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Binaural Background Noise Enhances Neuromagnetic Responses from Auditory Cortex

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Abstract: The presence of binaural low-level background noise has been shown to enhance the transient evoked N1 response at about 100 ms after sound onset. This increase in N1 amplitude is thought to reflect noise-mediated efferent feedback facilitation from the auditory cortex to lower auditory centers. To test this hypothesis, we recorded auditory-evoked fields using magnetoencephalography while participants were presented with binaural harmonic complex tones embedded in binaural or monaural background noise at signal-to-noise ratios of 25 dB (low noise) or 5 dB (higher noise). Half of the stimuli contained a gap in the middle of the sound. The source activities were measured in bilateral auditory cortices. The onset and gap N1 response increased with low binaural noise, but high binaural and low monaural noise did not affect the N1 amplitudes. P1 and P2 onset and gap responses were consistently attenuated by background noise, and noise level and binaural/monaural presentation showed distinct effects. Moreover, the evoked gamma synchronization was also reduced by background noise, and it showed a lateralized reduction for monaural noise. The effects of noise on the N1 amplitude follow a bell-shaped characteristic that could reflect an optimal representation of acoustic information for transient events embedded in noise.

Keywords: N1; P1; P2; gamma synchronization; efferent feedback; signal-to-noise ratio; gap detection

1. Introduction

Auditory perception is determined by the ratio between the stimulus and noise levels rather than the absolute stimulus intensity [1–3]. Background noise affects auditory perception dramatically and is commonly associated with reduced amplitudes and prolonged auditory-evoked responses [4–6]. However, researchers have shown that the hearing threshold decreased when adding an appropriate amount of noise to the target sound, although the improvement is small (1.4–1.7 dB) in normal-hearing participants [7].

Previous work about the effect of background noise on auditory-evoked responses focused on the N1 wave, the negative potential at about 100 ms after sound onset in the human electroencephalogram (EEG) and its equivalent in the magnetoencephalogram (MEG). N1 is elicited by an acoustic change in the auditory environment and may be functionally related to the readout of sensory registration and the update of an auditory memory before perception [8]. The N1 amplitude depends on the physical stimulus parameters, and background noise typically diminishes its amplitude [9]. However, a low-level background noise paradoxically yields an increase in the N1 amplitude when both the stimuli and noise were presented binaurally in MEG [10–12] and EEG studies [13–15]. In the study of Papesh, Billings and Baltzell [14], the noise-related increased in N1 amplitude recorded using EEG was more pronounced in binaural than monaural noise conditions.

Speech-in-noise intelligibility decreases when noise is presented at the same ear as the speech sound. However, some release from masking occurs when adding noise to the



Citation: Shen, D.; Alain, C.; Ross, B. Binaural Background Noise Enhances Neuromagnetic Responses from Auditory Cortex. *Symmetry* **2021**, *13*, 1748. https://doi.org/10.3390/ sym13091748

Academic Editor: Francisco José Germain Martínez

Received: 9 July 2021 Accepted: 20 August 2021 Published: 19 September 2021

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Copyright: © 2021 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (https:// creativecommons.org/licenses/by/ 4.0/). contralateral ear [16]. The activation of olivocochlear efferents [17] has been proposed as a neural mechanism underlying the release from ipsilateral masking. In addition to the olivocochlear efferent system, central auditory pathways may contribute to the masking asymmetry. Prior research revealed interactions between neural responses to a stimulus and contralateral noise along the central auditory pathway [18,19]. The present study aims to further clarify whether the noise-related enhancement reported in prior studies depends on the presence of binaural noise or whether monaural noise could also increase the transient evoked response by using MEG. Binaural auditory stimuli were presented while concurrent noise was either binaurally or monaurally presented. An N1 amplitude increase with binaural noise only would be in favor of the olivocochlear efferent hypothesis.

The present study also aimed to assess whether the observed N1 changes could be related to the impact of noise on earlier evoked responses. For instance, Alain, Quan, McDonald and Van Roon [11] revealed that the P1 amplitude, which appears in the EEG as a positive peak at about 50 ms after sound onset, decreased with increasing binaural noise. The P1 suppression could indicate preattentional sensory gating [20–22], which is essential in protecting the integrity of higher-order functions [23–26]. Here, we examine whether concurrent noise would show a sensory-gating-like modulation of the P1 and explore possible associations between P1 and N1 changes.

In addition to the sensation threshold, noise impacts perception. For example, gap detection thresholds, which measure auditory temporal acuity, are elevated in the presence of concurrent noise [27,28]. Cortical responses to stimulus gaps appear as P1-N1-P2 responses elicited by the trailing marker after the gap [29]. It remains unclear whether a low-level noise enhances the N1 gap response similar to the N1 onset response. Therefore, in the present study, stimuli with or without a gap were used to examine whether a noise-induced increase in the N1 onset response would generalize to correlates of temporal processing.

The evoked responses are accompanied by event-related changes in the spectral power of the MEG or EEG. Possibly, an association exists between noise-induced modulation of sensory processing and event-related changes in gamma oscillation. For example, gamma activity was suppressed in sensory gating [30–32]. Thus, the examination of gamma activities may help understand how the manipulation of background noise affects the N1 amplitude. Additionally, the effects of background noise on N1 might extend to P2. Alain, Quan, McDonald and Van Roon [11] showed that the P2 amplitude decreases with more extensive binaural background noise. Ross, et al. [19] presented target sound and noise separately to each ear and revealed reduced P2 amplitude with contralateral masking. The present study examines how monaural and binaural noise affects the P2 amplitude to binaural stimuli.

To further characterize the mechanism of noise-induced increase in sensory-evoked responses, we examined how noise presentation (binaural or monaural), the level of noise intensity (high, low, or no), and the type of stimuli (with or without a gap) affected auditory-evoked P1-N1-P2 responses and event-related spectral modulations (gamma oscillations). We hypothesized that monaural noise would reduce contralateral P1 but would not affect ipsilateral P1 since the P1 reflects the early cortical registration of sound onset. However, binaural noise was expected to reduce the P1 in both hemispheres. Based on previous EEG [14] and MEG studies [10–12], only the low-level binaural noise should increase the N1 amplitude. The stimulus gap would not affect the onset P1 and N1 responses. However, the gap would affect the P2 since the P2 reflects a later pattern-recognition process [29]. Moreover, if the gamma activity is an early sensory response, the effects of noise type should be similar to those on the P1.

2. Materials and Methods

2.1. Participants

Eight men and seven women between 18 and 32 years of age (mean: 25.0 years; standard deviation, 3.7 years) participated in this study. They had normal hearing thresholds less or equal to 20 decibels (dB HL) at the octave frequencies between 250 Hz and 8000 Hz

as measured by pure tone audiometry. Participants provided written consent after receiving information about the study. The study protocol was approved by the Research Ethics Board of Baycrest Centre and the University of Toronto.

We calculated the optimal sample size using G*Power (version 3.1.9.7), expecting effect sizes and data variance similar to a previous study using similar methods [11]. Provided the effect size f = 0.25 (partial $\eta^2 = 0.06$), we estimated the sample size n = 14 for type-I error rate $\alpha = 0.05$ and power of the test $(1 - \beta) = 0.8$.

2.2. Stimuli

The stimuli were complex tones, composed of the first six harmonics of the fundamental frequency of 200 Hz at the sine phase and equal intensity. The stimuli of 95 ms duration included 5 ms linear onset and offset slopes to minimize spectral splatter. Half of the stimuli contained a 15 ms gap in the middle of the sound. The offset of the leading marker and onset of the trailing marker were smoothed with 5 ms linear slopes. We chose a 15 ms gap because such stimulus elicited a clear N1 gap response in a previous study [29]. The stimuli were synthesized using Adobe Audition 1.5 at the sampling rate of 44,100 Hz. Stimulus presentation was controlled by Presentation software (Version 13.0, Neurobehavioral Systems, Berkeley, CA, USA). The sounds were delivered via Etymotic ER3A insert earphones and reflectionless plastic tubes of length 2.5 m. The stimuli were presented binaurally at an intensity of 80 dB sound pressure level (SPL). The intensities were calibrated using a Larson Davis System 824 SPL meter.

The higher intensity of the broadband Gaussian noise was 75 dB SPL, equivalent to the SNR of 5 dB, which characterizes a challenging listening situation for speech-in-noise understanding. The low noise intensity of 55 dB SPL was equivalent to the SNR of 25 dB. Thus, a normal-hearing individual would completely understand speech at this SNR despite the considerable noise. For convenience, we termed the two noise levels 'low' and 'higher' noise.

2.3. Procedure

Participants were presented with five experimental conditions: The binaural stimuli were presented without noise or with high or low binaural or monaural noise. The monaural noise was presented to the left ear. Binaural noise was identical in both ears. For each condition, a sequence of 300 stimuli was presented with an inter-stimulus interval uniformly randomized in 100 ms steps between 800 ms and 1200 ms, lasting about five minutes. Within each sequence, 150 stimuli with a gap and 150 without were presented in random order. Presentation of noise started before the onset of the sequence and was held constant throughout stimulus presentation. Each experimental condition was presented twice. Thus, each participant was presented with ten sequences (i.e., block) of stimuli. The Latin-square design was used for balancing the condition order. The participants watched a subtitled muted movie of their own choice during the auditory presentation and were instructed to ignore the stimuli and background noise. No specific task was required for the participants.

2.4. MEG Data Acquisition and Analysis

The MEG was recorded in a magnetically shielded room using a helmet-shaped 151-channel whole cortex neuro-magnetometer (OMEGA, CTF MEG, Coquitlam, BC, Canada). Participants sat in an upright position with their heads touching the top of the MEG helmet and were asked to minimize movement during recordings. The head position within the MEG was recorded before and after each block using head-indicator coils attached to the nasion and left and right preauricular points. Head movement was identified by a mismatch between both localizations of the fiducials. A block of MEG recording was repeated if the difference for any fiducial and any direction exceeded 5 mm. In nine participants, one block was repeated, in one participant, two blocks, and another participant three blocks. The MEG was lowpass filtered at 200 Hz and sampled at a rate of 625 Hz.

The Brainstorm toolbox [33] was used to analyze the MEG data. For each participant, the head shape was digitized using a 3-D digitizer (Polhemus Fastrak) before the MEG acquisition. A pseudo-individual anatomy was created by warping the MRI template into the head shape using the Colin 27 MRI template as the default anatomy. Artifact components accounting for eye blinks, eye movements, heartbeats, and muscle activity were identified with independent component analysis [34] and subtracted from the data. The MEG data were parsed into epochs, including 100 ms of prestimulus baseline and 500 ms of poststimulus activity. For each participant, sensor data were averaged separately in each of the five noise conditions.

Dynamical statistical parametric mapping (dSPM) [35] was used to estimate the sources underlying the MEG activity. The dSPM provides a statistical map of z-scores. It is a linear measure and can be expressed as imaging kernels. This measure has been recommended to estimate sources of an average response and normalize the participant average before a group analysis [33,36]. Two scouts (MNI coordinates, left: -42, -30, 8.8; right: 49.4, -28, 13.1) were created to display source activities based on the strongest activities. Each scout included 20 vertices. P1, N1, P2 peak amplitudes and latencies were examined. The measurement intervals were 40–60 ms for P1, 80–150 ms for N1, and 150–250 ms for P2, respectively. To further examine the gap responses, we subtracted the responses in the no-gap condition from those in the gap condition. The latency intervals for the gap responses were 95–115 ms for P1, 135–175 ms for N1, and 180–280 ms for P2.

We used a Morlet wavelet for time-frequency transform, which was defined by three cycles of sine and cosine functions within the full-width half-maximum of the Gaussian envelope [37]. The increase and decrease in spectral power compared to a baseline interval were called event-related synchronization (ERS) and event-related desynchronization (ERD), respectively [38,39]. Gamma (35–45 Hz) ERS was defined as the mean activity during the 50 ms to 100 ms interval.

2.5. Experimental Design and Statistical Analysis

We used a two × five factorial design with hemisphere (left, right) and stimulus condition (five levels: high binaural noise, high monaural noise, low binaural noise, low monaural noise, no-noise) as within-subjects factors. We examined the effects of hemispheres and noise type on P1, N1, and P2 sound onset and gap responses in auditory cortex source activity and gamma oscillation. The effects of noise on the evoked responses were further analyzed by subtracting the responses in the no-noise condition from those recorded in the monaural and binaural noise conditions and three-way ANOVAs (noise intensity: higher or low, noise laterality: binaural and monaural, hemisphere: left and right) were conducted. The Greenhouse–Geisser method was used to correct for inhomogeneity of the variance, if applicable. However, the nominal degrees of freedom were reported for the F-tests. SPSS (IBM, version 26) was employed to analyze data.

3. Results

3.1. Auditory-Evoked Responses

The butterfly plot of all MEG sensor signals in Figure 1A provides an overview of the time course of the auditory-evoked responses elicited by the stimulus onset under the no-noise condition. The corresponding topographic map of the magnetic field at the time of the N1 maximum revealed the dipolar patterns above the left and right temporal lobes (Figure 1B; MNI coordinates, left: -42, -30, 8.8; right: 49.4, -28, 13.1). The left and right auditory cortex waveforms (Figure 2A) exhibited prominent P1, N1, and P2 waves with peak latencies of about 50 ms, 100 ms, and 200 ms after sound onset.

The sound onset after the 15 ms gap in the stimulus elicited event-related fields, referred to as the gap response. The gap response showed a tri-phasic P1-N1-P2 response like the onset response, however, with smaller amplitudes (Figure 2B). The results of two-way ANOVAs on onset and gap P1-N1-P2 amplitudes and latencies are shown in Table 1, and the group means of peak amplitudes in Figure 3.



Figure 1. Auditory-evoked magnetic fields (AEF) in the no-noise condition, (**A**) butterfly plot of AEF time series from all MEG sensors, (**B**) 2-D magnetic field map at the N1 peak latency.



Figure 2. Source activities from bilateral superior temporal gyri for (**A**) onset responses (both stimuli with and without gap), (**B**) gap responses (difference between the gap and no-gap responses). hb: high binaural noise, hm: high monaural noise, lb: low binaural noise, lm: low monaural noise, no: no-noise.

		C		Error	Amplitude			Latency		
		Source	ce df	df	F	р	η_p^2	F	р	η_p^2
Onset Responses	P1	Н	1	14	1.624	0.223	0.104	6.037	0.028	0.301
		Ν	4	56	62.575	< 0.001	0.817	1.280	0.289	0.084
		$\mathrm{H} \times \mathrm{N}$	4	56	3.269	0.018	0.189	2.117	0.091	0.131
	N1	Н	1	14	27.353	< 0.001	0.661	6.811	0.021	0.327
		Ν	4	56	5.481	0.001	0.281	13.272	< 0.001	0.487
		$H \times N$	4	56	1.663	0.171	0.106	1.341	0.266	0.087
	P2	Н	1	14	4.050	0.064	0.224	6.171	0.026	0.306
		Ν	4	56	5.622	0.001	0.287	2.570	0.048	0.155
		$H \times N$	4	56	6.237	< 0.001	0.308	0.884	0.479	0.059
Gap Responses	P1	Н	1	14	4.920	0.044	0.260	10.010	0.007	0.417
		N	4	56	5.151	0.001	0.269	8.523	< 0.001	0.378
		$H \times N$	4	56	1.246	0.302	0.082	1.261	0.296	0.083
	N1	Н	1	14	1.739	0.208	0.111	3.620	0.078	0.205
		N	4	56	4.116	0.005	0.227	20.250	< 0.001	0.592
		$\mathbf{H}\times\mathbf{N}$	4	56	0.970	0.431	0.065	6.110	< 0.001	0.304
	P2	Н	1	14	7.287	0.017	0.342	1.699	0.213	0.108
		Ν	4	56	17.427	< 0.001	0.555	25.131	< 0.001	0.642
		$\mathrm{H} imes \mathrm{N}$	4	56	2.438	0.058	0.148	0.696	0.598	0.047

Table 1. Two-way ANOVA (hemisphere \times noise type) for P1, N1, and P2 peak amplitudes and latencies.

H = hemisphere; N = noise; H \times N = hemisphere \times noise type.

3.1.1. P1 Onset Response

Figure 4A shows the group mean P1 amplitude after subtracting the brain responses elicited during the no-noise condition from those obtained in each noise condition. Negative values reflect noise-related amplitude attenuation, whereas positive values indicate noise-related facilitation of the response amplitude.

The three-way ANOVA (noise intensity × noise laterality × hemisphere) revealed the main effects of noise intensity (*F* (1, 14) = 79.750, *p* < 0.001, η_p^2 = 0.851) and of noise laterality (*F* (1, 14) = 71.218, *p* < 0.001, η_p^2 = 0.836). The interactions between noise intensity and noise laterality (*F* (1, 14) = 22.577, *p* < 0.001, η_p^2 = 0.617) and between noise laterality and hemisphere (*F* (1, 14) = 5.012, *p* = 0.042, η_p^2 = 0.264) reached significance. Generally, P1 amplitude reduction was larger when the noise was presented binaurally than monaurally; and this reduction was more significant when the noise intensity was higher than low and more significant in the left than in the right hemisphere.

3.1.2. N1 Onset Response

The three-way ANOVA on the N1 amplitude (Figure 4B) revealed the main effects of noise intensity (F(1, 14) = 11.852, p = 0.004, $\eta_p^2 = 0.458$), and of noise laterality (F(1, 14) = 6.146, p = 0.027, $\eta_p^2 = 0.305$). Generally, N1 amplitude reduction was larger when the noise intensity was higher than it was low. N1 suppression was also more pronounced when the noise was presented monaurally than binaurally in low-intensity noise (p = 0.003) but not in higher-intensity noise (p = 0.804). Importantly, the N1 amplitude increased in the low binaural noise condition.



Figure 3. The effects of background noise on the mean values of (**A**) P1 amplitude, (**B**) N1 amplitude, and (**C**) P2 amplitude. * p < 0.10, ** p < 0.05, *** p < 0.01. Error bars represent +1 standard error.



Figure 4. Group mean amplitude after subtracting brain responses elicited in the no-noise condition from those obtained in each noise condition. Negative values reflect amplitude attenuation, whereas positive values indicate noise-related enhancement in response amplitude. (**A**) P1 amplitude, (**B**) N1 amplitude, and (**C**) P2 amplitude. * p < 0.10, ** p < 0.05, *** p < 0.01, **** p < 0.001. Error bars represent +1 standard error.

3.1.3. P2 Onset Response

The three-way ANOVA on P2 amplitudes (Figure 4C) revealed the main effects of noise laterality (*F* (1, 14) = 10.863, *p* = 0.005, $\eta_p^2 = 0.437$) and of hemisphere (*F* (1, 14) = 5.094, p = 0.041, $\eta_p^2 = 0.267$), and a three-way interaction between noise intensity, noise laterality, and hemisphere (*F* (1, 14) = 5.993, p = 0.028, $\eta_p^2 = 0.300$). In the right hemisphere, the P2 amplitude reduction was larger when the noise was presented binaurally than monaurally (*p* < 0.001). In the left hemisphere, there was a two-way interaction between noise intensity

and noise laterality (F(1, 14) = 11.764, p = 0.004, $\eta_p^2 = 0.457$). When the noise intensity was low, the P2 amplitude reduction was smaller when the noise was presented monaurally than binaurally (p = 0.039). However, when the noise intensity was higher, there was not such a difference (p = 0.274).

3.1.4. P1 Gap Response

The three-way ANOVA on P1 amplitudes (Figure 4A) revealed the main effects of noise intensity (F(1, 14) = 6.443, p = 0.024, $\eta_p^2 = 0.315$) and of noise laterality (F(1, 14) = 10.009, p = 0.007, $\eta_p^2 = 0.417$). The three-way interaction between noise intensity, noise laterality, and hemisphere (F(1, 14) = 11.744, p = 0.004, $\eta_p^2 = 0.456$) reached significance. In the right hemisphere, the P1 amplitude reduction was slightly larger when the noise was presented binaurally than monaurally (p = 0.069). In the left hemisphere, there was an interaction between noise intensity and noise laterality (F(1, 14) = 8.833, p = 0.010, $\eta_p^2 = 0.387$). When the noise intensity was low, the P1 amplitude reduction was larger when the noise presented binaurally than monaurally (p = 0.001); however, when the noise intensity was higher, there was not such a difference (p = 0.858).

3.1.5. N1 Gap Response

The three-way ANOVA on N1 amplitudes (Figure 4B) revealed a main effect of noise intensity (F(1, 14) = 6.222, p = 0.026, $\eta_p^2 = 0.308$) and a marginal interaction between noise intensity and hemisphere (F(1, 14) = 4.132, p = 0.061, $\eta_p^2 = 0.228$). Interestingly, background noise did not reduce the N1 amplitude; instead, the N1 amplitude increased. Generally, the increase of N1 amplitude was larger when the noise intensity was low than higher; this increase was more significant in the right (p = 0.025) than in the left hemisphere (p = 0.081).

3.1.6. P2 Gap Response

The ANOVA on P2 amplitude (Figure 4C) revealed the main effects of noise intensity (*F* (1, 14) = 23.285, *p* < 0.001, η_p^2 = 0.625) and of noise laterality (*F* (1, 14) = 27.609, *p* < 0.001, η_p^2 = 0.664), and a marginal effect of hemisphere (*F* (1, 14) = 3.810, *p* = 0.071, η_p^2 = 0.214). The interaction between noise intensity and hemisphere was significant (*F* (1, 14) = 4.992, *p* = 0.042, η_p^2 = 0.263). Generally, the P2 amplitude reduction was larger when the noise was presented binaurally than monaurally, and marginally larger in the right than in the left hemisphere; it was also larger when the noise intensity was higher than it was low, and this difference was more significant in the right (*p* = 0.001) than in the left hemisphere (*p* = 0.008).

3.2. Gamma (35-45 Hz) Synchronization

Stimuli in the no-noise conditions elicited transient gamma-band responses [40] in both hemispheres. However, gamma responses were attenuated in the presence of noise (Figure 5). Bar graphs of the grand-mean gamma amplitudes are shown in Figure 6, and the results of the two-way ANOVA are in Table 2.

The three-way ANOVA (noise intensity × noise laterality × hemisphere) on the gamma responses revealed the main effects of hemisphere (F(1, 14) = 5.819, p = 0.030, $\eta_p^2 = 0.294$), and a marginal effect of noise laterality (F(1, 14) = 3.145, p = 0.098, $\eta_p^2 = 0.183$). The interaction between noise laterality and hemisphere reached significance (F(1, 14) = 4.659, p = 0.049, $\eta_p^2 = 0.250$). Generally, the reduction of gamma responses was larger in the right than in the left hemisphere; it was also larger when the noise was presented binaurally than monaurally in the left (p = 0.049) but not in the right hemisphere (p = 0.956).



Figure 5. The effects of background noise on gamma synchronization after removing the evoked responses. hb: high binaural noise, hm: high monaural noise, lb: low binaural noise, lm: low monaural noise, no: no-noise.

Table 2.	Summary of	F ANOVA	(hemisphere ×	noise type)	on gamma	synchronization
14010 2.	Summary O		(incluspinete ×	noise type)	on gamma	synchronization

Source	df	Error df	F	p	η_p^2
Н	1	14	0.550	0.471	0.038
Ν	4	56	3.765	0.009	0.212
$\mathbf{H} imes \mathbf{N}$	4	56	3.200	0.020	0.186

H = hemisphere; N = noise type; H \times N = hemisphere \times noise type.



Figure 6. Group mean values of gamma synchronization (**A**) in the no-noise condition and the four noise conditions. (**B**) Group event-related synchronization (ERS) after subtracting brain responses elicited in the no-noise condition from those obtained in each noise condition. * p < 0.10, ** p < 0.05, *** p < 0.01. Error bars represent +1 standard error.

4. Discussion

The main findings of this study were that N1 amplitudes increased when stimuli were embedded in low-level binaural noise compared to stimuli presented in quiet conditions. The amplitude increases were observed for responses to the sound onset and gap responses. Monaural noise did not cause such amplitude increases. Moreover, increased response amplitudes in noise were specific to the N1 wave of the auditory-evoked responses. Background noise consistently attenuated P1 and P2 onset and gap responses, and the effects of the noise level and stimulation side showed distinct characteristics. The background noise also diminished the evoked gamma response, and the gamma power showed lateralized reduction for monaural noise.

4.1. Concurrent Noise Conditions

The auditory stimuli were presented binaurally under all experimental conditions. In the no-noise condition, the auditory signals were projected from both ears along predominantly contralateral auditory pathways to bilateral auditory cortices. Monaural noise to the left ear interfered peripherally with the left ear stimulus, resulting in a masked neural signal toward the contralateral right auditory cortex. In contrast, binaural noise affected the afferent projection along the bilateral auditory pathways. In addition, we assume effects of central interferences between noise and stimulus representations.

4.2. P1 Responses

The effects of continuous noise on the onset P1 showed reduced amplitudes with increasing background noise consistently with those observed in prior EEG and MEG studies [10–12,14,41]. We analyzed the early volley of the P1 wave, which showed an effect of contralaterality. Monaural left ear noise, aiming to suppress afferent projection to the right auditory cortex, reduced the P1 right but not left. The lateralized reduction of the P1 was similar to the low and higher level of monaural noise. Binaural noise reduced the P1 amplitude symmetrically, which was more substantial with a high noise level than low-level noise. The gap P1 was attenuated and delayed with binaural noise. However, a lateralized reduction as observed for the onset responses was not evident.

The interpretation of the functional significance of the P1 amplitude is challenging because multiple excitatory and inhibitory neural generators contribute to the extracranial signal [42]. The main P1 cortical sources are localized in or near the primary auditory cortex in the superior temporal gyrus [43–45]. The P1 is associated with the initial registration of the physical characteristics of a sound. For instance, the P1 is sensitive to tone frequency [46] and intensity [41,47]. However, the investigations on the P1 amplitude also indicate sensory gating modulated by the prefrontal cortex [44,48]. Similar to a paired stimulus paradigm [22,49], we interpret different P1 amplitudes elicited by the same stimulus as an indication of a sensory-gating-like modulation.

4.3. Evoked Gamma-Band Responses

Pantev, Makeig, Hoke, Galambos, Hampson, and Gallen [40] observed an eventrelated 40 Hz magnetic gamma-band response that originated in the superior temporal gyrus in the 20–130 ms interval after stimulus onset and suggested involvement in essential perceptual processing. The evoked gamma activity is thought to reflect the match of sensory information with memory contents [50] and feature binding [51–55]. Monaural noise to the left ear attenuated the right hemispheric gamma response but did not affect the evoked gamma activity in the left hemisphere. The gamma activity was almost absent in both binaural noise conditions. Thus, the contralateral dominance of the effect of noise was even more strongly expressed for the gamma-band response than the P1 response.

P1 and gamma-band responses appeared in overlapping time intervals. In previous studies, both types of response showed suppression in a paired stimulus paradigm, and therefore, both have been associated with sensory gating [30–32]. However, P1 and gamma activity exist in parallel and show different functional dependencies with age, hearing loss, and speech-in-noise understanding [56]. Additionally, the second onset after a 15 ms gap in the stimulus did not elicit a gamma activity, which is different from the P1. It further supported that the P1 and gamma responses are independent of each other [57,58].

4.4. N1 Responses

The N1 response was significantly more prominent in the right auditory cortex than the left, even though the stimuli were presented binaurally. Right hemispheric dominance for responses to simple auditory stimuli has been reported before [59–61]. The anatomical difference between left and right auditory cortices seems to contribute to the hemispheric asymmetry. A simulation study suggested that the larger left-hemispheric auditory cortex extends over multiple folded areas such that external electromagnetic fields could partly cancel each other. Therefore the right auditory source amplitudes may be up to 50% larger than in the left hemisphere [62].

In comparison to the generally larger N1 in the right hemisphere, the various noise conditions affected the N1 symmetrically. Specifically, the N1 increase with binaural noise was of the same order of magnitude from the left and right hemispheres. However, the earlier P1 and gamma responses showed asymmetries with monaural stimulation. These findings of different effects of noise on the N1 and the P1 suggest that these two responses index different aspects of auditory processing in agreement with previous studies [63,64].

The N1 amplitude was not attenuated by concurrent noise, although the SNR was strongly reduced, especially when presenting the high-level noise. In contrast, noise affected the earlier evoked responses. Therefore, we assume that a control mechanism exists that compensates for the loss in the SNR.

The finding that low binaural noise increased the N1 amplitude agrees with EEG studies [13–15] and previous MEG studies from our group [10–12], and this noise-induced enhancement could be related to efferent feedback connections between the auditory cortex and the thalamus, inferior colliculus, and auditory brainstem nuclei. Such efferent feedback would enhance the SNR in adverse listening situations. The present study showed that the noise-related increase in N1 occurs only with binaural noise. In contrast, monaural noise of the same intensity did not affect the N1 amplitude, consistent with EEG findings [14]. However, the effects of efferent feedback have been explicitly shown as improvements in detecting complex sounds in the presence of contralateral noise that resulted in the release from ipsilateral masking [16]. The new finding that only binaural noise enhanced the N1 amplitude seems consistent with efferent feedback connections to lower auditory centers. However, if the olivocochlear efferent is the reason for the N1 increase, it should first cause a similar effect on P1 amplitude. Since there was no noise-induced enhancement in the P1 amplitude, the efferent feedback is unlikely to be the reason for the noise-related increase in N1.

An alternative account is that the increased N1 amplitude may be a consequence of the effects of binaural noise on the earlier evoked P1 responses. P1 was substantially attenuated bilaterally with high-level binaural noise, while the N1 amplitude was not different from the no-noise condition. A smaller P1 amplitude could indicate reduced sensory gating, resulting in unreduced N1 amplitudes even though the concurrent noise impaired the afferent inputs bilaterally.

The findings that high-level binaural noise substantially attenuated the P1 but did not affect N1 can be viewed as an effect of reduced sensory gating in the case of noise. Sensory gating at an early stage of processing, as reflected by stronger P1 suppression, increased the SNR at the subsequent stage, allowing for clear detection of the target stimulus [63]. Such a control mechanism may be necessary for preserving the N1 amplitude under the high binaural noise condition. The increase in the N1 amplitude in the low binaural noise condition could have been caused by an 'over-initiation' of downregulating the sensory-gating mechanism at the earlier stage, which could explain the bell-shaped N1 amplitude characteristics as a function of the noise level.

The N1 amplitude was not affected by monaural noise and did not show an amplitude increase with low-level noise. One speculation is that in the case of left ear noise, the stimulus in the right ear was projected undisturbed along the afferent pathways and resulted in an unambiguous representation at the cortical level. Thus, no or less modulation of sensory gain was required for compensating a loss in the SNR. No N1 overshoot occurred because of the lesser effect of gain control. More research is necessary to understand the effects of peripheral and central interferences on sensory-gating mechanisms.

Energetic masking was used in the present study. Prior research revealed that masking with babble noise reduced N1 amplitude more than white noise [65,66]. One reason for the noise-induced N1 increase might be related to the weaker masking caused by the white noise, resulting in an 'over-initiation' at the sensory-gating stage. Namely, the human auditory system overestimates the masking effect caused by the low binaural white

noise. Accordingly, when noise only comes from one ear, the auditory system would not overestimate its impact.

Similar to the N1 amplitude elicited by the onset of stimuli, the N1 amplitude for the gap response was also larger in the low binaural noise condition than in other conditions. This means that the system can 'over-initiate' the sensory modulation mechanism more than once in a short-term period. Namely, low-level background noise could facilitate temporal segmentation of auditory events, facilitating sound object organization.

4.5. P2 Responses

P2 has been proposed to represent some aspects of higher-order perceptual processing [67–69]. The effects of noise on the P2 amplitude were mainly observed in the right hemisphere, with P2 amplitudes smaller in both binaural noise conditions than in the monaural and no-noise conditions. Only the amplitude in low binaural noise was smaller in the left hemisphere than in the low monaural condition. The gap P2 response amplitude was also smaller in the binaural than in monaural conditions for both hemispheres. This meant that, as the onset P2, the P2 amplitude elicited by the trailing gap marker was more reduced by the binaural noise than by the monaural noise.

Ross, Miyazaki, and Fujioka [19] showed that the central masking did reduce the P2 amplitude in the right hemisphere for right ear stimulation and contralateral noise. They proposed that the central masking affected auditory processing at the level of auditory object representation. In the present study, binaural noise reduced the P2 amplitude in both hemispheres more strongly than the monaural noise. However, the difference in the right hemisphere was more evident than in the left hemisphere. Besides the 'central masking' from the contralateral side, the ipsilateral noise also caused a 'peripheral masking' by interactions at the cochlea [19,70]. For binaural noise, the masking could be from both contralateral and ipsilateral sides. However, for the monaural noise to the left ear, the masking was mainly from one side (ipsilateral side for the left ear, or contralateral side for the right ear). Thus, the P2 amplitude was smaller in the binaural than in monaural noise conditions. Ipsilateral masking might also be more dominant since the difference in P2 amplitude between binaural and monaural noise conditions was larger in the right than in the left hemisphere.

Furthermore, the increase of N1 in the low binaural condition did not extend to the P2 amplitude. It appeared that there was no direct relationship between the increase of N1 and the results of P2 amplitude. This is consistent with our proposal that the noise-related increase in N1 amplitude reflects the outcome of an 'over-initiation' mechanism, and it did not change the perceptual processing.

5. Concluding Remarks

The P1 amplitude and gamma synchronization were both reduced with the increase of noise intensity. This means that the sensory modulation was initiated to suppress the noise at the early stage. The low binaural noise generated larger N1 amplitude, but other types of noise did not have the same effect as the low binaural noise did. Our findings extend those of earlier studies by showing that a noise-related increase in N1 amplitude takes place only under binaural noise conditions. This situation could cause 'over-initiation' of the inhibition mechanism at the earlier stage and result in a stronger involuntary switch of attention to the stimulus at a subsequent stage, and larger N1 amplitude was apparent. Of course, there might be other mechanisms under this phenomenon, and more research in this area needs to be carried out in the future.

Author Contributions: This study was designed by C.A. and B.R. The data were collected and analyzed by D.S. All three authors contributed to the interpretation of the findings and writing of the manuscript. All authors have read and agreed to the published version of the manuscript.

Funding: This research was supported by a grant from the Natural Sciences and Engineering Research Council of Canada (RGPIN-2016-05523) to CA and the Canadian Institute of Health Research to BR (Grant number MOP125925).

Institutional Review Board Statement: The study protocol was approved by the research ethics board at Baycrest Centre (REB # 06-31).

Informed Consent Statement: All participants provided written consent after being informed about the study.

Data Availability Statement: Data can be made available upon request and approval through the research ethics board.

Conflicts of Interest: The authors declare no competing financial interest.

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