB), the use of deviant tones, and the use of 500-ms stimulus onset-to-onset intervals, which were not ideal for capturing N1 and P2 responses. In summary, the relationship between cardiac phases and auditory processing remains unclear due to these inconsistent findings.

The primary aim of this study was to investigate the effects of cardiac phases on N1 and P2 amplitudes. Compared to Li et al. (2024), this study employed longer interstimulus intervals (ISIs) of 2,770–3,370 ms to record more considerable sensory-evoked potentials (Pereira et al., 2014). If cardiac afferent signals disrupt auditory processing, N1 and P2 amplitudes should be larger during diastole than systole. In this

study, ERPs elicited by "silent" stimuli (0 dB) were subtracted from those elicited by "real" tones (1,000 Hz, 70 dB) to eliminate cardiac-related artifacts. Although this subtraction method (Kimura, 2019; Li et al., 2024) has the disadvantage of increasing noise, it is technically more appropriate to completely remove the cardiac field artifact (CFA) effects. Previous studies have utilized independent component analysis (ICA) to identify CFAs. However, determining whether a specific independent component (IC) represents cardiac or brain activity can be challenging (Arnau et al., 2023). Specific studies have manually removed ICs reflecting CFAs (Bury et al., 2019; Kimura, 2019; Li et al., 2024; Marshall et al., 2022; Mizuhara et al., 2024), whereas others have employed automatic classification tools (Pion-Tonachini et al., 2019; Tamburro et al., 2019). Recently, novel approaches have been proposed using neural networks (Arnau et al., 2023) or a combination of ICA and automatic tools (Tanaka et al., 2023). However, cardiac-related artifacts contain not only artifacts associated with the QRS complex, but may also include *ballistocardiogram* artifacts that reflect small head and body movements due to cardiac blood ejection (Hari & Puce, 2023, pp. 156–157). Li et al. (2024) emphasized that the advantage of the subtraction method lies in its ability to completely eliminate (even unspecified) electrical artifacts related to the cardiac cycle. This procedure ensures that only ERP waveforms associated with stimulus processing are retained.

Previous research has also investigated whether another autonomic cycle, respiration, would influence the perceptual and cognitive processing of external events, as well as the brain activity of cortical and subcortical areas (e.g., Belli et al., 2021; Belli & Fischer, 2024; Ghibaudo et al., 2024; Grund et al., 2022; Herrero et al., 2018; Johannknecht & Kayser, 2022; Mizuhara & Nittono, 2023; Perl et al., 2019; Watanabe

et al., 2023; Zaccaro et al., 2024; Zelano et al., 2016; for a review, see Maric et al., 2020; Nakamura et al., 2023). For example, fearful facial expressions were detected faster and more accurately when the visual stimuli were presented during nasal inhalation than nasal exhalation, possibly due to the enhancement of the amygdala's neural activity (Mizuhara & Nittono, 2023; Zelano et al., 2016). However, it is also known that findings regarding the respiratory phase's effect on the visual discrimination (Mizuhara & Nittono, 2022, 2023; Zelano et al., 2016) or memory (Arshamian et al., 2018; Nakamura et al., 2018, 2022; Schaefer et al., 2024; Thunell et al., 2024; Zelano et al., 2016) are inconsistent.

Only a few studies have explored whether respiratory phases also affect auditory processing (e.g., Gallego et al., 1991; Mizuhara et al., 2024; Münch et al., 2019; Schulz et al., 2016; Waselius et al., 2022), and the direction and the size of the effect appear to vary with the nature of the task or stimuli and with differences in methodology. Schulz et al. (2016) reported that startle responses to auditory tones were greater during on-going exhalation than during the other phases (peak inhalation, on-going inhalation, and peak exhalation), regardless of spontaneous or 0.25 Hz paced breathing. They speculated that the relative unloading of phasic slow adapting pulmonary stretch receptors might lead to higher startle responses during on-going exhalation. Münch et al. (2019) showed that the healthy adults rated the intensity of an acoustic startle tone higher when it was presented during exhalation than inhalation in the 0.25 Hz paced breathing condition. They suggested that during exhalation, reduced input from respiratory phase receptors might free up attentional resources, allowing for greater processing of startling auditory stimuli, leading to higher subjective intensity ratings. This mechanism might involve the stretch receptors of the chest wall, which

send bottom-up information to the brain. These studies suggest that such afferent signals could interfere with auditory processing. Regarding ERP studies, Mizuhara et al. (2024) found that the amplitude of auditory MMN was larger during exhalation than inhalation by using the dataset from Li et al. (2024). Moreover, they found a significant effect of respiratory phases on P2 amplitudes elicited by the pure tone. Waselius et al. (2022) also observed that N1 amplitudes elicited by a tone were larger during exhalation than inhalation. During exhalation, the influence of signals from the pulmonary stretch receptors in the chest wall may be diminished, potentially resulting in larger ERP amplitudes. These findings suggest that respiratory phases can modulate auditory N1 and P2 amplitudes, though only a few studies have examined this effect in depth. The present study investigated whether respiratory phases influence N1 and P2 amplitudes elicited by a tone. Based on these previous studies, the present study hypothesized that N1 and P2 amplitudes would be larger during exhalation than inhalation. Similar to the analysis of cardiac phases, N1 and P2 amplitudes in the analysis of respiratory phases were calculated as the difference waveforms by subtracting waveforms elicited by silent stimuli from those elicited by real stimuli. This approach was chosen for two reasons. First, it is better to use the same type of analysis for cardiac and respiratory phases for comparison. Second, since respiratory phases also affect cardiac cycles (respiratory sinus arrhythmia: Berntson et al., 1993), the proportions of systole and diastole differ depending on the respiratory phase. Although the influence of cardiac-related artifacts might be minimal, calculating the difference waveforms could effectively eliminate them.

The current study tested the following hypotheses: (1) The N1 amplitude will be larger during diastole than systole (H1); (2) The P2 amplitude will be larger during

diastole than systole (H2); (3) The N1 amplitude will be larger during exhalation than inhalation (H3); (4) The P2 amplitude will be larger during exhalation than inhalation (H4).

2. Experiment 1

2.1 Methods

2.1.1 Participants

Forty university students (23 females and 17 males, 19–33 years old, $M = 22.9$ years old) participated. All participants were right-handed, as determined by the FLANDERS Handedness Questionnaire (Nicholls et al., 2013; Okubo et al., 2014). None of them reported hearing problems or a history of cardiac disease. We obtained the informed written consent from all the participants. The participants received a cash voucher (2,000 Japanese yen, approximately 15 USD) as compensation for participating. This study's sample size matched that of Li et al. (2024). The Behavioral Research Ethics Committee of the Osaka University School of Human Sciences in Japan (HB023-088) approved this study's protocol.

The study protocol was preregistered before data collection. Details of the preregistration can be found at https://osf.io/7h254, although the preregistration did not plan to investigate the effects of respiratory phases on auditory processing. The numerical data supporting the results of each experiment is available at https://osf.io/4azku/?view_only=bf3ae81515524e2cb76bef1aaf3c3046.

2.1.2 Stimuli and Procedure

The stimuli were generated using Audacity software (44,100 Hz, 16-bit resolution, monaural; https://www.audacityteam.org). The "real" tone consisted of a

1,000 Hz pure tone with a duration of 70 ms, including a 5 ms rise and fall period and an intensity of 70 dB SPL. The "silent" stimuli, which had the same duration as the real ones, were set at 0 dB SPL. The tone intensity was adjusted using a dummy head (SAMURA 3500; Southern Acoustics Co., Ltd., Kanagawa, Japan). An auditory signal detector (StimTrak; Brain Products GmbH, Gilching, Germany) was used to trigger the accurate timing of stimulus onset. Stimuli were delivered through earphones (MDR-EX650; SONY Marketing Inc., Tokyo, Japan) using an analog headphone amplifier (HA501; TEAC Corp., Tokyo, Japan) with equal phase and intensity in both ears.

Figure 1 illustrates the sequence of auditory stimuli. Each block consisted of 250 real and 250 silent stimuli presented alternately with randomized ISIs ranging from 1,350 to 1,650 ms. Thus, real tones were presented at intervals of 2,770 to 3,370 ms. All participants completed four blocks. At the start of each block, three additional real stimuli were presented, which were excluded from the analysis. Participants were instructed to watch a silent animated video (*Tom and Jerry*) without attending to the auditory stimuli. This was done to prevent the participants from falling asleep and to ensure that they maintained a relatively fixed gaze and kept their eyes open during the experiment.

2.1.3 Recordings

The recording setup followed the methods outlined by Li et al. (2024). All physiological data were recorded at a sampling rate of 2,048 Hz using the ActiveTwo System (BioSemi B.V., Amsterdam, the Netherlands) and ActiView 8.13 software, with a bandpass filter set at 0–400 Hz. Electrocardiogram (ECG) data were collected using UltraFlat active electrodes (BioSemi B.V., Amsterdam, the Netherlands) placed from the left lower rib to the right mastoid. Respiratory movements were measured using an air-

tube pneumatic sensor connected to a MaP2290DRA amplifier (Nihon Santeku Co., Ltd., Osaka, Japan), with a rubber bellow tube positioned around the participant's abdomen. Electroencephalogram (EEG) data were recorded from 64 scalp sites using an elastic cap with Ag-AgCl electrodes (BioSemi B.V., Amsterdam, the Netherlands), including bilateral mastoid sites. A common mode sense (CMS) active electrode and a driven right leg (DRL) passive electrode served as the reference for the amplifier. Additional electrodes were placed on the outer canthi and above/below the right eye. An electrode was placed on the nose tip for re-referencing offline, and the offset was maintained within ±25 mV, per the manufacturer's guidelines.

2.1.4 Data Reduction

The data reduction procedure followed the methods used by Li et al. (2024) and Mizuhara et al. (2024). Biosignal data were analyzed using Brain Vision Analyzer 2 (Brain Products GmbH, Gilching, Germany). Figure 2 illustrates the criteria for identifying systole and diastole phases used in this study. A 3–60 Hz bandpass filter was applied to the ECG data (Mulder, 1992). R- and T-wave peaks were detected by using EKG markers solution (v 1.11). Then, the detected ones were visually inspected and corrected when necessary. Each trial was classified into systole and diastole phases based on the timing of stimulus onset. Following Li et al. (2024), systole was defined as the period from 100 ms before to 100 ms after the T-wave peak, whereas diastole was defined as the period from 250 ms to 50 ms before the R-wave peak.

For respiration data, a bandpass filter of 0.05–10 Hz (12 dB/oct) was applied (Mizuhara & Nittono, 2023; Schulz et al., 2016). The inhalation-onset troughs and exhalation-onset peaks were also detected according to Matsuda and Ogawa (2011)’s algorithm, and then corrected when necessary (e.g., manually added the peak or trough

to the point where the detection was missed). The exhalation phase was defined as the interval between an exhalation-onset peak and the following inhalation-onset trough, whereas the inhalation phase was defined as the interval between an inhalation-onset trough and the subsequent exhalation-onset peak.

EEG data were resampled at 512 Hz and filtered with a 1–30 Hz bandpass filter. ICA was used to correct for ocular and cardiac artifacts. The cardiac artifact components were identified and excluded by visual inspection of 71 ICs (64 waveforms + bilateral mastoids + 4 electrodes around the eyes + ECG waveforms). On average, 1.3 components per participant were identified as cardiac artifacts and excluded. Data from a 600-ms window (100 ms before to 500 ms after stimulus onset) were averaged for real and silent stimuli in each cardiac and respiratory phase after excluding artifact periods. Artifacts were identified using the following criteria: (1) voltage exceeding ±60 μV, (2) voltage step greater than 50 μV/ms, and (3) peak-to-peak activity differences of less than 0.5 μV within 100 ms. The baseline period was defined as the 100 ms preceding stimulus onset. Difference waveforms were obtained by subtracting waveforms elicited by silent stimuli from those elicited by real stimuli. N1 and P2 amplitudes were measured as the average of a frontocentral electrode cluster (F1/2, Fz, FC1/2, FCz, C1/2, Cz). To determine latency windows, grand average difference waveforms were calculated across systole/diastole and inhalation/exhalation phases according to the collapsed localizer approach (Luck & Gaspelin, 2017). The most negative peak between 70–150 ms (N1) and the most positive peak between 100–250 ms (P2) was identified, and 40-ms windows centered around these peaks were analyzed.

2.1.5 Exclusion Criteria

Consistent with Li et al. (2024) and Mizuhara et al. (2024), we included the

participants' data in the analysis only if each cell (2 stimuli × 2 cardiac/respiratory phases) contained at least 150 trials.

2.1.6 Statistical Analysis

All statistical analyses were performed using JASP 0.17 (JASP Team, 2023). Two-tailed *t*-tests examined differences in mean ERP amplitudes between cardiac and respiratory phases. Additionally, Bayesian paired-sample *t*-tests were performed to calculate Bayes factors (BF_{10}) for testing the hypotheses regarding the effects of cardiac and respiratory phases on ERP amplitudes.

2.2 Results

2.2.1 Data Exclusion

We excluded one participant from the cardiac phase analysis based on the predetermined exclusion criteria. As a result, the final sample consisted of 39 participants for the cardiac phase analysis (23 women and 16 men; age range: 19–33 years, $M = 22.9$) and 40 for the respiratory phase analysis.

2.2.2 ERPs in Systole and Diastole Phases

On average, 6.4% of systole and 6.6% of diastole trials were excluded from averaging according to the artifact criteria. Table 1 presents the mean number of trials included in the ERP averaging. Table 2 summarizes the N1 and P2 amplitudes for each cardiac phase. Figure 3A shows the ERP waveforms elicited by each stimulus and ECG waveforms recorded from the lower rib. Figure 3B displays the grand mean real-minus-silent difference waveforms for ERPs and ECG signals. In the collapsed difference waveform across cardiac phases, the most negative and positive peaks were observed at 90 ms and 180 ms, respectively. Consequently, the periods from 70–110 ms for N1 and 160–200 ms for P2 were analyzed. Figure 3C provides topographic maps of the mean

amplitudes for each cardiac phase's N1 and P2 latency windows.

As illustrated in Figure 3D, two-tailed paired *t*-tests revealed no significant effects of cardiac phases on N1 or P2 amplitudes, $t(38) = 1.27$, $p = .211$, $d_z = 0.20$, and $t(38) = 1.84$, $p = .074$, $d_z = 0.29$, respectively. Bayesian paired *t*-tests further provided anecdotal evidence supporting the null hypotheses over the alternative hypotheses, $BF_{10} = 1/2.75$ for N1 and $BF_{10} = 1/1.26$ for P2. These results do not support H1 or H2, suggesting that cardiac phases did not influence auditory N1 or P2 amplitudes.

2.2.3 ERPs in Inhalation and Exhalation Phases

Table 1 summarizes the mean number of trials included in the ERP averaging. The mean percentage of excluded trials for inhalation and exhalation was 6.4 % and 6.7%, respectively. Table 2 presents the N1 and P2 amplitudes for each respiratory phase. Figure 4A shows the ERPs elicited by each stimulus, whereas Figure 4B displays the grand mean real-minus-silent difference waveforms of ERPs in each respiratory phase. We analyzed the latency windows of 70–110 ms for N1 (with a peak at 90 ms in the collapsed difference waveforms) and 162–202 ms for P2 (with a peak at 182 ms). Figure 4C shows the topographic maps of the mean amplitudes in the N1 and P2 time windows for each respiratory phase.

As shown in Figure 4D, a two-tailed paired *t*-test revealed no significant effects of respiratory phases on the N1 amplitude, $t(39) = 0.03$, $p = .975$, $d_z = 0.01$. A Bayesian paired *t*-test provided moderate evidence for the null hypothesis over the alternative, $BF_{10} = 1/5.86$. In contrast, a two-tailed paired *t*-test revealed significant effects of respiratory phases on P2 amplitudes, $t(39) = 2.20$, $p = .034$, $d_z = 0.35$. A Bayesian paired *t*-test provided anecdotal evidence in favor of the alternative hypothesis over the null, $BF_{10} = 1.47$. Therefore, H3 was supported, whereas H4 was not.

2.3 Discussion

For cardiac phases, null findings align with Li et al. (2024), who reported that cardiac phases did not significantly modulate auditory ERPs (N1, P2, and MMN). However, cardiac phases might influence auditory ERPs in tasks in which participants must respond to stimuli. Previous research has indicated that reaction times (RTs) to external stimuli differ between cardiac phases (Saltafossi et al., 2023; Schulz et al., 2020; Yang et al., 2017). For instance, Yang et al. (2017) demonstrated that RTs to auditory stimuli (75 dB, 400 Hz sine wave tone, 50-ms duration with a 5-ms rise) were faster when tones were presented during diastole than systole. We speculated that the longer RTs during systole may result from the activation of arterial baroreceptors. These findings imply that cardiac phases might modulate auditory processing, mainly when individuals must attend to auditory events.

In contrast, P2 amplitudes were larger during exhalation than inhalation. The significant difference in P2 amplitudes between inhalation and exhalation may reflect differences in attentional resources allocated to auditory stimuli. Previous studies have demonstrated that ERP amplitudes elicited by auditory stimuli are modulated by respiratory phases (Mizuhara et al., 2024; Waselius et al., 2022). For instance, Mizuhara et al. (2024) indicated that the amplitude of auditory MMN was larger during exhalation than during inhalation. During inhalation, the stretch receptors of the chest wall send bottom-up information to the brain, which may interfere with auditory processing. Therefore, it is speculated that more attentional resources are allocated to the processing of auditory stimuli during exhalation than inhalation, resulting in larger MMN and P2 amplitudes. Interestingly, the effect observed in the present study is opposite to previous studies showing improved visual attentional processing or lower perceptual thresholds

during inhalation (Kluger et al., 2021; Mizuhara & Nittono, 2023; Perl et al., 2019). Although the detailed mechanism remains unclear, this may suggest that the influence of respiratory phases on the processing of external events may differ between visual and auditory modalities. Future studies should explore the difference in the potential functional role between visual and auditory processing during respiratory phases. In addition, Zaccaro et al. (2024) recently reported that the amplitude of the heart-beat evoked potential was greater during exhalation than inhalation, suggesting that attention may be more attuned to interoceptive signals during exhalation than inhalation. This difference in balance between interoceptive and exteroceptive signals may also contribute to ERP amplitude differences related to respiratory phases.

Another possibility is that the difference in brain oxygenation between inhalation and exhalation may affect the auditory P2 amplitude. Muñoz et al. (2023) simultaneously recorded auditory evoked potentials and hemodynamic responses from the functional near-infrared spectroscopy and reported that P2 amplitude correlated with deoxyhemoglobin concentration over the right dorsolateral cortex. Although it is difficult to directly assess the effect, respiratory phase differences in ERP amplitudes may be partly due to differences in brain oxygenation.

The finding that P2 amplitude was significantly larger during exhalation than inhalation appears unrelated to the association between respiration and the cardiac cycle. The R–R interval is longer during exhalation than inhalation, a phenomenon known as respiratory sinus arrhythmia (RSA: Berntson et al., 1993). While the duration of diastole (when cardiac afferent signals are absent) varies with the R–R interval, the duration of systole remains constant. As a result, ERPs recorded during exhalation may contain a higher proportion of diastole periods than those recorded during inhalation.

However, the P2 amplitude differences between respiratory phases are likely unrelated to the cardiac phase because we found no significant difference in P2 amplitude between systole and diastole. In conclusion, this experiment supports previous findings (Münch et al., 2019; Schulz et al., 2016; Waselius et al., 2022) that auditory processing may be more advantageous during exhalation than inhalation.

3. Experiment 2

The aim of Experiment 2 was to investigate the effects of cardiac and respiratory phases on auditory processing when participants attended to tones. Participants were instructed to respond to tones by pressing buttons as quickly as possible. In addition to examining ERP amplitudes, we compared RTs to the tones and button-press error rates between the cardiac and respiratory phases. Studies have explored how respiratory phases affect RTs to external stimuli, though the results have been inconclusive (e.g., Beh & Nix-James, 1974; Buchsbaum & Callaway, 1965; Gallego et al., 1991; Muñoz-Caracuel et al., 2024). Therefore, we examined the relationship between cardiac and respiratory phases and motor responses to auditory stimuli.

3.1 Methods

3.1.1 Participants

Forty university students (28 females and 12 males, 18–29 years old, $M = 21.8$ years old) participated in this experiment. None of the participants had a history of hearing or cardiorespiratory problems. As in Experiment 1, all participants were right-handed, as assessed by the FLANDERS Handedness Questionnaire (Nicholls et al., 2013; Okubo et al., 2014). Participants provided written informed consent to participate

and received a cash voucher (2,500 Japanese yen, approximately 17 USD) for participation. This study's protocol was approved by the Behavioral Research Ethics Committee of the Osaka University School of Human Sciences, Japan (HB024-001).

3.1.2 Stimuli and Procedure

The stimuli, equipment, and presentation settings were identical to those used in Experiment 1, with the exception that the participants were instructed to press two buttons on a response box simultaneously with both thumbs whenever they heard a tone while watching a silent video with subtitles (*Tom and Jerry*).

3.1.3 EEG Preprocessing

The recording settings, data reduction procedures, and exclusion criteria for EEG preprocessing were identical to those used in Experiment 1. On average, 1.3 components per participant were removed as cardiac artifacts. We classified auditory stimuli not followed by a button press within 1,000 ms after stimulus onset as "omission errors" and excluded them from the analysis.

3.1.4 Statistical Analysis

As in Experiment 1, two-tailed *t*-tests and Bayesian factor calculations were conducted to assess the effects of cardiac and respiratory phases on mean ERP amplitudes. Deviating from the preregistration plan, we compared RTs to tones and omission error rates between the cardiac and respiratory phases. We treated RTs exceeding mean ± 3 (*SD*s) as outliers and excluded them from the RT analysis.

3.2 Results and Discussion

3.2.1 Data Exclusion

We excluded two participants from the cardiac and respiratory phase analyses and one participant from the cardiac phase analysis according to the exclusion criteria.

In addition, one participant was excluded from the analyses to avoid possible carryover effects because it was found that this individual had also participated in Experiment 1. As a result, the final sample for the cardiac phase analysis consisted of 36 participants (26 women and 10 men, age range, 18–29 years, $M = 21.9$) and 37 for the respiratory phase analysis (27 women and 10 men, age range, 18–29 years, $M = 21.8$).

3.2.2 ERPs in Systole and Diastole Phases

On average, 6.3% trials for both systole and diastole were excluded from the ERP averaging. Table 1 presents the mean number of trials in ERP averaging. Table 2 shows the mean amplitudes of N1 and P2 for each cardiac phase. Figure 5A illustrates the ERP waveforms elicited by each stimulus and ECG waveforms recorded from the lower rib. Figure 5B shows the grand mean real-minus-silent difference waveforms for ERPs and ECG signals in each cardiac phase. We analyzed the time windows of 70–110 ms for N1 (with a peak at 90 ms in the collapsed waveform) and 138–178 ms for P2 (with a peak at 158 ms). Figure 5C presents the topographic maps of the mean amplitudes in the N1 and P2 latency windows during systole and diastole.

As shown in Figure 5D, two-tailed paired *t*-tests revealed no significant effects of cardiac phases on N1 or P2 amplitudes, $t(35) = 0.63$, $p = .533$, $d_z = 0.11$ for N1, and $t(35) = 1.40$, $p = .171$, $d_z = 0.23$ for P2. Bayesian paired *t*-tests provided moderate evidence for the null hypothesis over the alternative for N1 ($BF_{10} = 1/4.65$) and anecdotal evidence for the null over the alternative for P2 ($BF_{10} = 1/2.29$). As in Experiment 1, no significant differences were observed in N1 and P2 amplitudes between systole and diastole, suggesting that cardiac phases did not influence auditory processing. These findings supported neither H1 nor H2.

3.2.3 ERPs in Inhalation and Exhalation Phases

The mean percentages of trials excluded from ERP averaging were 6.0 % and 6.0% for inhalation and exhalation. Table 1 shows the mean number of trials included in the ERP averaging. Table 2 shows the mean amplitudes of N1 and P2 for each respiratory phase. Figure 6A shows the ERP waveforms elicited by each stimulus, whereas Figure 6B displays the grand mean real-minus-silent difference waveforms for each respiratory phase. We analyzed the time windows of 70–110 ms for N1 (with a peak at 90 ms in the collapsed waveform) and 138–178 ms for P2 (with a peak at 158 ms). Figure 6C presents the topographic maps of the mean amplitudes in the N1 and P2 time windows during inhalation and exhalation.

As shown in Figure 6D, a two-tailed paired *t*-test revealed no significant effects of respiratory phases on N1 amplitude, $t(36) = 1.84$, $p = .075$, $d_z = 0.30$. A Bayesian paired *t*-test provided anecdotal evidence supporting the null hypothesis over the alternative, $BF_{10} = 1/1.25$. Similarly, a two-tailed paired *t*-test revealed no significant effects of respiratory phases on P2 amplitude, $t(36) = 1.01$, $p = .321$, $d_z = 0.17$. A Bayesian paired *t*-test provided moderate evidence for the null hypothesis over the alternative, $BF_{10} = 1/3.54$. These findings supported neither H3 nor H4. The lack of a significant difference in N1 amplitude between inhalation and exhalation was consistent with Experiment 1, whereas the absence of a significant difference in P2 amplitude differed from Experiment 1. These results suggest that the effects of respiratory phases on auditory processing may depend on whether participants are attending to the auditory stimuli.

3.2.4 Response Times and Omission Error Rates

For cardiac phases, the mean RTs during systole and diastole were 301.0 ms (*SD* = 118.8) and 290.3 ms (*SD* = 100.1), respectively. A two-tailed *t*-test revealed no

significant difference between the two phases, $t(35) = 0.81$, $p = .426$, $d_z = 0.13$. A Bayesian paired *t*-test provided moderate evidence supporting the null hypothesis over the alternative, $BF_{10} = 1/4.13$. Similarly, the omission error rate did not differ significantly between systole ($M = 1.76\%$, $SD = 2.94$) and diastole ($M = 1.51\%$, $SD = 2.64$), $t(35) = 1.56$, $p = .128$, $d_z = 0.26$. A Bayesian paired *t*-test provided anecdotal evidence in favor of the null hypothesis, $BF_{10} = 1/1.86$.

For respiratory phases, the mean RTs were 274.6 ms ($SD = 152.7$) during inhalation and 287.2 ms ($SD = 93.6$) during exhalation. Similar to the cardiac phases, we found no significant differences in RTs between respiratory phases, $t(36) = 0.71$, $p = .485$, $d_z = 0.12$. A Bayesian paired *t*-test provided moderate evidence supporting the null hypothesis, $BF_{10} = 1/4.49$. Furthermore, we observed no significant difference in omission error rates between inhalation ($M = 1.90\%$, $SD = 3.33$) and exhalation ($M = 1.73\%$, $SD = 3.01$), $t(36) = 0.70$, $p = .490$, $d_z = 0.12$. A Bayesian paired *t*-test provided moderate evidence in favor of the null hypothesis, $BF_{10} = 1/4.51$. These results suggest that neither cardiac nor respiratory phases influenced motor responses to auditory stimuli.

4. Omnibus Analyses

Omnibus analyses of variance (ANOVAs) were conducted, with cardiac or respiratory phases as within-participant factors and attention (ignored [Experiment 1] vs. attended [Experiment 2]) as a between-participant factor to examine the effects of attention to tones. We also performed Bayesian repeated-measures ANOVAs.

For cardiac phases, a two-way repeated-measures ANOVA on N1 amplitude showed no significant main effects of cardiac phase, $F(1, 73) = 0.10$, $p = .757$, $\eta_p^2 <$

0.01, BF_{10} = 1/6.84, or attention, $F(1, 73) = 0.62$, $p = .433$, $\eta_p^2 = 0.01$, BF_{10} = 1/2.36. The interaction between cardiac phase and attention was also not significant, $F(1, 73) = 1.68$, $p = .199$, $\eta_p^2 = 0.02$, BF_{10} = 1/8.92. However, significant main effects of cardiac phase, $F(1, 73) = 4.97$, $p = .029$, $\eta_p^2 = 0.06$, BF_{10} = 1.32, and attention, $F(1, 73) = 22.42$, $p < .001$, $\eta_p^2 = 0.24$, BF_{10} = 978.51 were found for the P2 amplitude. The P2 amplitudes were larger during diastole than systole and larger when participants ignored the tone than when they attended to it. The interaction between cardiac phase and attention was not significant, $F(1, 73) = 0.01$, $p = .936$, $\eta_p^2 < 0.01$, BF_{10} = 1/1.71. Based on the aggregated data of two experiments ($N = 75$), the effect size of cardiac phase on P2 amplitude was $d_z = 0.26$.

For respiratory phases, a two-way ANOVA on the N1 amplitude showed neither main effects of respiratory phase ($F(1, 75) = 1.62$, $p = .207$, $\eta_p^2 = 0.02$, BF_{10} = 1/3.64) nor attention ($F(1, 75) = 0.81$, $p = .371$, $\eta_p^2 = 0.01$, BF_{10} = 1/1.93), nor an interaction of respiratory phase and attention ($F(1, 75) = 1.74$, $p = .192$, $\eta_p^2 = 0.02$, BF_{10} = 1/5.11). However, the main effect of attention, $F(1, 75) = 21.67$, $p < .001$, $\eta_p^2 = 0.22$, BF_{10} = 684.38, and the interaction between respiratory phase and attention were significant for P2 amplitude, $F(1, 75) = 5.23$, $p = .025$, $\eta_p^2 = 0.07$, BF_{10} = 1.71. P2 amplitude was larger when participants ignored the tone than when they attended to it, and larger during exhalation than during inhalation when participants ignored the tone. However, the respiratory phase's main effect was nonsignificant, $F(1, 75) = 0.85$, $p = .359$, $\eta_p^2 = 0.01$, BF_{10} = 1/1.86.

5. General Discussion

This study investigated whether cardiac and respiratory phases influence

auditory processing. Pure tones were presented with randomized ISIs. In Experiment 1, we asked the participants to ignore the tones, whereas in Experiment 2, we asked them to respond to the tones by pressing buttons as quickly as possible. We observed no significant effects of cardiac phases on N1 and P2 amplitudes across the two experiments. However, a two-way mixed repeated-measures ANOVA with cardiac phase as a within-subject factor and attention (ignored, attended) as a between-subject factor revealed that P2 amplitude was larger during diastole than systole and was greater when participants ignored the tones than when they attended to them. The results of two-tailed *t*-tests and two-way ANOVAs on respiratory phases indicated that P2 amplitude was larger during exhalation than inhalation, but only when participants ignored the tones. We found no differences in N1 amplitude between respiratory phases. These findings suggest that visceral afferent signals can modulate auditory processing.

5.1 ERPs in Cardiac Phases

Li et al. (2024) did not observe N1 and P2 components in the difference waveforms calculated by subtracting waveforms elicited by 60 dB standard tones from those elicited by 80 dB standard tones. Li et al. (2024) attributed the lack of clear N1 and P2 components to the short ISIs of 500 ms. The present study used longer ISIs of 2,770–3,370 ms to address this issue. This study's N1 and P2 components align with typical latency and frontocentral scalp distribution (Crowley & Colrain, 2004; Näätänen & Picton, 1987). Moreover, the N1 and P2 amplitudes in both experiments were larger than those reported by Li et al. (2024), consistent with Pereira et al. (2014), who reported that longer ISIs elicited larger N1 and P2 components. Therefore, the experimental setup used in this study was appropriate for observing N1 and P2 components.

We found no significant differences in N1 and P2 amplitudes between cardiac phases when Experiments 1 and 2 were analyzed separately. However, the omnibus analysis revealed that P2 amplitudes were significantly larger during diastole than systole, albeit with a small effect size ($d_z = 0.26$). This finding suggests that cardiac afferent signals may have only a minor impact on auditory processing. Although Schulz et al. (2020) reported that P2 amplitudes elicited by startle tones were larger during systole, this may have been due to the influence of the CFA. Figures 3A and 5A show that ECG potentials differ between systole and diastole. If such differences are not accounted for, they could contaminate ERP amplitude differences. This study employed the subtraction method (Kimura, 2019; Li et al., 2024) to remove ECG-related differences, allowing us to identify the very small effect of the cardiac phase on ERP amplitudes.

5.2 ERPs in Respiratory Phases

The P2 amplitude was larger during exhalation than inhalation when participants were asked to ignore auditory stimuli. This finding suggests that greater cognitive and attentional resources were allocated to the tone during exhalation because the P2 component reflects later stages of stimulus processing that involve cognitive demands (Schulz et al., 2020). However, this effect disappeared when participants were required to respond to the tone as quickly as possible. One possible explanation for these findings is that other factors may overshadow the effect of respiratory phases on auditory processing. Mizuhara and Nittono (2022, 2023) explored the relationship between respiratory phases and visual emotional stimuli perception. They reported that the discrimination accuracy for identifying fearful facial expressions over neutral ones was higher during inhalation than exhalation when stimuli were presented automatically

(Mizuhara & Nittono, 2023). However, this effect vanished when participants controlled the timing of stimulus presentation to coincide with the onset of inhalation or exhalation by pressing a button (Mizuhara & Nittono, 2022). Mizuhara and Nittono suggested that the effect of respiratory phases on emotional discrimination was weak and easily masked by overlapping motor processing. Similarly, the current study suggests that while respiratory phases modulate auditory processing, the demands of motor responses can easily mask this modulation.

In contrast, neither experiment observed significant differences in N1 amplitudes between respiratory phases. This finding contrasts with Waselius et al. (2022), who reported that the N1 amplitude elicited by a tone was larger during exhalation than inhalation. The N1 component reflects stimulus characteristics or general pre-attentional and affective states (Schulz et al., 2020). One possible explanation for the null results observed in this study is the ISI. Waselius et al. (2022) presented tone stimuli with randomized intervals of 20–40 seconds, whereas Schulz et al. (2020) used randomized intervals of 8–12 seconds and reported larger N1 amplitudes during diastole than systole. In contrast, the present study used randomized intervals of approximately 3 seconds, shorter than previous studies. As a result, this may have potentially led to the absence of a significant difference in N1 amplitudes between respiratory phases.

P2 amplitudes were significantly larger when participants ignored the tones than when they responded to them. Typically, ERP amplitudes are larger for attended than for unattended stimuli. However, this pattern may differ within the paradigm used in this study (i.e., ignored vs. attended). Schulz et al. (2020) also reported that P2 amplitudes were tended to be smaller when participants were asked to respond to tones

than when they were asked to ignore them. The result of the current study is consistent with this. Salisbury et al. (2001) reported that the P3 amplitude elicited by oddball tones was smaller when participants responded to the tones than when they silently counted them. They speculated that movement-related artifacts could have contributed to this reduction. In the present study, the P2 amplitude was possibly attenuated by similar artifacts. Another possibility is that the P2 amplitude was apparently reduced due to the overlap of N2 and P3 waves elicited in the attended condition (Näätänen, 1992). A careful comparison of the ERP waveforms in the ignored (Experiment 1) and attended (Experiment 2) conditions shows that the P2 wave in the attended condition was followed by a negative deflection (200–300 ms) crossing the baseline and a positive deflection (300–400 ms), which were less obvious in the passive condition.

5.3 Effects of Cardiac/Respiratory Phases on Motor Responses

This study found no significant differences in RTs or omission error rates between cardiac and respiratory phases, suggesting that these cyclic autonomic nervous activities do not influence motor responses. These results are consistent with previous studies (Gallego et al., 1991; Thompson & Botwinick, 1970). However, other studies have reported conflicting findings (e.g., Beh & Nix-James, 1974; Buchsbaum & Callaway, 1965; Muñoz-Caracuel et al., 2024; Schulz et al., 2020). For instance, Beh and Nix-James (1974) found that responses to tones were significantly faster during inhalation than exhalation, whereas Buchsbaum and Callaway (1965) and Muñoz-Caracuel et al. (2024) observed the opposite effect. These discrepancies may be due to differences in diaphragm muscle contraction, relaxation, and afferent signals from pulmonary stretch receptors. Nevertheless, the conflicting findings suggest minimal effects of cardiac and respiratory phases on motor responses to stimuli.

6. Conclusions

This study provided two key findings. Firstly, the P2 amplitude elicited by tones is larger during diastole than systole, albeit with a small effect size. Secondly, when participants ignore tones, the P2 amplitude is larger during exhalation than inhalation. However, this effect disappeared when participants attended to tones. These findings suggest that visceral afferent signals modulate auditory processing. This study merely focused on cardiac activity and respiration. However, it is possible that various interoceptive signals are generated within the body, and their complex interactions influence the perception of external events. Further research is needed to develop a more comprehensive model of the association between auditory processing and internal bodily signals.

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Tables and Table captions

Table 1

Mean Number of Averaged Trials in Each Experiment's ERP Calculations

			Number of averaged trials		
Experiment	**Phase**	**Stimulus**	**Mean**	***SD***	**Range**
Experiment 1	Systole	Real	216	30	155–288
		Silent	216	27	164–278
	Diastole	Real	217	27	162–272
		Silent	214	31	152–310
	Inhalation	Real	354	59	234–576
		Silent	362	49	230–465
	Exhalation	Real	556	67	373–667
		Silent	548	62	401–665
Experiment 2	Systole	Real	220	27	171–296
		Silent	220	31	157–282

Diastole	Real	220	35	157–302
	Silent	225	31	173–290
Inhalation	Real	317	103	173–560
	Silent	371	118	204–635
Exhalation	Real	589	117	358–819
	Silent	545	113	327–746

Table 2

Each Cardiac and Respiratory Phase's Mean N1 and P2 Amplitudes

Experiment	ERP components	Phase	Mean (*SD*)	*t*	*p*	d_z	Stimulus	Mean (*SD*)
Experiment 1	N1 difference	Systole	−3.11 (1.81)	1.27	.211	0.20	Real	−3.00 (1.82)
							Silent	0.11 (0.78)
		Diastole	−3.30 (2.10)				Real	−3.45 (2.13)
							Silent	−0.16 (0.52)
		Inhalation	−3.27 (1.90)	0.03	.975	0.01	Real	−3.32 (1.87)
							Silent	−0.05 (0.33)
		Exhalation	−3.26 (1.92)				Real	−3.27 (1.86)
							Silent	−0.00 (0.33)
	P2 difference	Systole	2.76 (1.72)	1.84	.074	0.29	Real	3.08 (1.97)
							Silent	0.32 (0.96)
		Diastole	3.01 (1.74)				Real	2.60 (1.48)
							Silent	−0.41 (0.71)

		Inhalation	2.70 (1.75)	2.20	.034*	0.35	Real	2.70 (1.63)
							Silent	0.01 (0.40)
		Exhalation	2.94 (1.72)				Real	2.89 (1.69)
							Silent	−0.06 (0.24)
Experiment 2	N1 difference	Systole	−2.93 (1.75)	0.63	.533	0.11	Real	−2.58 (1.57)
							Silent	0.35 (0.68)
		Diastole	−2.82 (1.87)				Real	−3.02 (1.75)
							Silent	−0.21 (0.50)
		Inhalation	−2.81 (1.47)	1.84	.075	0.30	Real	−2.85 (1.47)
							Silent	−0.04 (0.35)
		Exhalation	−3.01 (1.64)				Real	−3.03 (1.55)
							Silent	−0.02 (0.28)
	P2 difference	Systole	1.17 (1.34)	1.40	.171	0.23	Real	1.82 (1.30)
							Silent	0.65 (0.86)
		Diastole	1.45 (1.20)				Real	1.12 (1.26)

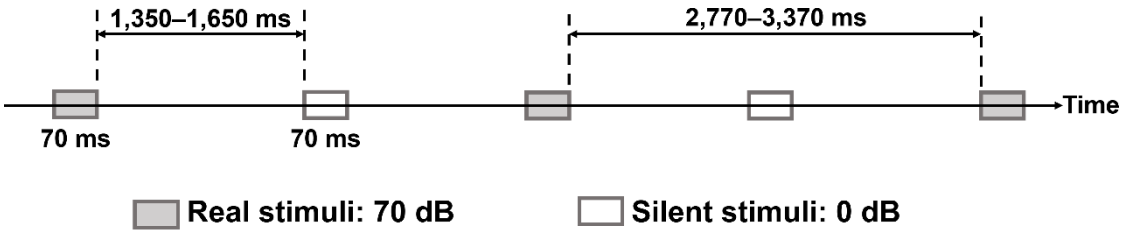
						Silent	−0.32 (0.71)
	Inhalation	1.30 (1.32)	1.01	.321	0.17	Real	1.23 (1.23)
						Silent	−0.07 (0.44)
	Exhalation	1.19 (1.17)				Real	1.21 (1.08)
						Silent	0.02 (0.28)

* $p < .05$

Figures and Figure Captions

Figure 1

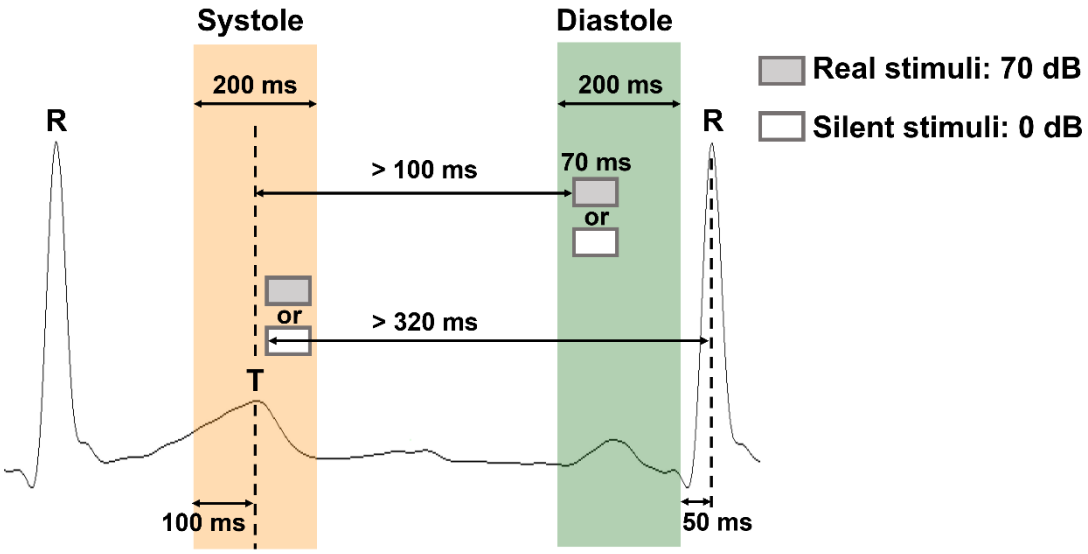
Example of Auditory Stimuli Sequence in Experiments 1 and 2



Note. We alternately presented real (70 dB) and silent stimuli (0 dB). The interstimulus interval (ISIs) between stimuli were randomly generated and ranged from 1,350 to 1,650 ms. Therefore, the ISIs between consecutive real stimuli ranged from 2,770 to 3,370 ms.

Figure 2

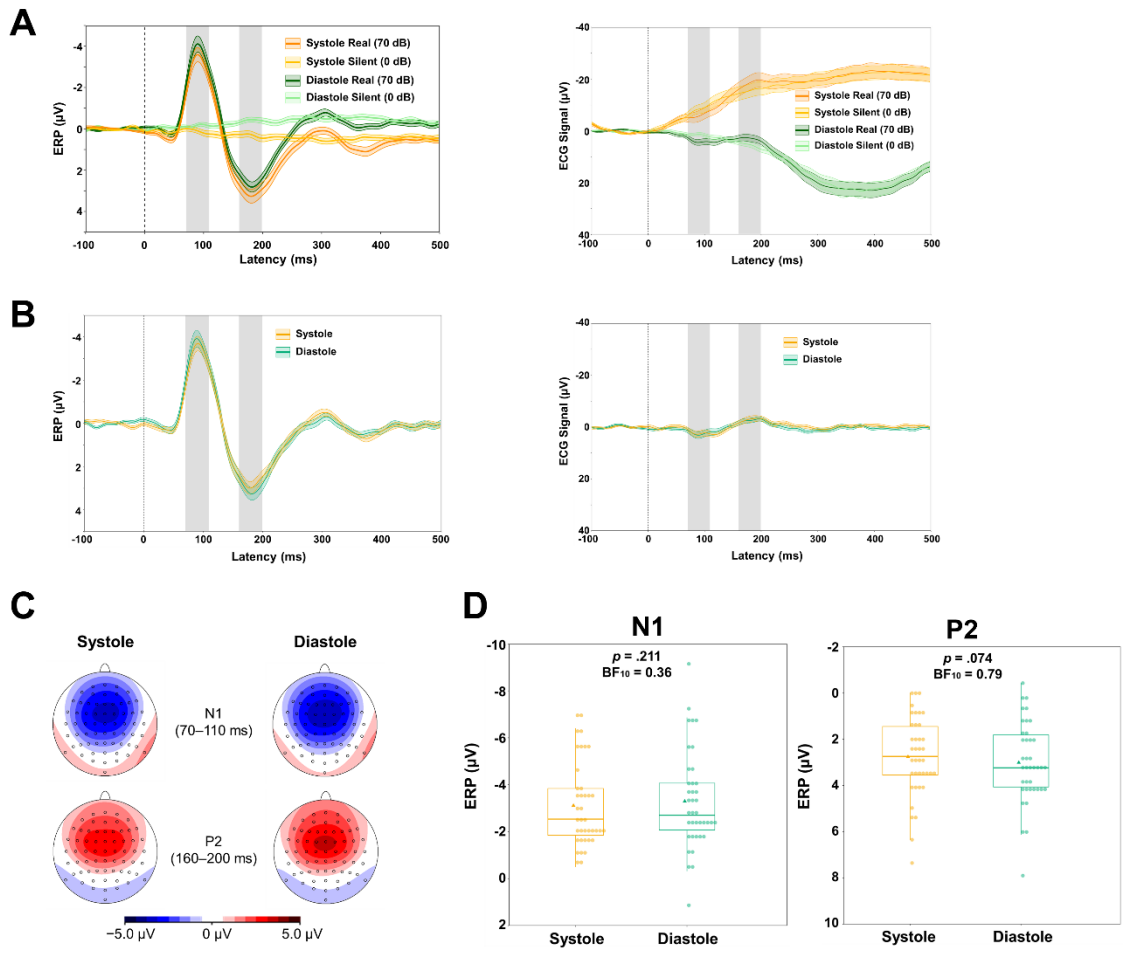
Illustrations of Defined Systole and Diastole Phases in Experiments 1 and 2



Note. We defined systole as the period from 100 ms before to 100 ms after the T-wave peak and diastole as the period from 250 ms to 50 ms before the R-wave peak. We categorized stimuli into systole or diastole phases based on their timing relative to these cardiac events. Systole trials met the following criteria for the timing of stimulus onset: (1) within 100 ms before or after the T-wave peak, and (2) the interval to the next R-wave peak was greater than 320 ms. Diastole trials met the following criteria for the timing of stimulus onset: (1) within 250 ms to 50 ms before the R-wave peak, and (2) the interval to the preceding T-wave peak was greater than 100 ms.

Figure 3

Grand Mean Waveforms for Cardiac Phase Analyses in Experiment 1

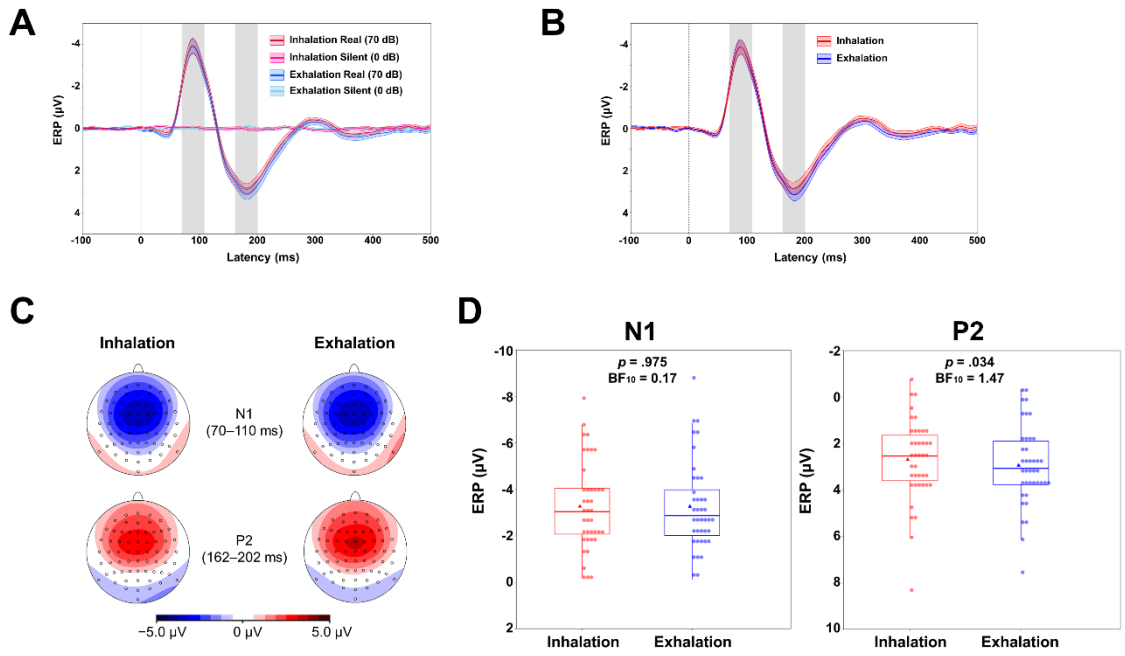


Note. (A) The left panel displays the grand mean ERP waveforms and standard errors from the frontocentral channel group (F1/2, Fz, FC1/2, FCz, C1/2, and Cz) in response to real (70 dB) and silent (0 dB) stimuli across different cardiac phases ($N = 39$). The right panel shows ECG waveforms recorded from the left lower rib using the same bandpass filter settings as in the EEG data analysis. The gray-shaded areas (70–110 ms and 160–200 ms) represent the N1 and P2 latency windows, respectively. (B) The left panel illustrates the grand mean difference waveforms and their standard errors, measured at the frontocentral site for each cardiac phase. The right panel shows residual

ECG-related waveforms calculated by subtracting waveforms elicited by silent stimuli from those elicited by real stimuli. (C) Topographic maps of the N1 and P2 latency windows for each cardiac phase. (D) Mean amplitudes of ERPs within the designated time windows for each cardiac phase. The dots represent individual participants' data, whereas the triangles represent the mean amplitudes.

Figure 4

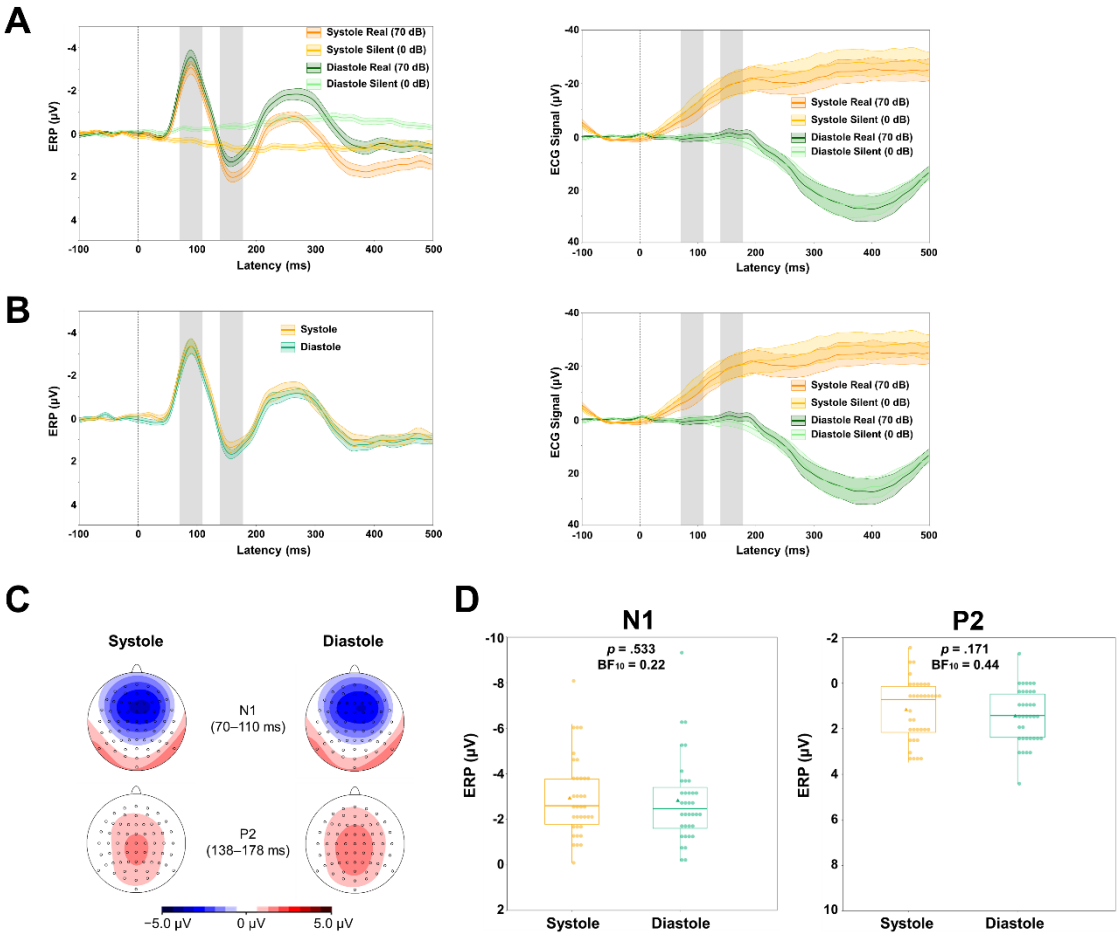
Grand Mean Waveforms for Respiratory Phase Analyses in Experiment 1



Note. (A) Grand mean ERP waveforms with standard errors from the frontocentral site elicited by real (70 dB) and silent stimuli (0 dB) during inhalation and exhalation (N = 40). The N1 and P2 latency periods (70–110 ms and 162–202 ms) are indicated for each phase, highlighted by shaded regions. (B) We calculated Grand mean difference waveforms with standard errors at the frontocentral site for each respiratory phase by subtracting ERP waveforms elicited by silent stimuli from those elicited by real stimuli. (C) Topographic maps of the N1 and P2 latency windows for each respiratory phase. (D) Mean ERP amplitudes within the designated time windows for each respiratory phase. The dots represent individual participants' data, whereas the triangles represent the mean amplitudes.

Figure 5

Grand Mean Waveforms for Cardiac Phase Analyses in Experiment 2

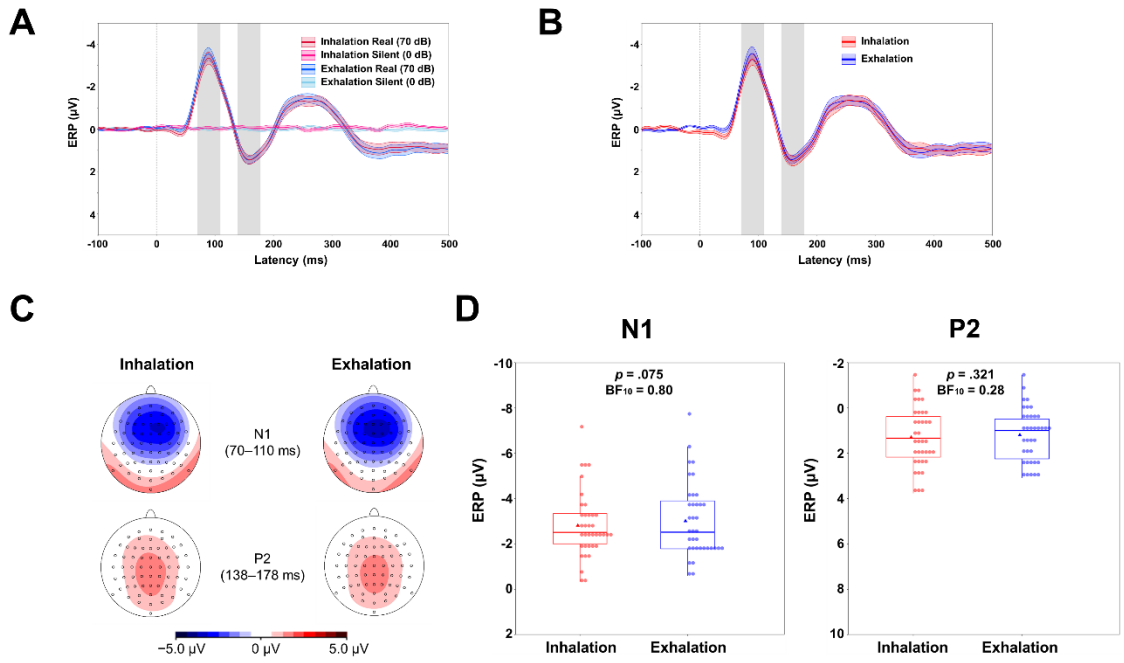


Note. (A) The left panel displays grand mean ERP waveforms with standard errors at the frontocentral site, elicited by real (70 dB) and silent (0 dB) stimuli during systole and diastole ($N = 36$). The right panel shows ECG waveforms recorded from the left lower rib. Gray-shaded areas (70–110 ms and 138–178 ms) indicate the N1 and P2 latency windows, respectively. (B) The left panel presents grand mean difference waveforms with standard errors at the frontocentral site for each cardiac phase. The right panel displays residual ECG-related waveforms calculated by subtracting waveforms elicited by silent stimuli from those elicited by real stimuli. (C) Topographic

maps of the N1 and P2 time windows for each cardiac phase. (D) Mean ERP amplitudes in the designated time windows for each cardiac phase. The dots represent individual participants' data, whereas the triangles represent the mean amplitudes.

Figure 6

Grand Mean Waveforms for Respiratory Phase Analyses in Experiment 2



Note. (A) Grand mean ERP waveforms with standard errors at the frontocentral site, elicited by real stimuli (70 dB) and silent stimuli (0 dB) during inhalation and exhalation ($N = 37$). Gray-shaded areas (70–110 ms and 138–178 ms) indicate the N1 and P2 time windows, respectively. (B) We calculated Grand mean difference waveforms with standard errors at the frontocentral site for each respiratory phase by subtracting ERP waveforms elicited by silent stimuli from those elicited by real stimuli. (C) Topographic maps of the N1 and P2 latency windows for each respiratory phase. (D) Mean ERP amplitudes in the designated time windows for each respiratory phase. The dots represent individual participants' data, whereas triangles represent the mean amplitudes.