No evidence for peripheral mechanism attenuating auditory ERPs to self-induced tones

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Abstract

The N1 and P2 event-related potentials (ERPs) are attenuated when the eliciting sounds coincide with our own actions. Although this ERP attenuation could be caused by central processes, it may also reflect a peripheral mechanism: the co-activation of the stapedius muscle with the task-relevant effector, which reduces signal transmission efficiency in the middle ear, reducing the effective intensity of concurrently presented tones, which, in turn, elicit lower-amplitude auditory ERPs. Because stapedius muscle contraction attenuates frequencies below 2 kHz, no attenuation should occur at frequencies above 2 kHz. A self-induced tone paradigm was administered with 0.5, 2.0, and 8.0 kHz pure tones. Self-induced tones elicited attenuated N1 and P2 ERPs, but the magnitude of attenuation was not affected by tone-frequency. This result does not support the hypothesis that ERP attenuation to self-induced tones are caused by stapedius muscle contractions.

Introduction

Whereas sensory processing is often investigated by presenting various types of stimulation to passive participants, in everyday life most sensory events happen as we perform various actions; some of the sensory events are even caused by our actions. Actions may influence stimulus processing in the periphery by directly changing the physical interaction between the stimulation and the receptors (e.g. squinting in bright light). Actions may also change central aspects of sensory processing: for example, self-initiated head-movements lead to the suppression of the corresponding vestibular re-afference (Cullen, Brooks, Sadeghi, 2009). There has been growing interest in the influence of actions on auditory sensory processing, because event-related potential (ERP) and -magnetic field (ERF) correlates of auditory event detection are attenuated when the eliciting sounds are preceded or induced by actions. Whereas it is generally assumed that the attenuation is rooted in central processes, the available evidence does not rule out the potential involvement of a peripheral mechanism. The goal of the current study was to test the viability of this alternative hypothesis.

A number of studies reported that self-generated speech sounds elicited attenuated auditory N1 ERPs and ERFs (e.g. Curio, Neuloh, Numminen, Jousmaki, & Hari, 2000; Flinker, Chang, Kirsch, Barbaro, Crone, Knight, 2010; Heinks-Maldonado, Mathalon, Gray & Ford, 2005; Heinks-Maldonado, Nagarajan & Houde, 2006; Houde, Nagarajan, Sekihara & Merzenich, 2002; Numminen & Curio, 1999; Ventura, Nagarajan, Houde, 2009); moreover, a number of studies showed N1-attenuation for non-speech-related actions and concurrent (speech or non-speech) sounds as well (Aliu, Houde, & Nagarajan, 2009; Baess, Horváth, Jacobsen, & Schröger, 2011; Baess, Jacobsen, & Schröger, 2008; Ford, Gray, Faustman, Roach, & Mathalon, 2007; Knolle, Schröger, Baess & Kotz, 2012; Martikainen, Kaneko, & Hari, 2005; McCarthy & Donchin, 1976; Schäfer & Marcus, 1973; Sowman, Kuusik,

Johnson, 2012). Because N1 reflects the sensory processing of auditory events (Näätänen & Picton, 1987), it is generally assumed that these results reflect sensory attenuation driven by the same central mechanism - *internal forward modeling* (Miall & Wolpert, 1996). Based on the outgoing motor commands, internal forward modeling allows one to adjust sensory processing in advance to accommodate predictable re-afference caused by self-initiated movements. Forward modeling has been confirmed to support many functions in the neural system (Davidson & Wolpert, 2005), and it seems plausible that consistent action-effect patterns are represented by forward models. To date, however, there is no conclusive evidence regarding the role of forward modeling in auditory N1-attenuation. A couple of studies found attenuated N1 ERPs and ERFs to sounds which merely coincided, but were not causally related to the given action (Hazemann, Audin, Lille, 1975; Horváth, 2013, and Horváth, in press; Horváth, Maess, Baess, Tóth, 2012), which suggests that these ERP attenuations may be related to the action itself and not to an action-sound association.

It is well-known that the N1 waveform is sensitive to the physical characteristics of the eliciting sounds. Importantly, N1 amplitude decreases with decreasing sound intensity (Picton, Woods, Baribeau-Braun, & Healey, 1977; Näätänen & Picton, 1987), therefore an attenuated N1 response could reflect a mechanism changing auditory signal transmission efficiency at the periphery. One such mechanism in humans is the contraction of the middle-ear stapedius muscle, which leads to the effective attenuation of incoming sound intensity. Although stapedius muscle contraction can be triggered by the presentation of high intensity sounds through the stapedius reflex (see e.g. Mukerji, Windsor & Lee, 2010), stapedius muscle activation can be observed when vocalization, chewing or even bodily movements are initiated (i.e. the contraction starts 50-100 ms before the actual movement, Carmel & Starr, 1963, Salomon & Starr, 1963; Simmons, 1964), and it may be also under voluntary control in some individuals (Liberman & Guinan, 1998). Middle-ear muscle activity also seems to occur

in conjunction with other motor events during sleep (Slegel, Benson, Zarcone, & Schubert, 1991). The transmission decrease caused by the acoustic activation of the stapedius muscle is about 10 dB (Rabinowitz, 1977), but much larger decreases may also occur (Pang & Peake, 1987; Simmons, 1964). The contraction of the stapedius muscle primarily attenuates frequencies lower than 2 kHz (Borg & Zakrisson, 1974; Rabinowitz, 1977; Zakrisson & Borg, 1974). It has been suggested that the function of stapedius muscle contraction is to reduce the level of physiological noise resulting from our own movements (especially those generated in or near to our head, e.g. speech, chewing, head-movements, see Simmons, 1964), and to prevent the upward spread of masking (Liberman & Guinan, 1998; Pang & Guinan, 1997), that is, it may allow one to counteract the nonlinearly growing masking of higher frequencies by low frequency sounds (for a summary, see Moore, 2012).

Based on these findings, we propose that N1 attenuation may be brought about by the co-activation of the stapedius muscle with the task-relevant effector, which leads to the effective presentation of a softer tone. Though this hypothesis has been mentioned in the literature (Ford, Mathalon, Kalba, Whitfield, Faustman, Roth, 2001), its implications, to our knowledge, have not been empirically tested. An overview of the literature on N1-attenuation in self-initiation- or coincidence-based paradigms shows that most studies used spectrally rich sounds (clicks, noise, or speech) or pure tones in the 0.5-2.0 kHz range, which matches the frequency range directly affected by the stapedius muscle contraction. If N1 attenuation was brought about by co-activation of the stapedius muscle with the task-relevant effector, then the magnitude of N1-attenuation should diminish for frequencies above 2 kHz, because of the decreased effect of stapedius muscle contraction on transmission for these frequencies. In the present study this implication of the peripheral hypothesis was tested by administering a self-induced tone paradigm with 500, 2000, and 8000 Hz pure tones. Furthermore, the effect of tone frequency was assessed not only for N1, but also for P2-attenuation. In contrast to N1,

the functional role of the auditory P2 waveform is largely unknown (Crowley & Colrain, 2004). Although many previous studies did not explicitly investigate the P2-attenuation co-occuring with N1-attenuation (which was nonetheless observable in the ERPs, see e.g., Baess et al., 2011; Ford & Mathalon, 2004; Schäfer & Marcus, 1973), recent studies showed that N1-attenuation is consistently followed by P2-attenuation (e.g. Knolle, Schröger, & Kotz, 2013; Sowman et al., 2012), even though the attenuation-effects could be differentially manipulated (Horváth et al., 2012; Knolle et al., 2012; Knolle, Schröger, & Kotz, in press; SanMiguel, Todd, & Schröger, in press).

Methods

Participants

14 young adult volunteers (six women, aged 18-25 years, mean 21 years, thirteen right-, one left-handed) participated in the experiment for monetary compensation. Data from two participants were not used in the analyses due to the exceeding number of ocular artifacts contaminating the EEG recordings (over 40% of the epochs in some conditions). All participants reported normal hearing and no history of neurological disorders. Before the experiment, participants gave written informed consent after the experimental procedures were explained to them.

Stimuli and procedures

Participants were sitting in an armchair in a sound-proofed room during the experiment. There were three types of conditions: In the Motor-Auditory condition, participants were instructed to press a rod-mounted button held in their dominant hand at regular 3 s intervals, and more importantly, count the tones triggered by the button-presses. Button-presses resulted in the presentation of a tone for the first 61 to 70 button-presses (randomly with uniform distribution). The randomness of the time-point at which the button

"no longer produced tones" allowed monitoring whether participants attended the tones. Due to the constraints of the stimulation equipment, there was a 7 ms delay between the button-press and tone onset, which was taken into account during EEG processing. At the end of the block, participants reported the number of tones, and received feedback about accuracy. In the Motor condition, participants were instructed to press the button at regular 3 s intervals until instructed to stop. In the Auditory condition, the first 51-60 tones (random uniform distribution) from the sequence delivered in the preceding Motor-Auditory condition was replayed. Participants were instructed to count the tones, and report the number of tones at the end of the block. Due to a programming error, tone onset-to-onset intervals were 10 ms longer than in the Motor-Auditory condition, which, compared to the average onset-to-onset interval of 3 s, is unlikely to significantly affect the present results. The presentation of the conditions was organized into block-triplets. Each triplet started with a Motor-Auditory, followed by a Motor-, and concluded by the Auditory condition block. The experiment started with a practice triplet, in which the tone was a 50 ms long white-noise burst (including 10-10 ms linear rise and fall times). In the following experimental phase six triplets with 50 ms long pure tones (including 10-10 ms linear rise and fall times) of 500, 2000 and 8000 Hz frequency were administered. Each frequency was presented in two triplets. The order of triplets was randomized so that each type of triplet was delivered once in the first half and once in the second half of the experiment. The sound level was individually adjusted to 60 dB sensation level (above hearing threshold level) using the 2000 Hz tone. To compensate for the higher normal hearing threshold level (Suzuki, & Takeshima, 2004), the sound level of the 8000 Hz tone was set 10 dB higher than for the other two tones.

Analysis of the behavioral data

Between-key-press-intervals were analyzed in repeated-measures analysis of variance (ANOVA) using condition (Motor-Auditory vs. Motor) and frequency (500, 2000 and 8000

Hz) factors. Only the first fifty events in each experimental block were used in the analyses. The number of counting errors were analyzed in a repeated-measures condition (Motor-Auditory vs. Auditory) × frequency ANOVA. For all ANOVAs generalized eta-squared effect sizes (Olejnik and Algina, 2003; Bakeman, 2005) are reported; also Greenhouse-Geisser correction was applied to correct for potential violations of the sphericity assumption (in all such cases the corrected p-value, ε and the uncorrected degrees of freedom are reported). The alpha-level was set to 0.05. For all analyses all significant effects are reported.

EEG-recording and analysis.

The electroencephalogram (EEG) was recorded with Ag/AgCl electrodes mounted on an elastic cap (EASYCAP, Herrsching, Germany) according to the 10% system (Nuwer, Comi, Emerson, Fuglsang-Frederiksen, Guerit, Hinrichs et al., 1998) with a sampling rate of 1000 Hz and on-line, 200 Hz low-pass filtering. Additional electrodes were placed at the mastoids. The reference was placed on the tip of the nose, the ground on the forehead. Horizontal electrooculogram (EOG) was obtained by an electrode placed under the right eye. Vertical EOG was calculated off-line by subtracting the horizontal EOG signal from the signal recorded at Fp2. The EEG was off-line re-referenced to average reference, 20 Hz low-pass filtered, and segmented into epochs of 600 ms corresponding to tone-onsets (Motor-Auditory and Auditory conditions) and time-points when a tone onset would have been if key-presses would have triggered a tone (Motor conditions), including a 200 ms interval preceding the onset of the tone (or the time-point when a tone would have occurred, see above). Amplitude calculations were referred to the average signal measured in this (baseline) interval for each channel. Only the first fifty epochs of each experimental block were used in the analyses. Epochs with a signal range exceeding 100 μV on any EEG of EOG channels were discarded.

The epochs were individually averaged for each type of condition and tone-frequency. Individual N1 and P2 amplitudes were calculated as average signals in 20 ms long intervals centered at the N1 and P2 peaks of the group average waveforms measured at Fz and Cz in the Auditory conditions. For each frequency, the tone-related ERPs elicited by the self-induced tones were estimated by subtracting the ERPs obtained in the Motor condition from the ERP obtained in the Motor-Auditory conditions, that is, Motor-Auditory-minus-Motor difference ERPs were calculated. Because N1 and P2 typically peak at Fz and Cz, respectively, and often show reversed peak amplitudes at the mastoids when an average reference is used, N1 and P2 amplitudes were measured as Fz-minus-average-mastoid, and Cz-minus-average-mastoid signals, respectively. This also maximizes the chance to detect potential between-frequency differences in the magnitude of attenuation. These were normalized by the amplitudes measured in the corresponding Auditory conditions (i.e., the individual amplitudes for each frequency were divided by the group-mean amplitudes from the corresponding Auditory condition), and submitted to a repeated-measures ANOVA using stimulation (self-induced vs. replayed) and frequency (500 Hz, 2000 Hz and 8000 Hz) factors. Normalization was necessary, since N1 and P2 amplitudes could be different for tones with different frequencies. In these analyses different attenuation-ratios for different frequencies would be signaled by Stimulation × Frequency interactions. Because it could be a priori assumed that the attenuation-ratio would be more modest in the 8000 Hz condition than in the others, planned comparisons of the attenuation-ratios between 8000 Hz and the other two frequencies were also conducted (paired Student's t-tests; we also report JZS Bayes factors - BF_{01} – based on the default Bayesian t-test, as proposed by Rouder, Speckman, Sun, Morey, & Iverson, 2009, to quantify evidence in favor of the null hypothesis). To test whether N1- and P2-attenuation was significant at each frequency, amplitudes in the auditory and motor-auditory conditions were compared by Student's t-tests as well. To test whether the

topographical distributions of the attenuation effects differed from the topographical distributions of the corresponding N1 and P2 ERPs elicited in the auditory conditions, stimulation (motor-auditory, auditory) × electrode (Fz, Cz, Pz, Oz) ANOVAs were conducted with the amplitudes vector-normalized following the method described by McCarthy & Wood (1985) for each frequency. In these analyses, a significant interaction would mean that the attenuation effect was not a "pure" modulation of the underlying ERP, rather, that it was brought about by the emergence of a different ERP component. The choice of midline electrodes is motivated by the fact that Horváth (2013, and in press) found that action-tone coincidence related ERP attenuation in the N1 time-interval was (in part) due to the emergence of a posterior, midline ERP effect.

Results

Behavioral measures

Counting performance is presented in Figure 1. The condition × frequency ANOVA of the number of erroneous counts showed that participants made more errors in the motor-auditory than in the auditory conditions (significant condition main effect): $F(1,11)=7.86$, $\eta_G^2=0.06$, $p<0.05$.

The condition × frequency ANOVA showed that between-key-press intervals were shorter in the motor-auditory (2874 ± 157 ms, mean ± standard deviation) than in the motor (3098 ± 239 ms) conditions (significant condition main effect): $F(1,11)=16.50$, $\eta_G^2=0.19$, $p<0.01$.

ERPs

Figure 2 shows the ERPs elicited at selected midline electrodes and the average of the mastoid signals for all tone frequencies and conditions. The topographies of the N1 and P2

waveforms in the tone-related ERPs, as well as the corresponding (reversed) attenuation-effects are shown in Figure 3.

N1 peaked at 97, 93, and at 93 ms at Fz following the onsets of the 500, 2000, and 8000 Hz tones, respectively. The ANOVA of the normalized amplitudes showed a significant stimulation main effect only: $F(1,11)=65.58$, $\eta_G^2=0.13$, $p<0.001$ (for the frequency main effect: $F(1,11) = 0.15$,$\varepsilon = 0.86$, $p = 0.83$; and for the interaction: $F(2,22) = 0.14$, $\varepsilon = 0.98$, $p = 0.87$). The attenuation ratio was significantly different from zero at each frequency: It was 24 ± 24 %, $t(11) = 3.44$, $p<0.01$, $BF_{01} = 0.104$; 29 ± 17 %, $t(11) = 5.85$, $p<0.001$, $BF_{01} = 0.003$; and 27 ± 26 %, $t(11) = 3.68$, $p<0.01$, $BF_{01} = 0.073$, respectively for the 500, 2000 and 8000 Hz tones. The planned direct comparisons of the attenuation-ratios showed no significant differences: $t(11) = 0.15$, $p = 0.89$, $BF_{01} = 4.61$, for the 500 vs. 8000 Hz comparison; and $t(11) = 0.63$, $p = 0.54$, $BF_{01} = 3.86$, for the 2000 vs. 8000 Hz comparison. The stimulation (motor-auditory, auditory) × electrode (Fz, Cz, Pz, Oz) ANOVAs of the vector-normalized amplitudes conducted separately for each frequency showed no significant interactions.

P2 peaked at 180, 181 and at 180 ms at Cz following the onsets of the 500, 2000, and 8000 Hz tones, respectively. The ANOVA of the normalized amplitudes showed only a significant stimulation main effect: $F(1,11)=17.49$, $\eta_G^2=0.30$, $p<0.01$ (for the frequency main effect: $F(1,11) = 0.28$, $\varepsilon = 0.71$, $p = 0.68$; for the interaction: $F(2,22) = 0.52$, $\varepsilon = 0.80$, $p = 0.56$). Attenuation ratios were significantly different from zero at each frequency: they were 57 ± 53 %, $t(11) = 3.73$, $p<0.01$, $BF_{01} = 0.067$; 63 ± 65 % $t(11) = 3.35$, $p<0.01$, $BF_{01} = 0.121$; and 71 ± 62 %, $t(11) = 4.02$, $p<0.01$, $BF_{01} = 0.043$, respectively, for the 500, 2000 and 8000 Hz tones. The planned direct comparisons of the attenuation-ratios showed no significant differences: $t(11) = 1.07$, $p = 0.31$, $BF_{01} = 2.76$, for the 500 vs. 8000 Hz comparison; and $t(11) = 0.70$, $p = 0.50$, $BF_{01} = 3.72$, for the 2000 vs. 8000 Hz comparison. The stimulation (motor-

auditory, auditory) × electrode (Fz, Cz, Pz, Oz) ANOVAs of the vector-normalized amplitudes conducted separately for each frequency showed no significant interactions.

Discussion

Self-induced tones elicited attenuated N1 and P2 ERPs, which confirms previous results (e.g. Aliu et al., 2009; Baess et al., 2008; Baess et al., 2011; Ford et al., 2007; Knolle et al., 2012; Martikainen et al, 2005; McCarthy & Donchin, 1976; Schäfer & Marcus, 1973; Sowman et al., 2012), and extends the range of pure tone frequencies at which N1/P2 suppression was observed. The results of the topographical analyses are compatible with the interpretation that the ERP-effects were modulations of the N1 and P2 waveforms (i.e. they were not caused by the emergence of other components). Importantly, no significant difference in the magnitude of attenuation as a function of tone-frequency was found. The direct comparisons gave some evidence for the equality of the attenuation effects (i.e. JZS Bayes factors larger than 3 in favor of the respective null-hypotheses, see Rouder, et al., 2009, except for the P2 attenuation-ratios in the 0.5 vs. 8 kHz comparison, where P2-attenuation was numerically stronger in the 8 kHz condition). This suggests that N1 and P2 attenuations across tone-frequencies do not decrease or change substantially in the 0.5-8.0 kHz range. The results do not support the hypothesis that auditory ERP attenuation for self-induced tones is caused by the co-activation of the stapedius muscle with the keypress-related movement, and are compatible with the notion that these N1/P2 attenuations are caused by non-frequency-specific, possibly central mechanisms.

In the motor-auditory and the auditory conditions participants performed a tone counting task, and participants had to keep a steady key-pressing pace in the motor and the motor-auditory conditions. In contrast to previous studies (in which no task was given in the auditory condition) this arrangement allowed us to make sure that participants attended the

tones in the auditory condition in a qualitatively similar fashion to that in the motor-auditory condition. On the other hand, however, counting performance was lower in the motor-auditory than in the auditory condition. This difference in performance may signal that less attentional resources were allocated to the tones in the motor-auditory condition. Because N1 elicited by sounds is enhanced when attention is directed towards the sound channel (see e.g. Hillyard, Hink, Schwent & Picton, 1973, Okamoto, Stracke, Wolters, Schmael, & Pantev, 2007), it could be argued that N1 and P2 may be elicited with higher amplitude in the auditory than in the motor-auditory condition, because more attention is directed towards the tones in the auditory condition. Although this is a well-known shortcoming of these types of paradigms (see Baess, et al., 2011), Timm, SanMiguel, Saupe, & Schröger (2013) suggest that such an attentional contribution to the observed effects is not substantial.

The present results do not allow direct conclusions on whether the ERP attenuation observable for sounds induced by speech-producing movements is caused by peripheral or central processes. Because it is only an assumption that the mechanism underlying auditory ERP attenuation is the same for speech- and non-speech-related actions, and because the peripheral hypothesis seems more plausible for speech-producing movements, it is possible that ERP attenuation for speech-producing actions is based (in part) on the co-activation of the stapedius muscle. On the other hand, if these central processes are triggered even for the arbitrary association of a button-press and a tone, then such processes probably play a substantial role when it comes to highly familiar patterns of action-sound correspondence like in speech.

In summary, the present results do not support the hypothesis that auditory ERP attenuation for self-induced tones is caused by the co-activation of the stapedius muscle with the tone-inducing movement.

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Author Notes

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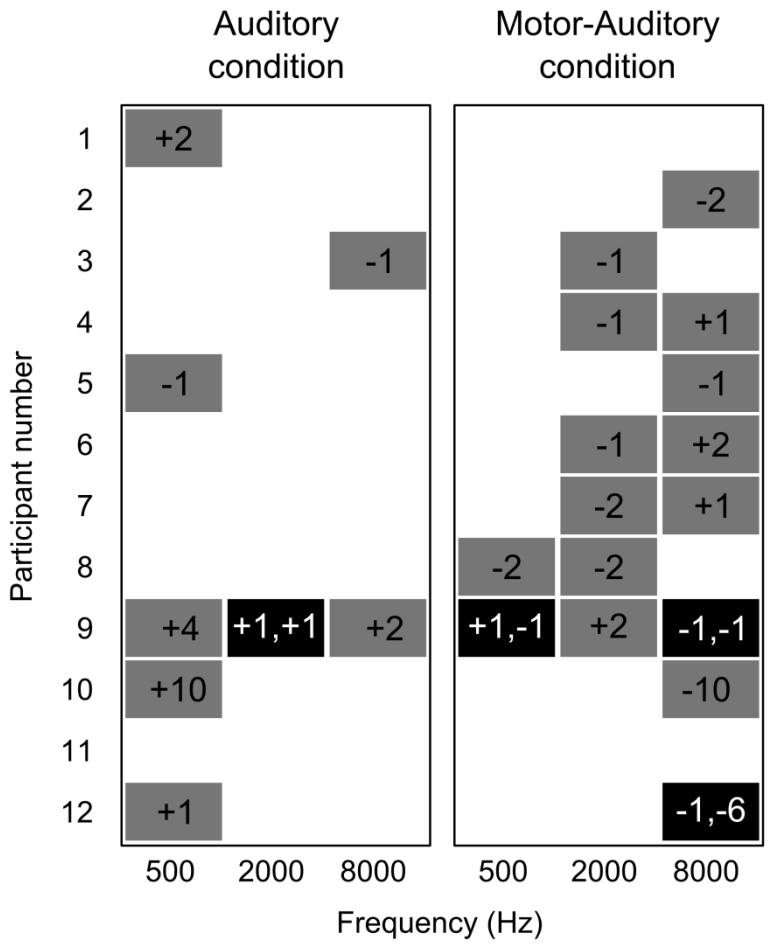
Figure captions

Figure 1. Counting performance for each participant, in each Auditory and Motor-Auditory condition with each tone-frequency. Gray fields indicate that the given participant miscounted in one of the two blocks of the given condition, whereas black fields indicate miscounting in two blocks. The positive or negative numbers in these fields indicate the difference between the reported and the correct number of tones.

Figure 2. Group-average ERP waveforms for the three frequencies in the Auditory-, Motor-, and Motor-Auditory conditions, and the corresponding Motor-Auditory-minus-Motor difference waveform (corrected Motor-Auditory waveform) measured at Fz, Cz, Pz, Oz leads, and the average of the left and right mastoids signals (M). Tone onset is at the crossing of the axes.

Figure 3. Group-average topographical distributions of the N1 and P2 ERPs elicited in the Auditory conditions (average signals in 20 ms long windows centered at the group-average peak latencies), and the corresponding reversed attenuation effects for the three tone frequencies. The signal range differs between the individual topographical distributions in order to emphasize similarities or differences in shape.



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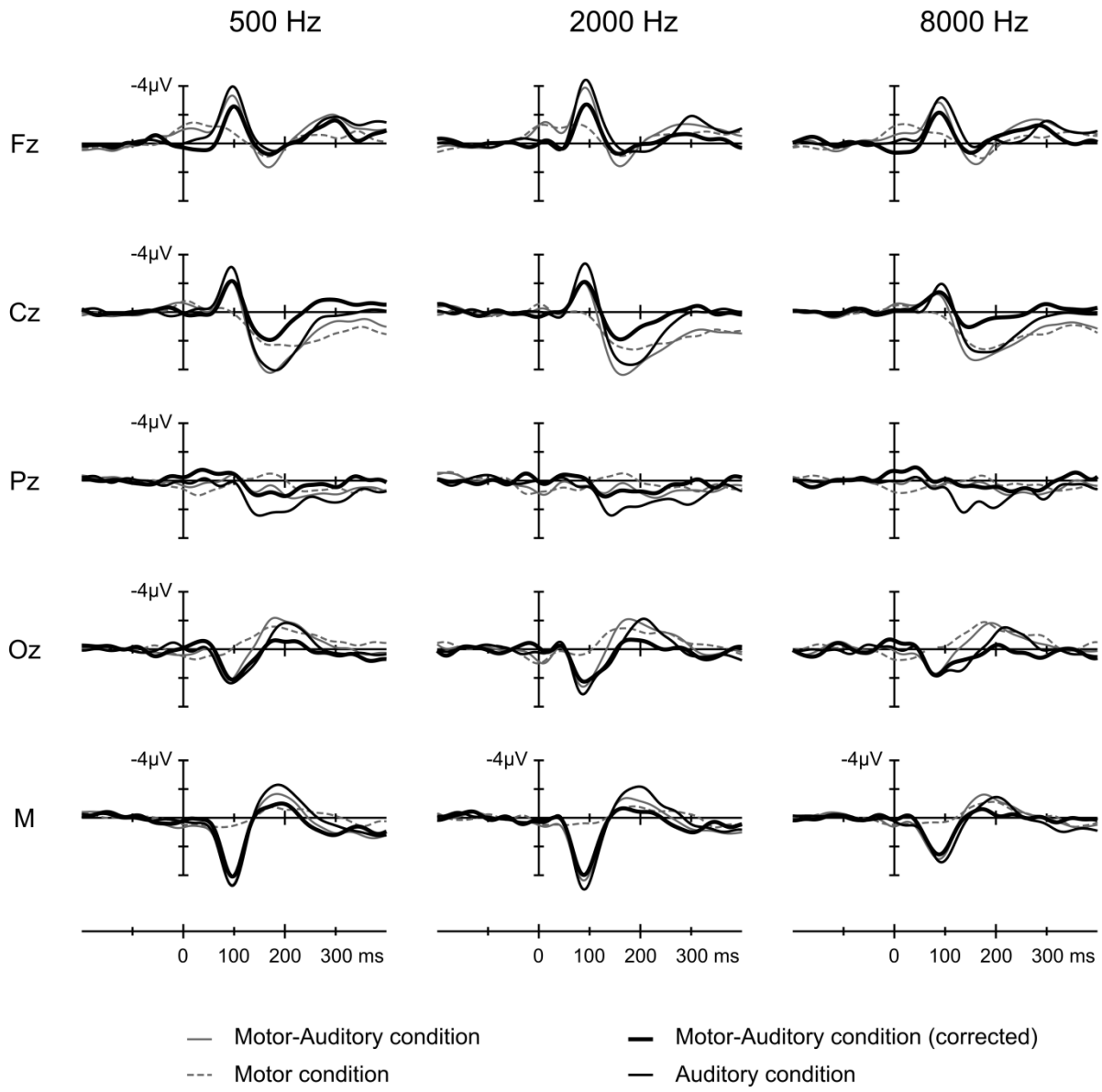

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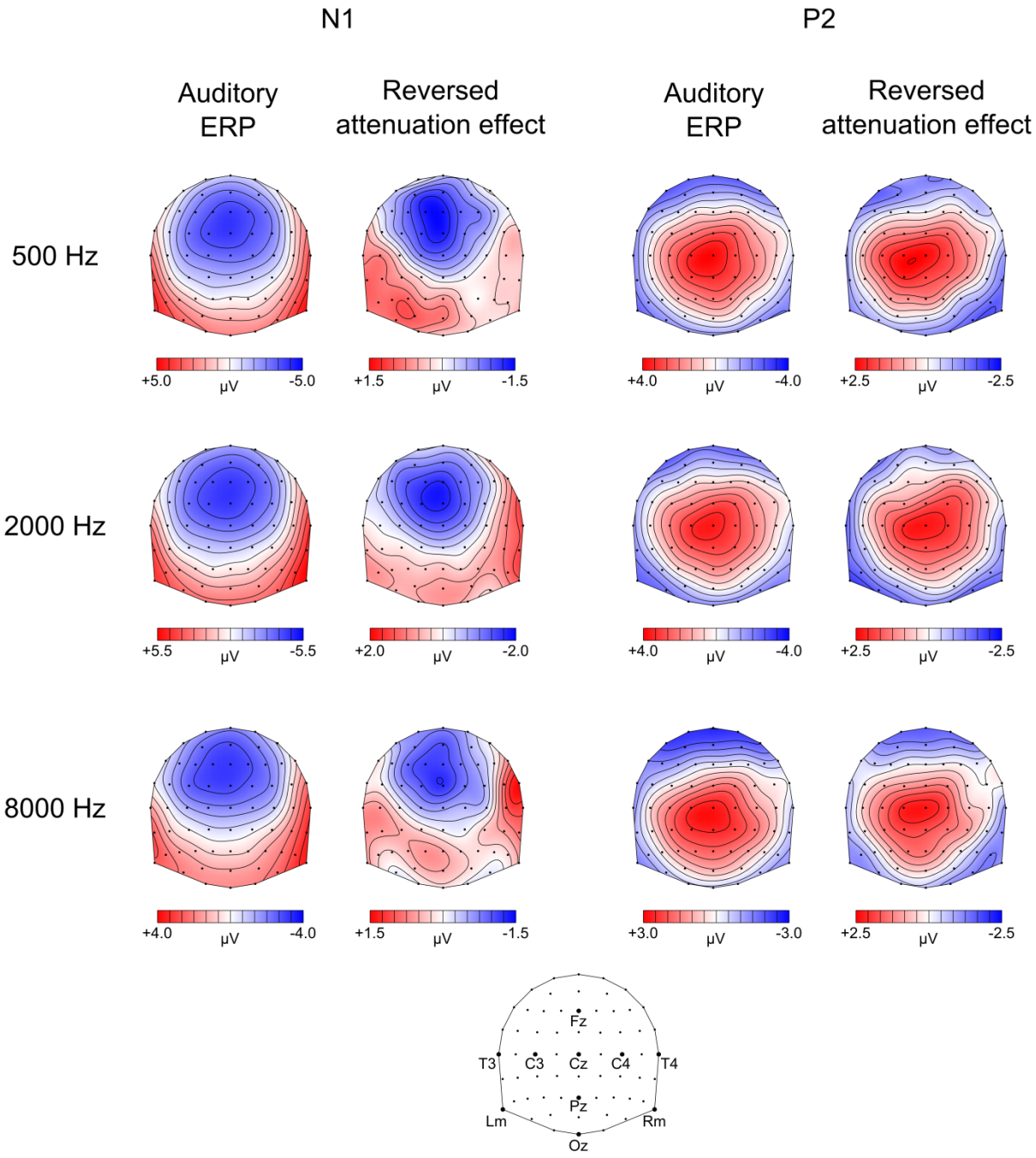

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