A Signature of Passivity? An Explorative Study of the N3 Event-Related Potential Component in Passive Oddball Tasks

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Short title: N3 ERP Component

Key words: Cortical Preparation, Current Source Density, Oddball, Principal Component Analysis (PCA), Uncertainty

Abstract

Many passive oddball experiments show a negative deflection N3 peaking between 400 and 500 ms, but this wave has never been analyzed properly. We conducted five passive oddball experiments, in which the number of deviants (i.e., one or two), their alleged meaning and their distinctiveness varied. Mastoid- or common-referenced waveforms showed a fronto-central N3 in all experiments. The data were CSD (Current Source Density) transformed and underwent a Principal Component Analysis (PCA). The PCA revealed N3 containing two subcomponents with very stable peak latencies of about 415 and 455 ms, respectively. Both topography of the subcomponents and their variation with experimental conditions were very similar but different from other negative ERP components N1 and N2. The two N3 subcomponents were maximally negative over midfrontal areas but positive over the parietal and particularly over the posterior temporal cortex. Both frontal negativity and posterior positivity exhibited clear oddball effects, i.e., they were larger to deviants than to standards. Positive components P3a and P3b were also analyzed, which replicated the previously known effects. In the Discussion, we speculate about possible meaning of N3; on this basis, specific hypotheses are formulated and further studies are suggested to test these hypotheses.

More than half a century has passed since we first learned that low-frequent stimuli, presented among highly frequent ones, elicit in the EEG a slow late positive wave with a typical peak latency slightly longer than 300 ms (Sutton et al., 1965). Already the first systematic review on this effect is almost four decades old [Donchin, 1981]. Later on, this late positivity of event-related potential (ERP), called P300 or P3, was found to entail two distinct components, P3a and P3b [e.g., Courchesne et al., 1975]. Moreover, even P3b was shown to be not a unitary wave but consists of further subcomponents [e.g., Falkenstein et al., 1994].

The eliciting condition, referred to as "oddball task" [Duncan-Johnson & Donchin, 1977] standardly involved an active task to respond (overtly or covertly, e.g., mental count) to the rare stimuli or to both rare and frequent stimuli. In the former case, the factors rarity (or probability) and task-relevance are confounded. This fact underlay the idea to obtain the same effect without any active task requirement [Polich, 1987]. It was shown that such a passive oddball paradigm also yields the P3 ERP component to the rare stimulus, although its amplitude is considerably smaller than in the active oddball paradigm [Lang et al., 1997; Polich, 1989]. Although the exact component structure of this passive P3 remains unclear, the data indicate that it is not just a P3a but contains at least a portion of the typical P3b [e.g., Polich and McIsaak, 1994; Bennington and Pollich, 1999; Lang et al., 1997].

The interest to the passive P3 paradigm was particularly stimulated by clinical consideration, specifically by the idea to use cognitive ERP components for the analysis of cortical functions in behaviorally unresponsive patients, e.g., in vegetative state, minimally conscious state, in the late stages of neurodegenerative diseases or in patients with Guillain-Barré syndrome. As these patients do not have any behavior, their cognitive abilities can only be examined by means of direct neurophysiological techniques, and the oddball ERP paradigm appears to be a convenient one. However, for the same

reason of behavioral unresponsiveness the patients cannot be given an overt task – and if a task is covert (e.g., to count a rare stimulus), we cannot know whether they really perform it.

Several control studies aimed at the development of the passive oddball paradigm for severely brain damaged patients confirmed the presence of P3, but the waveforms presented in these, studies also indicated a later negative deflection with a peak latency between 400 and 500 ms. Thus Bostanov & Kotchoubey [2006] used a continuous wavelet transform and clearly showed this wave in both time and time-frequency analyzed responses. Erlbeck et al., [2014] directly compared ERPs in the oddball paradigm (a) with an active instruction to respond to stimuli, (b) with active distraction of attention away from stimuli, and (c) without any active task. The negativity following P3 to deviant stimuli can be clearly seen in the last condition, but not in the other two. Unfortunately, the hypotheses these authors had developed to their oddball experiments concerned the Mismatch Negativity (MMN) only, and the later components were not analyzed. Furthermore, a clear fronto-central negative can be seen in the results obtained in passive oddball conditions with broadly various kinds of stimulation [e.g., Oades et al., 1995a, 1995b; Potts, 2004; Justen and Herbert, 2016]. However, most of these studies were concentrated on P300 and/or MMN and did not analyze or even notice the post-P300 negativity. For example, O'Donnell et al. [1992] carefully describe their ERPs as consisting of two negative-positive complexes N1-P2 and N2-P3 but ignore a clear negativity that follows P3; this was one of the first studies were such negativity can be seen in Figures.

Only Barry et al. [2006, 2009] included this wave into their analysis and named it N3. The principal interest of this group was, however, the relationship between the phase-locked ERPs and the underlying EEG activity. Therefore even they did not perform a regular analysis of N3 even though they noticed some regularities of this ERP component.

To our best knowledge, the present study is the first ERP report explicitly devoted to N3. For this reason, the study is largely explorative. Of course, this is a limitation, but facts must be accumulated before precise hypotheses can be formulated. At present, on the basis of the previous unsystematic knowledge we suggested the following preliminary hypotheses:

- All oddball paradigms without an active behavioral task will elicit a complex P3a/P3b followed by a negative wave with a peak latency 400-500 ms (i.e., N3);
- N3 differs from other ERP negativities;
- Because meaningful stimuli are, in a sense, “less passive” than meaningless stimuli, N3 will be smaller to stimuli having a meaning for an individual;
- Because P3 responses in the passive oddball paradigms were shown to habituate [Polich, 1987; Polich and McIsaak, 1994], we expected a decrease of N3 amplitude with repetition.

General Methods

Participants of all experiments were healthy individuals between 19 and 35, having had no diseases of the nervous system or hearing disorders in the past. All of them reported being well aroused and in a good mood at the beginning of the experiment. None reported the use of any medicaments during the last weeks before the experiments. They were seated in comfortable chair and asked to close their eyes during EEG recording and to listen attentively to the stimuli presented through earphones. No other instruction was given. The instruction to close eyes resulted, on the one hand, in the almost complete absence of ocular activity, on the other hand, in some participants whose data were later dismissed due to signs of sleep stages N1 or even N2 [AASM sleep criteria) in their EEG. In the following, we shall describe only the samples of participants whose data were included into the analyses.

Most subjects participating in Experiment I also participated in Experiments III and IV. In all of them, the order was the same: first Experiment I, then III, then IV.

Recording

The EEG was recorded using 64 active ActiCHamp electrodes (Easycap GmbH, Herrsching, Germany) located according to the extended 10-20 system. In addition to the EEG, the vertical and horizontal electrooculograms were recorded. The resistance was below 25 kOhm. During recording, the EEG was referred to Cz, but the following processing was performed after re-referencing to average mastoids. The digitalization rate was 1000 Hz, and the low pass filter was 500 Hz.

Preprocessing

The off-line inspection of the recordings revealed in some traces poor data quality in one or two of the 64 channels. These channels (usually 2 to 4 per experiment) were replaced with interpolation of the adjacent electrodes. After this, an Independent Component Analysis (ICA) was employed to separate and remove activity related to ocular artifacts according the AMICA algorithm [Palmer et al., 2012]. The EEG was then broken into segments starting from 200 ms prior to tone onset and ending 800 ms after this. The segments that still contained artifacts notwithstanding the preceding ICA correction were dismissed. The ERPs were filtered within a band from 0.1 to 30 Hz and averaged in relation to a baseline from -200 ms to 0 ms.

Current source density (CSD)

We computed reference-free CSD estimates ($\mu V/cm^2$ units; 10-cm head radius) by mean of the spherical spline surface Laplacian [Perrin et al., 1989] with the following parameters: 50 iterations; $m = 4$; smoothing constant $\lambda = 10^{-5}$ (for detailed description of the procedure see Kayser and Tenke [2006]). CSD transformation allows to represent data in a reference-free manner and help to avoid problems associated with the choice of a recording reference. The CSD pattern of sources and sinks creates a clear image of the current generators of ERP components. This is especially important for auditory ERP when

usage of linked-mastoids reference lead to cancelling out potentials generated by sources in temporal – auditory cortex.

Figure 1 shows a comparison between SCD and two kinds of reference for Experiment I (three-stimulus oddball with one standard and two deviant stimuli, see below).

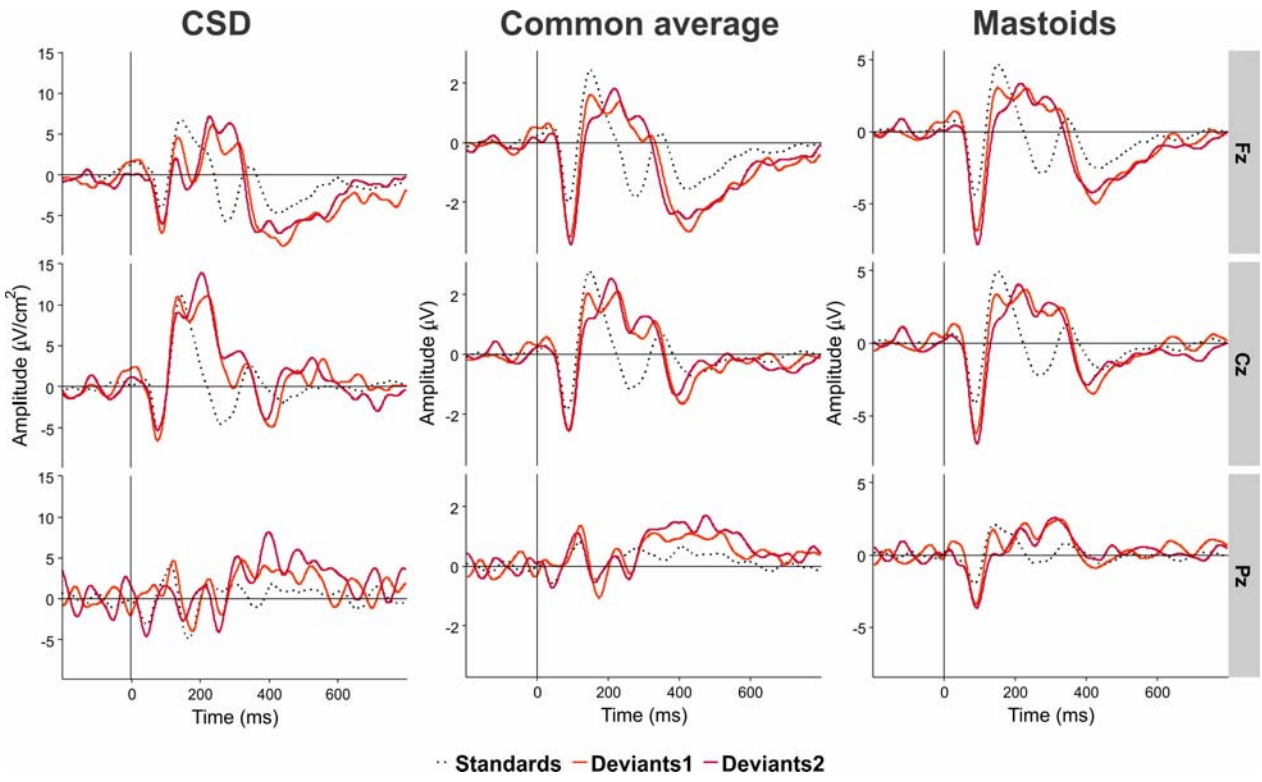

Figure 1. An illustration of the effect of electrical reference, for the results of Experiment I at three midline electrodes Fz, Cz, and Pz. Black dotted line, standards. Light red line, Deviant 1. Dark red line, Deviant 2. Positivity at this and following figures is plotted upwards.

Principal components analysis (PCA)

After preprocessing, data were imported into the ERP PCA Toolkit [Dien, 2010]. For the first 4 experiments a temporal PCA with a Promax rotation was conducted to combined dataset of averaged across trials CSD-ERPs where time intervals, conditions, electrodes sites and participants considered as

observations and time points as variables. In order to determine the number of factors to retain, the parallel test [Horn, 1965] was applied. The covariance matrix and Kaiser normalization were used for the PCA. The same procedure was applied to Experiment V data separately. As a result of the parallel test, 55 temporal factors were extracted for rotation for Experiments I to IV and 44 for Experiment V. The waveforms for each factor were then reconstructed by multiplying factor scores by their corresponding loadings and standard deviations. CSD of reconstructed waveforms representing principal components of interest were averaged in 50 ms around peak values (±25 ms) in ROIs, time intervals and conditions. These data were used for statistical calculations.

Because many studies standardly report ERP data using average mastoid reference, we additionally carried out the same PCA for mastoid-referred data to enhance comparability with the literature. The results were very similar for all experiments. Therefore, we performed an analysis including the data of all five experiments together. In the experiments with two deviant stimuli, responses to all deviants were averaged together to obtain one standard and one deviant waveform. A total of 41 Factors were extracted. The following analysis was performed like for CSD data. The results of the statistical analysis of mastoid-referenced data will not be reported, because this would considerably increase (almost double) the volume of the article without an added information value.

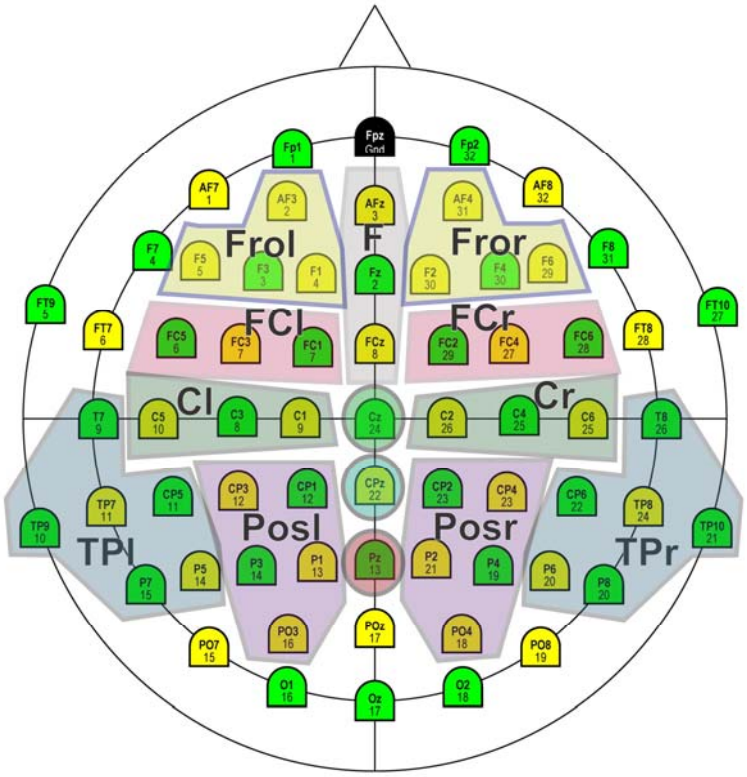

Figure 2. Selected regions of interest for statistical analysis.

Statistical analysis

As ERP components in oddball experiments are most typically investigated at the midline, our first analysis included only midline locations: frontal (mean of AFz, Fz and FCz), central (Cz), centroparietal (CPz) and parietal (Pz) leads. The repeated-measures ANOVA included the factors Stimulus (2 or 3 levels in different experiments), Site (4 levels), and Time (3 levels). To analyze the activity over the hemispheres, five regions of interests (ROIs) were selected for each hemisphere: frontal (Fro), frontocentral (FC), central (Ce), posterior (Pos), and temporoparietal (TP), as shown in Figure 2. The corresponding repeated-measures ANOVA included the factors Stimulus (2 or 3 levels), Area (i.e., ROI, 5 levels), Hemisphere (2 levels), and Time (3 levels).

The large total number of analyses might result in false positive effects [e.g., Luck & Gaspelin, 2017], and to date no exact method of correction exists. As a first approximation, we regarded the obtained Principal Components (PCs) as independent repetitions. Because in each experiment we analyzed 6 PCs (see below), we shall generally report effects as "statistically significant" when $p < 0.05/6 = 0.0083$. However, the effects with $.05 > p > .0083$ will also be reported if they are repeatedly presented in similar conditions.

Experiment I

Participants were seven males and 13 females, aged 22 to 29 (mean 25.6).

Three kinds of harmonic tones were presented binaurally by means of pneumatic earphones (3M E-A-RTONE). One of them (standard, consisting of the frequencies 330, 660, 1320, 2640, and 5280 Hz), was presented 280 times, two other tones (deviant 1, consisting of 200, 400, 800, 1600, and 3200 Hz; and deviant 2, consisting of 130, 260, 520, 1040, and 2080 Hz) were presented 60 times each. The order of presentation was random except that one and the same rare tone could not be delivered more than two times in a row. Tone duration was 200 ms with stimulus-onset asynchrony (onset-to-onset) varying between 950 and 1050 ms. Tone intensity was kept about 70 dB above the average threshold. Preliminary testing with eight healthy individuals showed that none of them had any difficulty to distinguish between the tones.

To explore the hypothesized changes with time, the responses were averaged separately for the first, second, and third thirds of the whole sequence of 400 stimuli. Each average included at least 18 (usually 20) deviants of each kind. The three levels of the factor Time will be referred to as T1 (stimuli 1 – 133), T2 (stimuli 134 – 266), and T3 (stimuli 267 – 400).

Experiment II

Experiments II to IV each entailed two phases: Acquisition and Test. The Test phase was a three-stimulus oddball identical to that employed in Experiment I, but the preceding Acquisition phase varied. Only the results of the Test phase were analyzed in the present study. That is, ERPs in Experiments I to IV were obtained in response to the same stimulation, but after a different pre-stimulation.

23 subjects (14 females) aged 19 to 33 (mean 25.2) participated in Experiment II. The same sample but one subject was used in the Experiment V.

The three tones (chords) used in Experiment II were identical to those in Experiment I. In the Acquisition phase chord 1 was pared 21 times with the own name of the corresponding participant (SON), chord 2 was randomly paired 21 times with three other familiar names (OFN), and chord 3 was presented 21 times without any relation to other stimuli. The order of presentation was randomized but neither chord 1 nor chord 2 was allowed to appear more than 3 times in a row. The SOA within a pair chord-name was 300 ms. The SOA after a pair was 1700-1800 ms, and after chord 3 (that was not accompanied by any word) the SOA was 1150-1250 ms. The average duration of the own name and the other names was 669 ms (SD = 9 ms) and 676 ms (SD = 12 ms) respectively ($t = 0.78$, $p = .44$). Other names originated from the same pool of the most frequent German names used for each subject's own name, and always contained the same number of syllables as the own name. In the following Test phase Chord 3 was presented as standard, and chords 1 and 2, as two deviants, exactly as in Experiment I.

Experiment III

Twenty one subjects took part in this experiment (12 females, aged 22 – 29, mean 25.7). The design was identical to Experiment II. However, the words presented during Acquisition were made completely unrecognizable at the same time preserving all auditory features that SON and OFN had in Experiment II. Note that a simple reversion of the audio track would not be enough for this sake because correctly pronounced and inverted names have quite different dependence of intensity on time. Therefore, a

more complex masking procedure was employed. First, the first 25% of time points of an original name were multiplied by a linearly spaced vector of coefficients from 1.5 to 0, and the remaining 75% points were set to 0. Second, the first 25% of time points of the same name played backwards were multiplied by a linearly spaced vector of coefficients from 0 to 1.5, and the last 75% time points remained unchanged. Third, the two files were added. The processing was done in MatLab. A pilot experiment with 40 medical students (not participating in the EEG experiments) revealed than none of them was able to recognize the presented words, including each participant' masked own name.

Experiment IV

Participants were 13 females and 9 males, aged 22 – 29, mean 25.7. All of them except one were also included in the analysis of the preceding experiment. The experiment was similar to Experiment II with three differences. First, different tones were used. Chord 1 (associated with SON) contained the harmonic components of 300, 600, 120, 2400, and 4800 Hz; chord 2 (associated with OFN) contained the harmonic of 195, 390, 780, 1560, and 3120 Hz; and chord 3 contained the components of 495, 990, 1980, 3960, and 7920 Hz. Second, chords 1 and 2 were presented monaurally, one to the left ear and the other one to the right ear, with the side of presentation being counterbalanced among participants. Third, the test phase included partial reinforcement. Of the 60 presentation of chord 1 in the test phase, 9 were paired with SON. Likewise, chord 2 was nine times paired with OFN. The partial reinforcement aimed at the refreshment of the association between tones and names.

Experiment V

The experiment was, in fact, designed for a different purpose (investigation of affective conditioning), but included into the present report for a simple reason. Experiments I to IV all employed three stimuli and the similarity of their results might be attributed to this fact. In Experiment V, however, ERP were recorded to two stimuli, and we wondered whether the same N3 effects would be obtained in this

condition too. Also Experiment V entailed an Acquisition phase and a Test phase. Five negative and 5 positive emotional exclamations were used. These stimuli and their effect on EEG and magnetoencephalographic (MEG) responses were reported in Kotchoubey et al. [2003], Bostanov and Kotchoubey [2004], and Kotchoubey et al. [2009]. Emotionally positive and negative exclamations did not differ from each other in terms of the average duration and fundamental frequency (F0), and their average loudness was equalized as well. The Acquisition phase consisted of 50 presentations of chord 1 paired with five positive exclamations (10 times each exclamation) and 50 presentations of chord 2 paired with five negative exclamations (10 times each). The two chords were 440 + 880 + 1760 + 3520 + 7040 Hz and 150 + 300 + 600 + 1200 + 2400 Hz, and their linkage to positive and negative emotional exclamations was counterbalanced among participants. Tone duration was 200 ms, SOA between the tone and the exclamation 300 ms, and SOA between pairs randomly varied from 2000 to 2280 ms. The following Test phase was an oddball paradigm: one of the chords presented during Acquisition was randomly selected as the standard (presented 300 times), and the other one, as the deviant (presented 60 times). Emotional exclamations were not presented in the Test phase anymore. SOA in the Test phase randomly varied between 950 and 1050 ms. The order of presentation was the same as in test phases of the preceding experiments.

Results

Although there were no significant differences between post-experimental reports, one tendency can be mentioned. After Experiment IV, all participants correctly reported that they had heard three different sounds, and that at the beginning one of the sounds had been linked with their own name, and another one with several different names. Likewise, participants of Experiment V correctly reported that they

had heard two tones, originally related to different affective exclamations. Reports after Experiments II and III were less definite. Many participants reported having heard “two or three” different sounds. The number of such reports was yet larger after Experiment I. This observation agrees with the fact that two participants fell asleep during Experiment I (Non-REM stage 2), and their data were excluded.

The PCA conducted across the four very similar test phases of Experiments I to IV showed that the first eight principal components (PCs) with eigenvalues > 1 revealed close similarity with visually detected ERP peaks (see Figure 3 for scalp distribution of the PCs, Figure 6 for their dynamics in the regions of their best expression). Particularly, PC1 with the peak latency of 93 ms was interpreted as N1; PC2 (412 ms), as an earlier peak of N3; PC3 (294 ms), as N2b, a negative peak between P3a and P3b; PC4 (456 ms), as a second subcomponent of N3; PC5 (215 ms), as P3a; PC6 (138 ms), as P2; and PC7 (334 ms), as P3b.

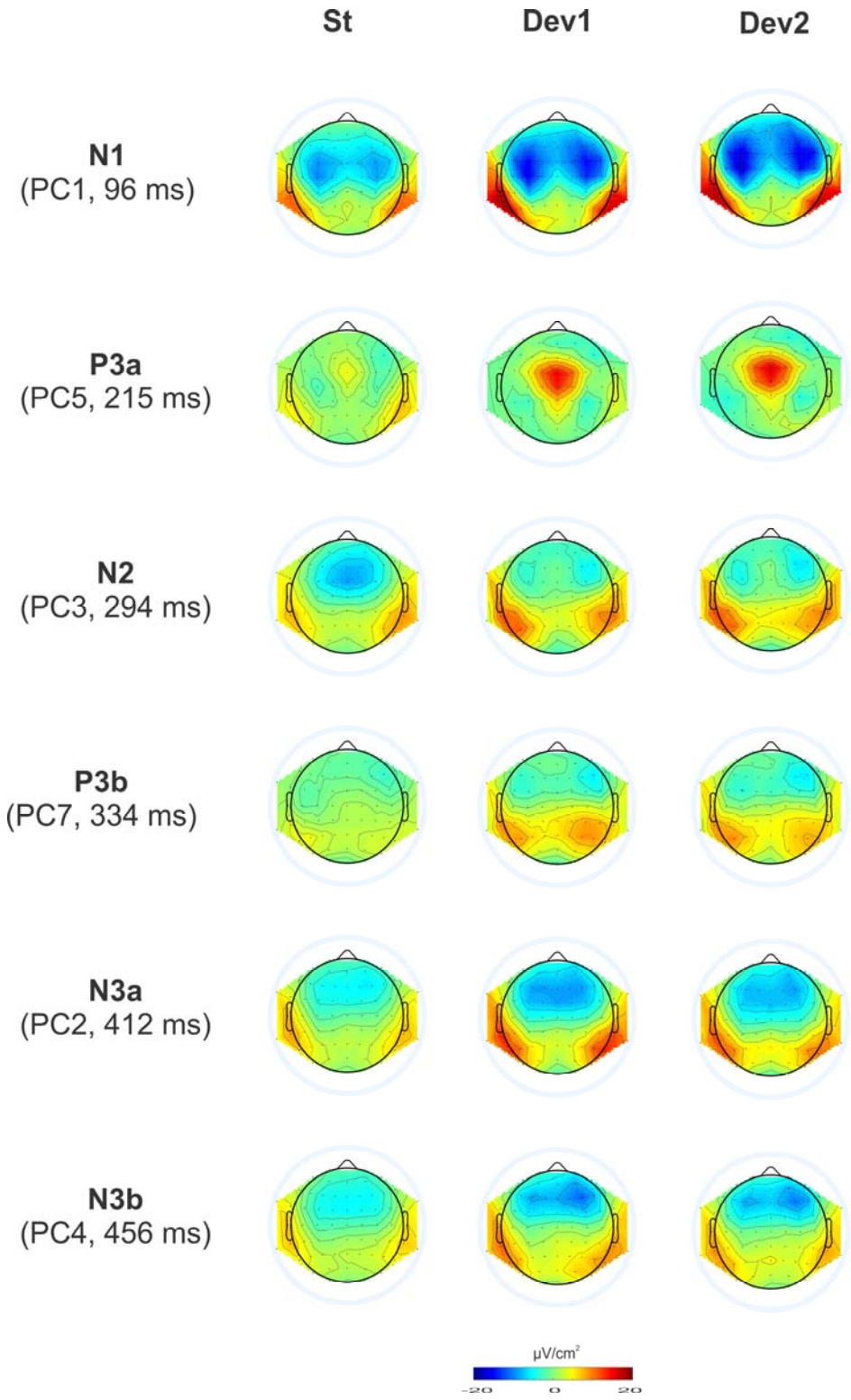

Figure 3. Topographic representation of each principal component at their peak latency for Experiments I-IV.

Because we had no prediction about the dynamics of P2, and to avoid unnecessary increase of the number of analyses, we did not analyze P2. Other components will be reported below with different number of details: (A) The N3 component will be reported in all details because, firstly, it is the main object of the study and, secondly, it has not been extensively studied in the literature, see above. (B) Other negative components N1 and N2b were analyzed not for their own sake but to compare them with N3. Finally, (C), the positive components P3a and P3b are, in contrast to N3, very well investigated in various kinds of oddball paradigm. Their analysis in the current study aimed solely at replication of already known effects.

ERP waveforms before PCA are shown in Figures 4 and 5.

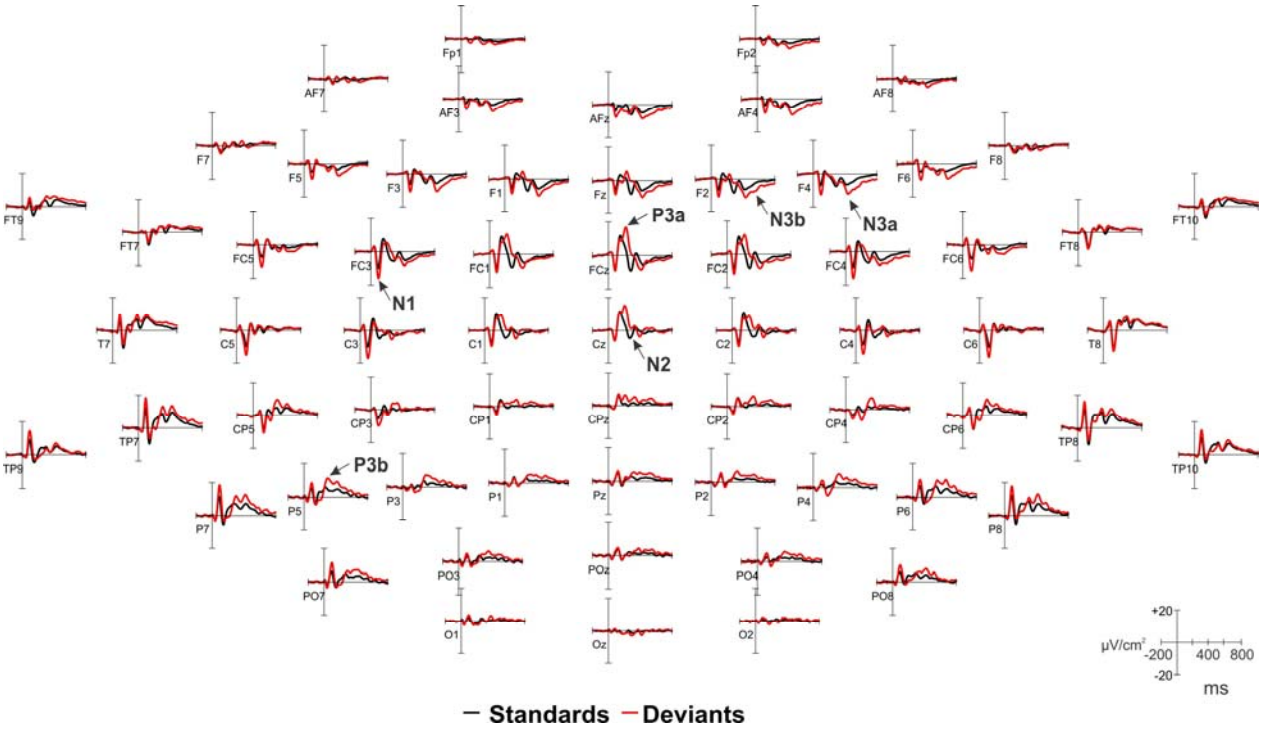

Figure 4. CSD ERP waveforms averaged across all experiments (black line, standard; red line, deviant) show the selected components that correspond to the principal components shown in Figure 2. In Experiments I to IV, in which two deviants were used, their responses were averaged together.

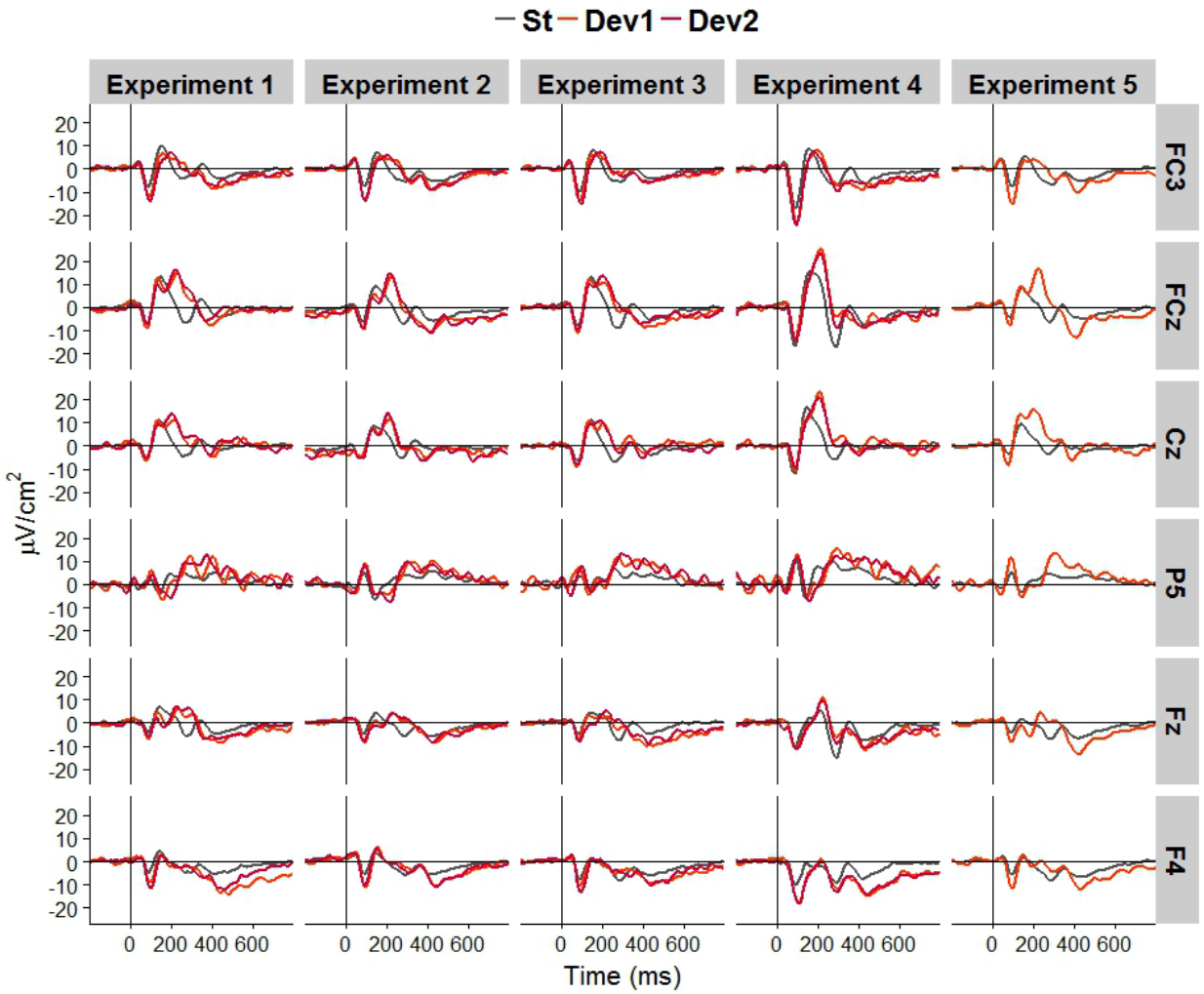

Figure 5. Average CSD ERP waveforms in each of the five experiments, at selected locations.

N3

The N3 wave, the main object of the present study, revealed in the PCA as entailing two principal components, **PC2** and **PC4**, with peak latencies of 412 and 456 ms, respectively.

Experiment I

Changes of PC2 and PC4 are illustrated in Figure 6. Both PCs were negative at frontal sites and positive at Pz (main midline Site effect: $F(3,60) = 21.87$, $\eta^2 = .52$; and 17.73, $\eta^2 = .48$, for PC2 and PC4,

respectively; both $p < .001$). The Stimulus x Site interaction was significant: $F(6,120) = 6.55$, $p = .001$, $\eta^2 = .25$, and 2.85, $p = .039$, $\eta^2 = .13$, for PC2 and PC4, respectively. The amplitude was more negative to deviants than standards at frontal sites ($F(2,40) = 6.46$, $p = .004$, $\eta^2 = .24$, 3.95, $p = .032$, $\eta^2 = .17$ for PC2 and PC4, respectively), and more positive to deviants than standards at Pz ($F(2,40) = 4.51$, $p = .023$, $\eta^2 = .18$, and 3.33, $p = .051$, $\eta^2 = .15$ for PC2 and PC4, respectively).

A similar anterior-posterior negativity-positivity slope was also significant in lateral sites (main effect of Area: $F(4,80) = 45.48$, $\eta^2 = .70$, and 48.48, $\eta^2 = .72$ for PC2 and PC4, respectively; both $p < .001$). Strong Stimulus x Area interactions ($F(8,160) = 6.55$, $\eta^2 = .25$, and 5.90, $\eta^2 = .24$ for PC2 and PC4, respectively; both $p < .001$) indicated increasing negativities to deviants over the Fro ($F(2,40) = 4.34$, $p = .021$, $\eta^2 = .18$, and 3.40, $p = .011$, $\eta^2 = .22$ for PC2 and PC4, respectively) and FC areas ($F(2,40) = 4.47$, $p = .030$, $\eta^2 = .19$, and 13.32, $p < .001$, $\eta^2 = .41$ for PC2 and PC4, respectively), as well as over the central (Ce) areas only for PC4 ($F(2,38) = 6.70$, $p = .003$, $\eta^2 = .26$). On the other hand, increasing positivities to deviants were found over TP areas ($F(2,40) = 4.34$, $p = .020$, $\eta^2 = .18$, and 10.87, $p < .001$, $\eta^2 = .26$ for PC2 and PC4, respectively). This temporoparietal positivity of PC2 increased with time (main effect of Time: $F(2,40) = 7.61$, $p = .003$, $\eta^2 = .28$). No such effect was observed for PC4.

Experiment II

At midline, the only significant effect was the negative-positive slope in the posterior direction, indicated by the main Site effect ($F(2,44) = 12.70$, $\eta^2 = .36$, and 14.53, $\eta^2 = .42$ for PC2 and PC4, respectively; both $p < .001$). In the lateral areas, strong effects of Area ($F(4,88) = 36.26$, $\eta^2 = .62$, and 48.25, $\eta^2 = .69$ for PC2 and PC4, respectively; both $p < .001$) and Stimulus x Area interactions ($F(8,176) = 5.34$, $p = .004$, $\eta^2 = .20$, and 7.08, $p < .001$, $\eta^2 = .24$ for PC2 and PC4, respectively) indicated the same differential effects of standards and deviants as in Experiment I above. However, clear local effects were observed only for PC4, i.e. larger negativities for deviants than standards over the Fro ($F(2,44) = 11.23$, p

< .001, η^2 = .34) and FC areas (F(2,44) = 8.11, p = .001, η^2 = .27), and larger positivites for deviants than standards over the TP areas (F(2,44) = 9.43, p = .001, η^2 = .30). Over the Ce areas PC4 was more negative to chord 2 than chord 1 (main Stimulus effect: F(2,44) = 6.51, p = .004, η^2 = .23; comparison chord 2 vs. chord 1: t(22) = 2.90, p = .008, η^2 = .28). Regarding PC2, only the negativity to deviants over the FC areas was significant (F(2,44) = 10.49, p < .001, η^2 = .32).

Experiment III

The main effect of Site at midline was similar to Experiments I and II (F(3,60) = 21.87, η^2 = .52, and 17.76, η^2 = .47 for PC2 and PC4, respectively; both p < .001). PC2 was again more negative to deviants than standards at frontal sites (F(2,40) = 6.46, p = .004, η^2 = .24), and this effect was inverted at Pz (F(2,40) = 4.51, p = .023, η^2 = .18). PC4 was more negative to deviants than standards at Cz (F(2,40) = 4.11, p = .024, η^2 = .17) with a similar non-significant frontal tendency (F(2,40) = 3.12, p = .057, η^2 = .14) and the lack of Stimulus effect at CPz and Pz. This resulted in significant Stimulus x Site interactions for both PC2 (F(6,120) = 6.55, p = .001, η^2 = .25) and PC4 (F(6,120) = 3.20, p = .026, η^2 = .15).

Lateral effects were identical for PC2 and PC4, i.e., the amplitudes were significantly negative (and more negative to deviants than standards) over the Fro and FC areas, and significantly positive (and more positive to deviants than standards) over the Pos and TP areas, yielding strong main effects of Area (F(4,80) = 45.48, η^2 = .70, and 28.32, η^2 = .59, for PC2 and PC4, respectively; both p < .001) and Stimulus x Area interactions (F(8,160) = 6.55, p = .001, η^2 = .25, and 4.12, p = .003, η^2 = .17 for PC2 and PC4, respectively).

Experiment IV

At midline, only the main Site effect replicated that in the previous experiments (F(3,63) = 20.45, η^2 = .49, and 36.25, η^2 = .63 for PC2 and PC4, respectively; both p < .001), but Stimulus x Site interactions

were weak and did not attain the corrected significance level of .0083. FC2 revealed, in addition, a significant Site x Time interaction ($F(6,126) = 4.38$, $p = .003$, $\eta^2 = .17$), indicating the increasing positivity at Pz at T3 as compared with T1 and T2 (main Time effect at Pz: $F(2,42) = 3.46$, $p = .038$, $\eta^2 = .15$). Interestingly, the Stimulus x Site interaction for PC2 was significant when only two deviant stimuli were compared ($F(3,63) = 6.09$, $p = .006$, $\eta^2 = .26$) indicating a significantly more negative frontal amplitude to chord 2 than chord 1 ($t(21) = 2.57$, $p = .018$, $\eta^2 = .24$).

In the lateral areas the effects of Area ($F(4,84) = 39.54$, $\eta^2 = .65$, and 45.84, $\eta^2 = .69$, for PC2 and PC4, respectively; both $p < .001$) and Stimulus x Area interactions ($F(8,168) = 5.09$, $\eta^2 = .19$, and 5.81, $\eta^2 = .22$, for PC2 and PC4, respectively; both $p = .001$) were similar to the other experiments. The negativity was larger to both kinds of deviants than to standards over the Fro ($F(2,42) = 9.87$, $\eta^2 = .32$, and 12.40, $\eta^2 = .37$, for PC2 and PC4, respectively; both $p < .001$) and FC areas ($F(2,42) = 11.59$, $p < .001$, $\eta^2 = .36$, and 5.90, $p = .006$, $\eta^2 = .22$ for PC2 and PC4, respectively). Contrary to the other experiments, the Pos and TP positivities did not significantly differ between standards and deviants.

The main lateral effect of Stimulus for PC2 ($F(2,42) = 4.41$, $p = .018$, $\eta^2 = .17$) resulted from a larger FC negativity to chord 2 as compared with chord 1 ($F(1,21) = 7.22$, $p = .014$, $\eta^2 = .26$) and a larger TP positivity to chord 2 as compared with chord 1 at T1 (Stimulus x Time: $F(2,42) = 3.62$, $p = .036$, $\eta^2 = .15$). Note, however, that these effects were “significant” only with the *uncorrected* significance level.

PC1/N1

At midline, this component was characterized by a frontal maximum (main effect of Site in Experiment I: $F(3,57) = 5.99$, $p = .006$, $\eta^2 = .24$; Experiment II: $F(3,66) = 6.70$, $p = .006$, $\eta^2 = .23$; Experiment III: $F(3,60) = 8.54$, $p = .004$, $\eta^2 = .30$; Experiment IV: $F(3,63) = 34.17$, $p < .001$, $\eta^2 = .62$). Only in Experiment III there was also a main effects of Stimulus ($F(2,40) = 5.87$, $p = .006$, $\eta^2 = .23$) with a significantly larger

negativity to chord 1 (previously linked with SON) than chord 2 (previously linked with OFN), and an intermediate amplitude to the standard stimulus.

The analyses of lateral amplitudes were characterized by strong regional differences with positive amplitudes over TP areas and negative amplitudes in all other areas (main effect of Area in Experiment I: $F(4,76) = 28.85$, $\eta^2 = .60$; Experiment II: $F(4,88) = 38.78$, $\eta^2 = .64$; Experiment III: $F(4,80) = 24.65$, $\eta^2 = .55$; Experiment IV: $F(4,84) = 59.46$, $\eta^2 = .74$; all $p < .001$) and more negative amplitudes in response to deviants than standards (main effect of Stimulus in Experiment I: $F(4,76) = 69.35$, $\eta^2 = .79$; Experiment II: $F(4,88) = 33.72$, $\eta^2 = .61$; Experiment III: $F(4,80) = 31.20$, $\eta^2 = .61$; Experiment IV: $F(4,84) = 28.25$, $\eta^2 = .74$; all $p < .001$). This latter general effect was, however, inverted over TP areas where the amplitudes were more positive to deviants than standards, resulting in significant Stimulus x Area interactions (Experiment I: $F(8,152) = 9.78$, $\eta^2 = .34$; Experiment II: $F(8,176) = 11.48$, $\eta^2 = .34$; Experiment III: $F(8,160) = 10.07$, $\eta^2 = .34$; Experiment IV: $F(8,168) = 6.48$, $\eta^2 = .24$; all $p < .001$). The anterior difference (i.e., larger negativity to deviants) was significant in Fro (Experiment I: $F(2,38) = 16.45$, $p < .001$, $\eta^2 = .46$; Experiment II: $F(2,44) = 16.53$, $p < .001$, $\eta^2 = .43$; Experiment III: F(1,20 = 7.44, $p = .013$, $\eta^2 = .27$; Experiment IV: $F(2,42) = 19.21$, $p < .001$, $\eta^2 = .48$), FC (Experiment I: $F(2,38) = 37.82$, $\eta^2 = .67$; Experiment II: $F(2,44) = 20.95$, $\eta^2 = .49$; Experiment IV: F(2,42) = = 13.42, $\eta^2 = .39$; all $p < .001$, but no effect in Experiment III), and Ce areas (Experiment I: $F(2,38) = 20.00$, $\eta^2 = .51$; Experiment II: $F(2,44) = 31.19$, $\eta^2 = .59$; Experiment IV: $F(2,42) = 11.03$, $\eta^2 = .34$; all $p < .001$; and in Experiment III at T2 and T3: Stimulus x Time interaction with $F(2,40) = 8.38$, $p = .001$, $\eta^2 = .30$). The temporoparietal inversion of this effect was significant in Experiment I ($F(2,38) = 9.63$, $p < .001$, $\eta^2 = .34$), Experiment II ($F(2,44) = 11.27$, $p < .001$, $\eta^2 = .34$), and Experiment IV ($F(2,42) = 4.80$, $p = .021$, $\eta^2 = .19$), but not in Experiment III.

The interaction between Area and Hemisphere was significant only in Experiment II ($F(4,88) = 4.53$, $p = .007$, $\eta^2 = .17$). Over posterior areas, where no stimulus effect was found, the amplitudes were more

negative on the left than right side ($t(22) = 3.56$, $p < .001$, $\eta^2 = .47$), while the opposite hold true for TP areas ($t(22) = 2.91$, $p = .008$, $\eta^2 = .40$).

PC3/N2b

This component, peaking between P3a and P3b, was generally characterized by positive values in response to deviants but negative to standards, resulting in a main effect of Stimulus at midline (Experiment I: $F(2,38) = 21.63$, $p < .001$, $\eta^2 = .53$; Experiment III: $F(2,40) = 5.79$, $p = .009$, $\eta^2 = .23$; Experiment IV: $F(2,42) = 8.98$, $p = .001$, $\eta^2 = .30$). In Experiment II this effect was modified by Time (Stimulus x Time: $F(4,88) = 4.27$, $p = .008$, $\eta^2 = .16$), as chord 2 elicited positive PC3/N2b values at T3 like the standard.

The amplitudes were maximal (negative) over the frontal cortex and positive at CPz and Pz, yielding a main effect of Site (Experiment II: $F(3,66) = 3.89$, $p = .028$, $\eta^2 = .15$; Experiment III: $F(3,60) = 4.98$, $p = .010$, $\eta^2 = .20$; Experiment IV: $F(3,63) = 19.86$, $p < .001$, $\eta^2 = .46$). In Experiment IV, in addition the effects of Stimulus and Site significantly interacted as shown in Figure 6 ($F(6,126) = 6.03$, $p < .001$, $\eta^2 = .22$).

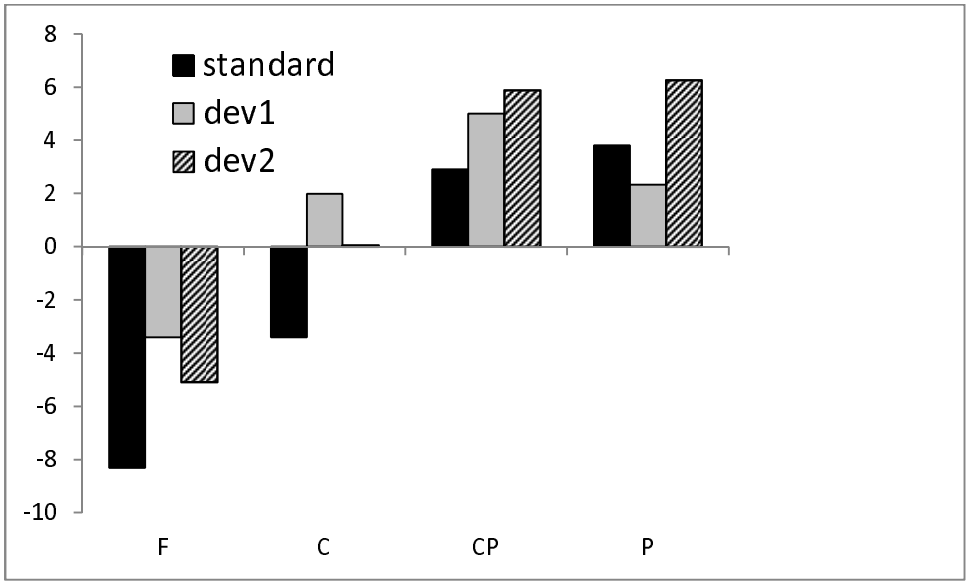

Figure 6. Stimulus x Site interaction for N2b in Experiment IV. Amplitudes in $\mu V^2/cm$.

Also at the lateral sites the same effects of Stimulus (i.e., positivity to deviants, negativity to standards: $F(2,38) = 24.50$, $p < .001$, $\eta^2 = .56$; $F(2,44) = 20.90$, $p < .001$, $\eta^2 = .49$; $F(2,40) = 27.21$, $p < .001$, $\eta^2 = .58$; and $F(2,42) = 6.18$, $p = .005$, $\eta^2 = .22$, for Experiments I, II, III, and IV, respectively) and topographic differences (i.e., significantly positive amplitudes in the Pos and TP areas, and slightly negative amplitudes in the Fro and FC areas: $F(4,76) = 10.12$, $\eta^2 = .35$; $F(4,88) = 27.79$, $\eta^2 = .56$; $F(4,80) = 17.38$, $\eta^2 = .46$; and $F(4,84) = 37.84$, $\eta^2 = .64$, for Experiments I, II, III, and IV, respectively; all $p \leq .001$) were found. In Experiments I and III, Fro and FC negativities significantly decreased with Time (main effect of Time: $F(4,76) = 10.12$, $p = .001$, $\eta^2 = .35$; and $F(2,40) = 4.48$, $p = .020$, $\eta^2 = .18$, for Experiments I and III, respectively).

Positive components

Oddball conditions are typically characterized by increased late positive components P3a and P3b, which, on the basis of the present PCA, were identified as PC5 and PC7, respectively (see Figure 1).

PC5/P3a was consistently larger to deviants than standards (main Stimulus effect in Experiment I: $F(2,38) = 8.86$, $p = .002$, $\eta^2 = .32$; Experiment II: $F(2,44) = 15.83$, $p < .001$, $\eta^2 = .42$; Experiment III: $F(2,40) = 20.22$, $p < .001$, $\eta^2 = .50$; Experiment IV: $F(2,42) = 37.80$, $p < .001$, $\eta^2 = .60$). This difference always had its maximum at Cz and minimum at Pz (Stimulus x Site interaction in Experiment I: $F(6,114) = 6.06$, $p = .004$, $\eta^2 = .24$; Experiment II: $F(6,132) = 3.77$, $p = .020$, $\eta^2 = .15$; Experiment III: $F(6,120) = 5.17$, $p = .001$, $\eta^2 = .21$; Experiment IV: $F(6,126) = 9.39$, $p < .001$, $\eta^2 = .31$). Moreover, the amplitude was generally largest at Cz, although the main effect of Site attained significance only in Experiments I ($F(3,57) = 4.53$, $p = .025$, $\eta^2 = .19$) and II ($F(2,44) = 9.57$, $p < .001$, $\eta^2 = .30$).

Further, the amplitude of PC5/P3a habituated from T1 to T2 and T3 (i.e., main effect of Time) in Experiment II ($F(2,44) = 3.88$, $p = .049$, $\eta^2 = .15$), III ($F(2,40) = 5.52$, $p = .009$, $\eta^2 = .22$), and IV ($F(2,42) = 10.55$, $p < .001$, $\eta^2 = .33$).

The most consistent effect over the **lateral areas** was the Stimulus x Area interaction (Experiment I: $F(8,152) = 6.28$, $\eta^2 = .25$; Experiment II: $F(8,146) = 14.13$, $\eta^2 = .39$; Experiment III: $F(8,160) = 6.70$, $\eta^2 = .25$; Experiment IV: $F(8,168) = 14.06$, $\eta^2 = .40$; all $p \leq .001$). The positivity to standards increased in the posterior direction and reached its maximum in TP areas. The positivity to deviants was, in contrast, maximal in FC areas and minimal in TP areas. The main effect of Stimulus (larger positivity to deviants) was significant only in Experiment II ($F(2,44) = 6.43$, $p = .008$, $\eta^2 = .23$). In TP areas the amplitude (particularly in response to standards) was more positive on the right than left side, which manifested itself in the interaction between Area and Hemisphere (Experiment II: $F(4,76) = 3.33$, $p = .029$, $\eta^2 = .15$; Experiment III: $F(4,80) = 4.68$, $p = .008$, $\eta^2 = .19$; Experiment IV: $F(4,84) = 3.31$, $p = .026$, $\eta^2 = .14$).

PC7/P3b at midline increased from frontal to parietal sites in Experiments III ($F(3,60) = 9.44$, $p < .001$, $\eta^2 = .33$) and IV ($F(3,63) = 9.81$, $p = .001$, $\eta^2 = .32$). At Pz, the amplitude was larger to deviants than standards (Stimulus x Site interaction in Experiment II: $F(6,132) = 3.36$, $p = .020$, $\eta^2 = .13$; and Experiment III: $F(6,120) = 2.92$, $p = .037$, $\eta^2 = .13$). At T2 and T3 chord 2 (previously linked with OFN) elicited larger responses than chord 1 (previously linked with SON) in Experiment IV (Stimulus x Time: $F(4,84) = 3.16$, $p = .033$, $\eta^2 = .13$; main effect of Stimulus at T3: $F(2,42) = 5.74$, $p = .007$, $\eta^2 = .22$, with the mean amplitudes of 1.47, 0.26 and 2.97 $\mu V/cm^2$ to standard, chord 1, and chord 2, respectively).

In the lateral areas, responses to both deviants were larger than to standards (main effect of Stimulus in Experiment I: $F(2,38) = 5.99$, $p = .011$, $\eta^2 = .24$; Experiment II: $F(2,44) = 12.04$, $p < .001$, $\eta^2 = .35$; Experiment III: $F(2,40) = 10.18$, $p = .001$, $\eta^2 = .34$; Experiment IV: $F(2,42) = 3.47$, $p = .048$, $\eta^2 = .14$). The maximal positive deviant-standard difference was found in Pos and TP areas, whereas in Fro and FC

areas the amplitudes were more negative to deviants than standards (main Area effect in Experiment I: $F(4,76) = 6.82$, $p = .001$, $\eta^2 = .26$; Experiment II: $F(4,88) = 17.77$, $p < .001$, $\eta^2 = .45$; Experiment III: $F(4,80) = 16.58$, $p < .001$, $\eta^2 = .45$; Experiment IV: $F(4,84) = 19.55$, $p < .001$, $\eta^2 = .48$; Stimulus x Area interaction in Experiment I: $F(8,152) = 3.75$, $p = .009$, $\eta^2 = .16$; Experiment II: $F(8,176) = 5.76$, $p = .001$, $\eta^2 = .21$; Experiment III: $F(8,160) = 3.11$, $p = .018$, $\eta^2 = .13$; Experiment IV: $F(8,168) = 6.88$, $p < .001$, $\eta^2 = .25$). Anterior negativities were larger on the right than left side (Area x Hemisphere interaction in Experiment I: $F(4,76) = 5.00$, $p = .004$, $\eta^2 = .21$; Experiment II: $F(4,88) = 3.15$, $p = .036$, $\eta^2 = .13$; Experiment III: $F(4,80) = 3.70$, $p = .016$, $\eta^2 = .16$). Finally, the positivity in the Pos and TP areas mostly decreased from T1 to T2 (main Time effect in Experiment II: $F(2,44) = 9.56$, $p < .001$, $\eta^2 = .30$; Experiment III: $F(2,40) = 3.97$, $p = .042$, $\eta^2 = .17$; Experiment IV: $F(2,42) = 4.58$, $p = .021$, $\eta^2 = .18$; Time x Area interaction in Experiment I: $F(8,152) = 3.55$, $p = .023$, $\eta^2 = .16$; Experiment II: $F(8,176) = 3.12$, $p = .013$, $\eta^2 = .12$).

Three experiments

Because Experiments I, III and IV involved largely the same participants, additional ANOVAs were performed with the data of all three experiments. The analyses included the same repeated measures factors plus the additional factor Experiment (3 levels). As regards **PC5/P3a**, both the mean component amplitude and its topographical differences were larger in Experiment IV than in the two other experiments (main effect of Experiment: $F(2,36) = 21.24$, $p < .001$, $\eta^2 = .54$; Experiment x Site interaction: $F(6,108) = 3.55$, $p = .022$, $\eta^2 = .17$). As regards **PC7/P3b**, there were no differences between the experiments at midline. However, the topographical differences between the lateral areas (i.e., Fro and FC negativities, Pos and TP positivities) were significantly smaller in Experiment I than in Experiments III and IV (Experiment x Area: $F(8,144) = 4.33$, $p = .003$, $\eta^2 = .19$).

The analysis of **N3** revealed only one interaction that approached (though not reached) the corrected significance level of .0083. The interaction resulted from topographic differences for PC4 being larger in

Experiment IV than in Experiments I and III (Experiment x Area interaction for lateral electrodes: $F(8,144) = 3.43$, $p = .009$, $\eta^2 = .16$). Figure 7 illustrates the comparative dynamics of different ERP components at the sites of their maximal expression.

Comparison between N1 and N3

Given the apparent similarities in the distribution of N1 and N3, we conducted additional ANOVAs to find differences between these negative components. These analyses were similar to those reported above but included an additional factor Principal Component (PC) having three levels (PC1, PC2, and PC4). Since absolute differences in magnitude between the components are not very informative, only interactions of PC with other factors will be reported.

The results were almost identical in all experiments, as shown in Figure 7. Highly significant interactions in lateral areas were obtained for the factors PC and Stimulus (Experiment I: $F(4,76) = 8.09$, $\eta^2 = .30$; Experiment II: $F(4,88) = 13.57$, $\eta^2 = .38$; Experiment III: $F(4,80) = 12.81$, $\eta^2 = .39$; Experiment IV: $F(4,84) = 8.69$, $\eta^2 = .29$; all $p < .001$), PC and Area (Experiment I: $F(8,152) = 5.39$, $p = .002$, $\eta^2 = .22$; Experiment II: $F(8,176) = 4.12$, $p = .022$, $\eta^2 = .16$; Experiment III: $F(8,160) = 3.71$, $p = .035$, $\eta^2 = .16$; Experiment IV: $F(8,168) = 22.64$, $p < .001$, $\eta^2 = .52$), as well as between PC, Stimulus, and Area (Experiment I: $F(16,304) = 2.85$, $p = .002$, $\eta^2 = .13$; Experiment II: $F(16,352) = 2.45$, $p = .039$, $\eta^2 = .10$; Experiment III: $F(16,320) = 3.13$, $p = .007$, $\eta^2 = .14$; Experiment IV: $F(16,336) = 2.36$, $p = .010$, $\eta^2 = .10$). An inspection of these interactions showed that the difference between standards and deviants was significantly larger for PC1 than for PC2 and PC4. Particularly, significant PC x Stimulus interactions (all $p \leq .002$) were obtained in FC and Ce areas, where both the absolute negative values and the differences between standards and deviants were substantially larger for PC1/N1 than for N3. When PC1/N1 was excluded from the analysis (that is, the factor PC was taken with two levels), no effect of PC whatsoever approached significance.

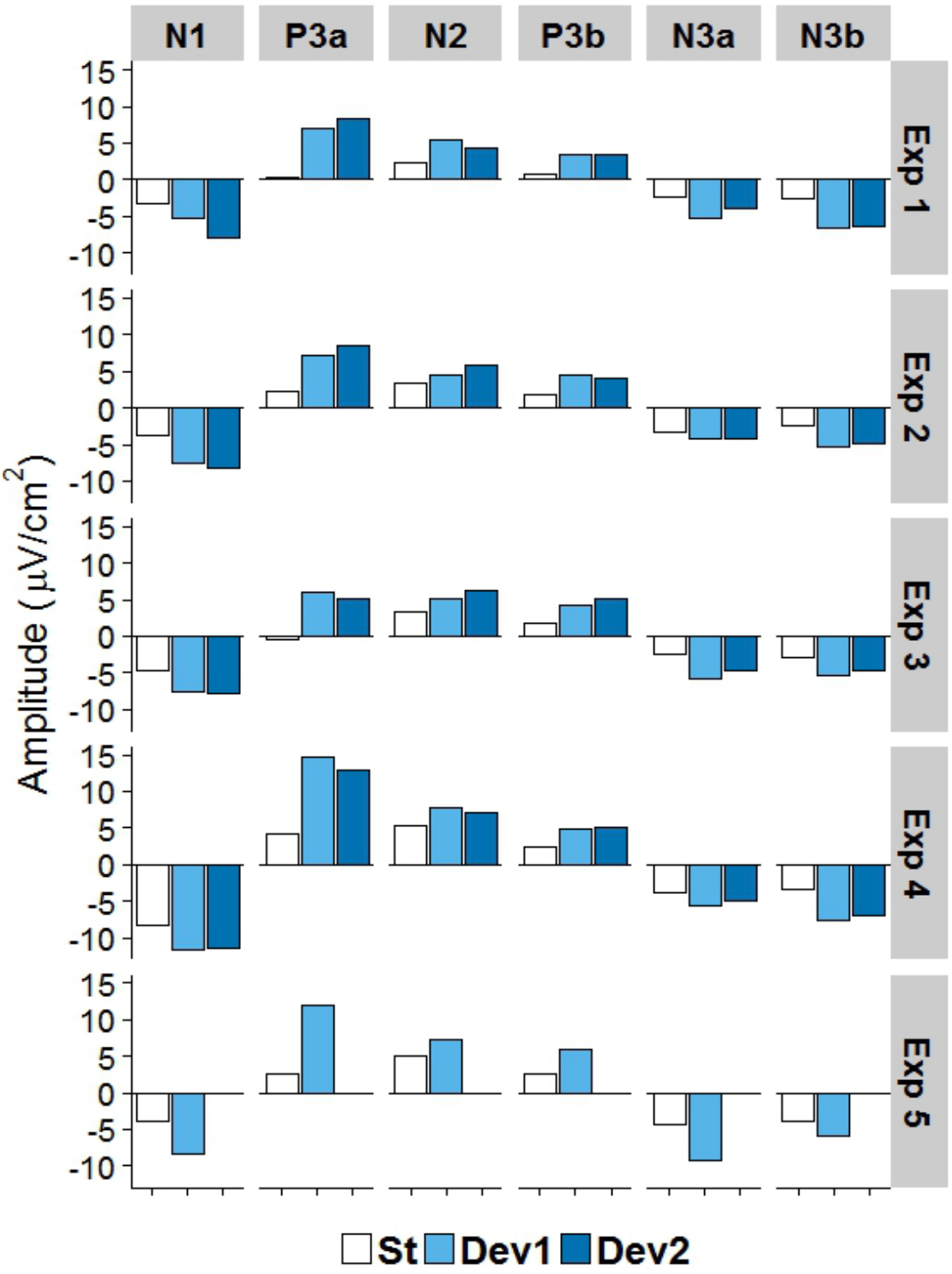

Figure 7. Mean amplitude at each ERP component in the region of its maximal expression (N1, frontocentral right; P3a, Cz; N2, temporoparietal left; P3b, temporoparietal left; N3a and N3b, frontocentral right). White bars, standard; light blue bars, Deviant 1 (or the only deviant in Experiment V); dark blue, Deviant 2.

Significant PC x Time interactions were obtained in Experiments I ($F(4,76) = 5.39$, $p = .002$, $\eta^2 = .22$) and IV ($F(4,84) = 4.31$, $p = .005$, $\eta^2 = .17$). The PC1/N1 amplitude significantly decreased with time (linear trend: $F(1,21) = 8.25$, $p = .009$, $\eta^2 = .28$; and $F(1,21) = 15.87$, $p = .001$, for Experiments I and IV, respectively), but the amplitudes of N3 did not. Finally, only Experiment IV showed a significant interaction between PC and Site *at midline* ($F(6,126) = 5.40$, $p = .001$, $\eta^2 = .20$). While all negativities were characterized by the common trend to smaller negative (or larger positive) values in the posterior direction, the exact distribution was different: the negative-to-positive inversion took place already at Cz for N3, but only at Pz for N1.

Experiment V

ERP waveforms in this experiment are presented in Figure 9. Despite numerous methodological differences between this experiment and the previous four, the PCA resulted in surprisingly similar distribution of the main components. The only major difference was the presence of PC7 (peak latency 376 ms) that could not be identified as a peak in the ERP waveform. As shown in Table I, other PCs were almost identical to those in Experiments I – IV, only their order was different.

Table I. Principal components selected in the experiments with three stimuli (Experiment I to IV) and in Experiment V with two stimuli. Note very close peak latencies of the PCs obtained with different stimulation

Peak name		N1	P2	P3a	N2b	P3b	N3early	N3late
Exp I-IV	Principal component	PC1	PC6	PC5	PC3	PC7	PC2	PC4
	Latency, ms	96	136	215	294	334	412	456
Exp V	Principal component	PC4	PC3	PC6	PC2	PC5	PC1	PC8
	Latency, ms	90	129	207	294	336	416	459

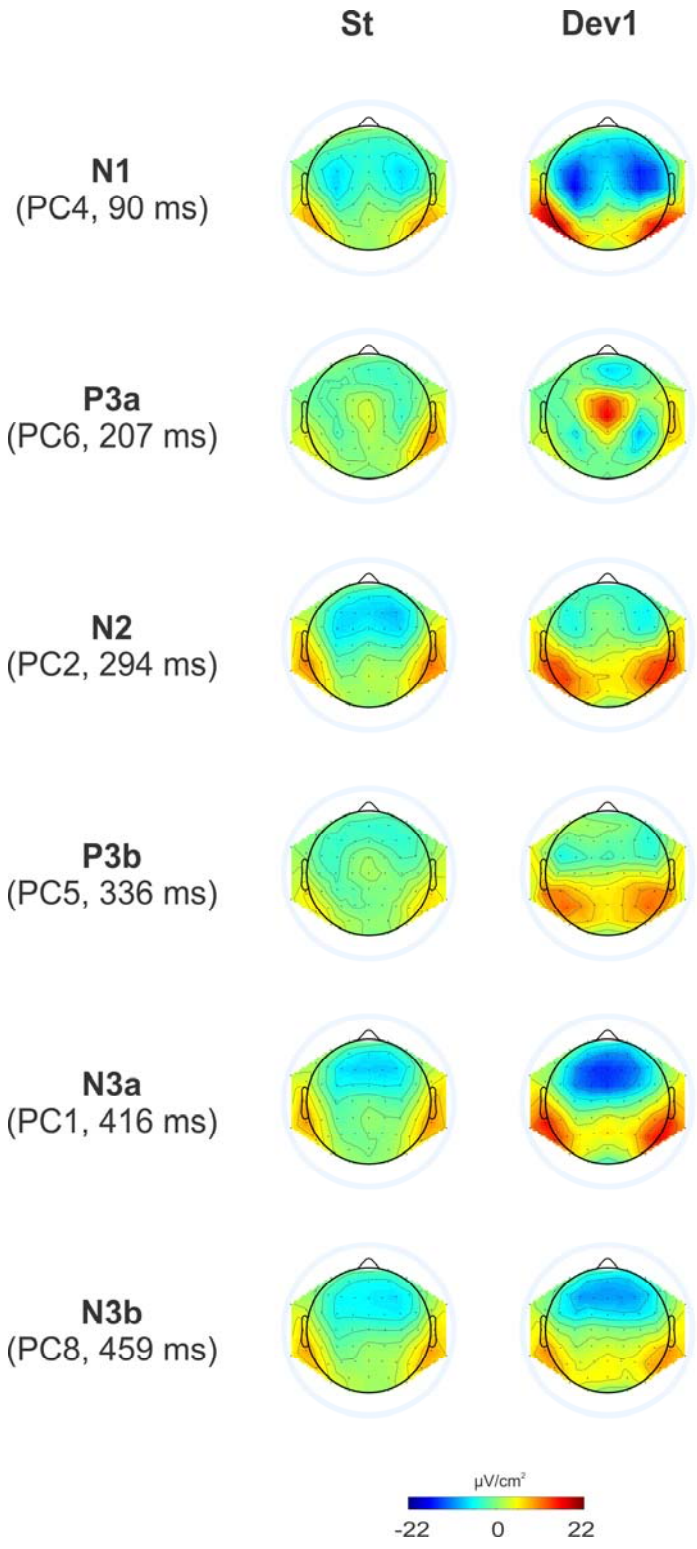

Figure 8. Topographic representation of each principal component at their peak latencies for Experiment V.

The main object of the present study, **N3**, was again represented as two subcomponents, PC1 and PC8. At midline, both were negative at frontal and central sites but positive at Pz (main Site effect: $F(3,63) = 42.94$, $\eta^2 = .67$, and $F(3,63) = 44.29$, $\eta^2 = .68$, for PC1 and PC8, respectively; both $p < .001$). Both fronto-central negativities and parietal positivities were about twice larger to deviants than to standards (Stimulus x Site interactions: $F(3,63) = 15.39$, $p < .001$, $\eta^2 = .42$, and $F(3,63) = 5.98$, $p = .004$, $\eta^2 = .22$, for PC1 and PC8, respectively). The analysis over lateral sites revealed negative amplitudes in Fro and FC areas, and positive amplitudes in Pos and TP areas (main effect of Area: $F(4,84) = 66.25$, $\eta^2 = .76$, and $F(4,84) = 59.92$, $\eta^2 = .74$, for PC1 and PC8, respectively; both $p < .001$). The topographical differences were larger for deviants than standards (Table II), resulting in highly significant Stimulus x Area interactions: $F(4,84) = 21.88$, $\eta^2 = .51$, and $F(4,84) = 7.24$, $\eta^2 = .26$, for PC1 and PC8, respectively; both $p < .001$). The amplitude of PC1 (but not PC8) appeared to decrease with time, particularly at frontal sites on midline and lateral Fro and FC areas, but neither the main effect of Time, nor the Time x Site and Time x Area interaction attained the corrected significance level.

Table II. Anterior-posterior differences in the amplitudes (in $\mu V/cm^2$) of two Principal Components (PCs) reflecting N3. Fro – frontal, TP – temporoparietal. "Lateral" means averaged values for the left and right hemispheres, because the factor Hemisphere was not significant.

		Fro midline	Pz	Fro lateral	TP lateral
PC1	standard	-2.9	-9.8	-4.2	-11.1
	deviant	1	4.8	4.7	4.4
PC8	standard	-2.7	-5.3	-4.8	-6
	deviant	1.9	3.7	2.9	5.1

PC4/N1 had its midline maximum at frontal and fronto-central sites with a decreasing amplitude toward Pz ($F(3,63) = 18.50$, $p < .001$, $\eta^2 = .47$). The amplitude to deviants was larger than to standards at all sites except Pz, resulting in a significant interaction between Stimulus and Site ($F(3,63) = 7.04$, $p = .003$, $\eta^2 = .25$). At lateral sites, the amplitude was negative (and more negative to deviants than standards) in the Fro and FC areas, and positive (and more positive to deviants than standards) in the TP areas, leading to strong main effects of Area ($F(4,84) = 50.00$, $p < .001$, $\eta^2 = .70$), Stimulus ($F(1,21) = 73.22$, $p < .001$, $\eta^2 = .78$; mean amplitudes -1.25 ± 0.21 $\mu V/cm^2$ and -2.9 ± 0.32 $\mu V/cm^2$ for standards and deviants, respectively), and their interaction ($F(4,84) = 34.15$, $p < .001$, $\eta^2 = .62$).

PC2/N2b was positive related to the baseline at Cz, CPz, and Pz and close to zero frontally (main effect of Site: $F(3,63) = 9.73$, $p = .001$, $\eta^2 = .32$). At lateral sites its amplitude became more positive in the posterior direction (main effect of Area: $F(4,84) = 50.31$, $p < .001$, $\eta^2 = .71$). Also, the amplitude was generally positive to deviants and negative to standards (main effect of Stimulus at midline: $F(1,21) = 19.21$, $\eta^2 = .48$; at lateral sites: $F(1,21) = 45.47$, $\eta^2 = .68$; both $p < .001$).

Both positive components, **PC6/P3a** and **PC5/P3b**, decreased at midline with time ($F(2,42) = 3.55$, $p = .039$, $\eta^2 = .15$; and 4.23, $p = .022$, $\eta^2 = .17$, for P3a and P3b, respectively) and were more positive to deviants than standards (PC6/P3a: $F(1,21) = 23.00$, $p < .001$, $\eta^2 = .52$; PC5/P3b: $F(1,21) = 9.12$, $p =.007$, $\eta^2 = .30$). The stimulus effect on P3a was best expressed at Cz (Stimulus x Site interaction: $F(3,63) = 13.01$, $p < .001$, $\eta^2 = .39$), and the stimulus effect on P3b, at Pz (Stimulus x Site interaction did not reach significance: $F(3,63) = 3.34$, $p =.045$, $\eta^2 = .14$).

The lateral distribution of the positive components strongly differed between standards and deviants. Although both stimuli elicited negative **PC6/P3a** amplitudes in Fro areas, the maximum positivity to standard was observed in TP areas, but the maximum positivity to deviant was much more anterior, in FC areas (main Area effect: effect $F(3,63) = 6.02$, $p =. 005$, $\eta^2 = .22$; Stimulus x Area interaction: $F(3,63) =$

9.13, $p <. 001$, $\eta^2 = .30$). **PC5/P3b** amplitudes to standards were slightly negative in almost all areas (Fro, FC, Ce, Pos), but positive in TP areas, whereas PC5/P3b amplitudes to deviants were negative in FC areas, zero in Fro areas, and positive in Ce, Pos and TP areas (main Area effect: $F(3,63) = 13.97$, $p <. 001$, $\eta^2 = .38$; Stimulus x Area interaction: $F(3,63) = 6.57$, $p =. 003$, $\eta^2 = .24$). Averaged across all areas, mean amplitudes were -0.37 and +1.90 $\mu V/cm^2$ to standard and deviant, respectively (main effect of Stimulus: $F(1,21) = 35.33$, $p <. 001$, $\eta^2 = .63$).

N3 versus N1

Like in the previous experiments, we directly compared N1 with N3 in Experiment V because superficially they appear to display similar dynamics. PC1 was taken as the best representative of N3, and PC4, as the representative of N1. Thus the factor PC had 2 levels.

In the lateral ANOVA the factor PC strongly interacted with Stimulus ($F(1,21) = 30.13$, $p <. 001$, $\eta^2 = .59$) and Area ($F(4,84) = 9.09$, $p <. 001$, $\eta^2 = .30$), also yielding a three way PC x Stimulus x Area interaction ($F(4,84) = 4.71$, $p =. 006$, $\eta^2 = .18$). Generally, the difference between standards and deviants was significantly larger for PC4/N1 than for PC1/N3. The topographic distributions were different: PC4/N1 was negative in the Fro, FC, and Ce areas, zero in the Pos areas, and positive only in the TP areas, and the mean difference between the most negative Fro areas and the most positive TP areas was 8.8 $\mu V/cm^2$. PC1/N3 was negative in the Fro and FC areas, zero in the Ce areas, and positive in the Pos and TP areas, and the difference between the most negative and most positive areas was 12.3 $\mu V/cm^2$. The maximal effect of stimulus (> 4 $\mu V/cm^2$ difference between deviant and standard) was obtained in the Ce areas for PC2/N1 but in the FC and TP areas for PC1/N3.

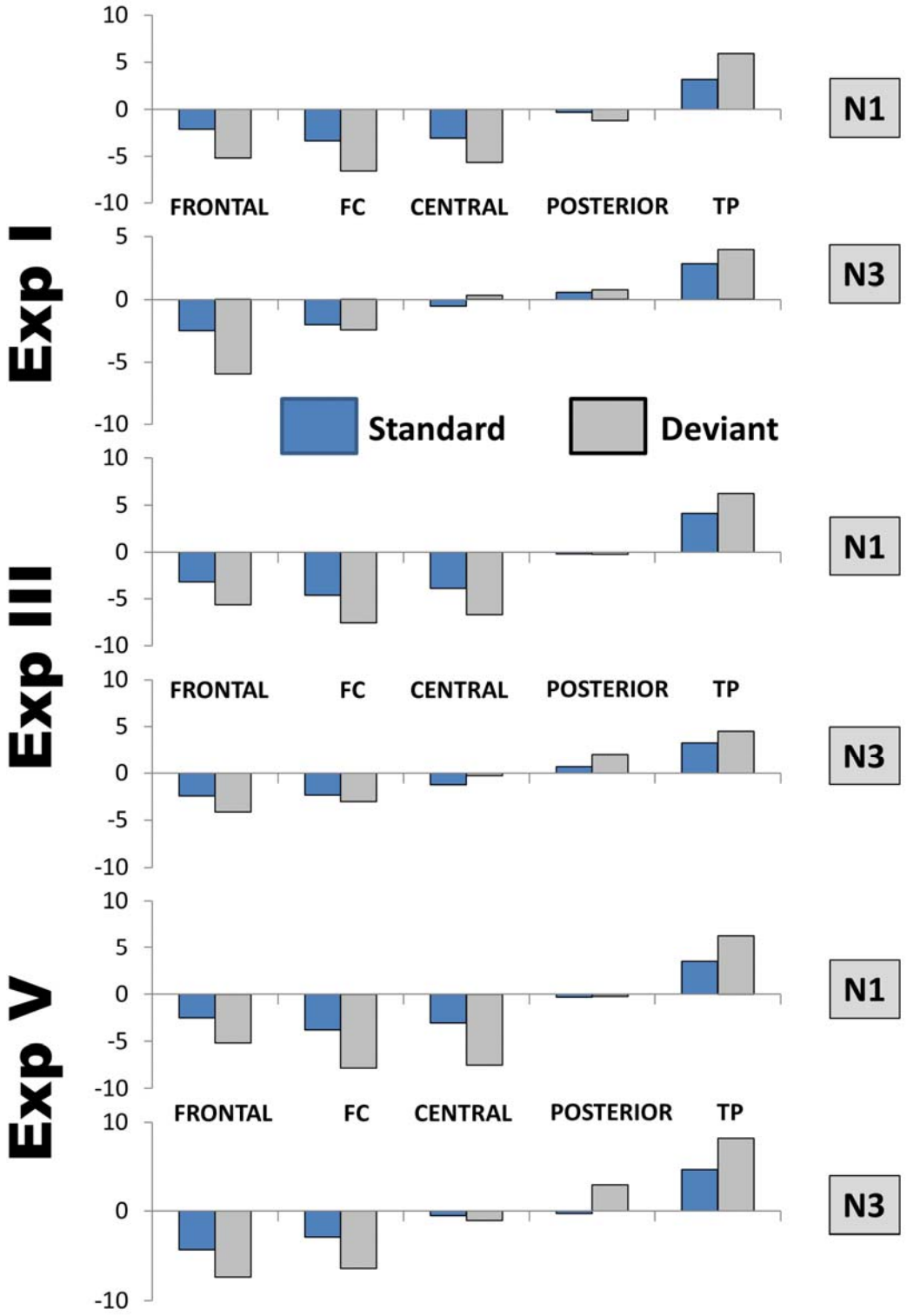

Figure 9. Comparison between the distributions of N1 and N3 over lateral areas. FC, frontocentral areas; TP, temporoparietal areas. To save space only the data of three experiments are presented, but the distributions were not qualitative different also in the other two. Left and right areas are averaged, as well as Deviant1 and Deviant 2 in Experiments with two deviants. Amplitudes are in µV/cm2.

The analysis at midline replicated the interactions with topography (PC x Site: $F(3,63) = 8.19$, $p =. 001$, $\eta^2 = .28$; PC x Stimulus x Site: $F(3,63) = 4.84$, $p =. 016$, $\eta^2 = .19$). Like at the lateral sites described above, the transition from negative to positive values happened more posteriorly for PC1/N3 than for PC4/N1, and the difference between maximal negative and maximal positive values was larger for the former than the latter (5.1 and 8.1 $\mu V/cm^2$ for PC4/N1 and PC1/N3, respectively). The largest effect of stimulus was at Cz for PC4/N1 but at frontal leads for PC1/N3. Figure 9 presents the comparison between N3 and N1 in three experiments.

For control, this analysis was repeated using PC8 instead of PC2. According to our inspection, PC8 reflected a later subcomponent of the same component N3. In fact the analysis of PC1 versus PC8 did not reveal any significant interactions of the factor PC.

Mastoid references

As reported in Methods, for comparison with literature data we also analyzed ERPs referred to averaged mastoid electrodes. As can be seen in Figure 10, principal component peaks had latencies very similar to CSD-extracted components: 96 ms for N1 (PC1), 215 ms for P3a (PC5), 334 ms for P3b (PC7), 412 and 456 ms for two subcomponents of N3 (PC6 and PC9, respectively). The main difference between CSD and mastoid referenced N3 is that in the latter case, N3 decreases in the posterior direction but does not reverse its polarity; that is, it remains negative at all electrodes.

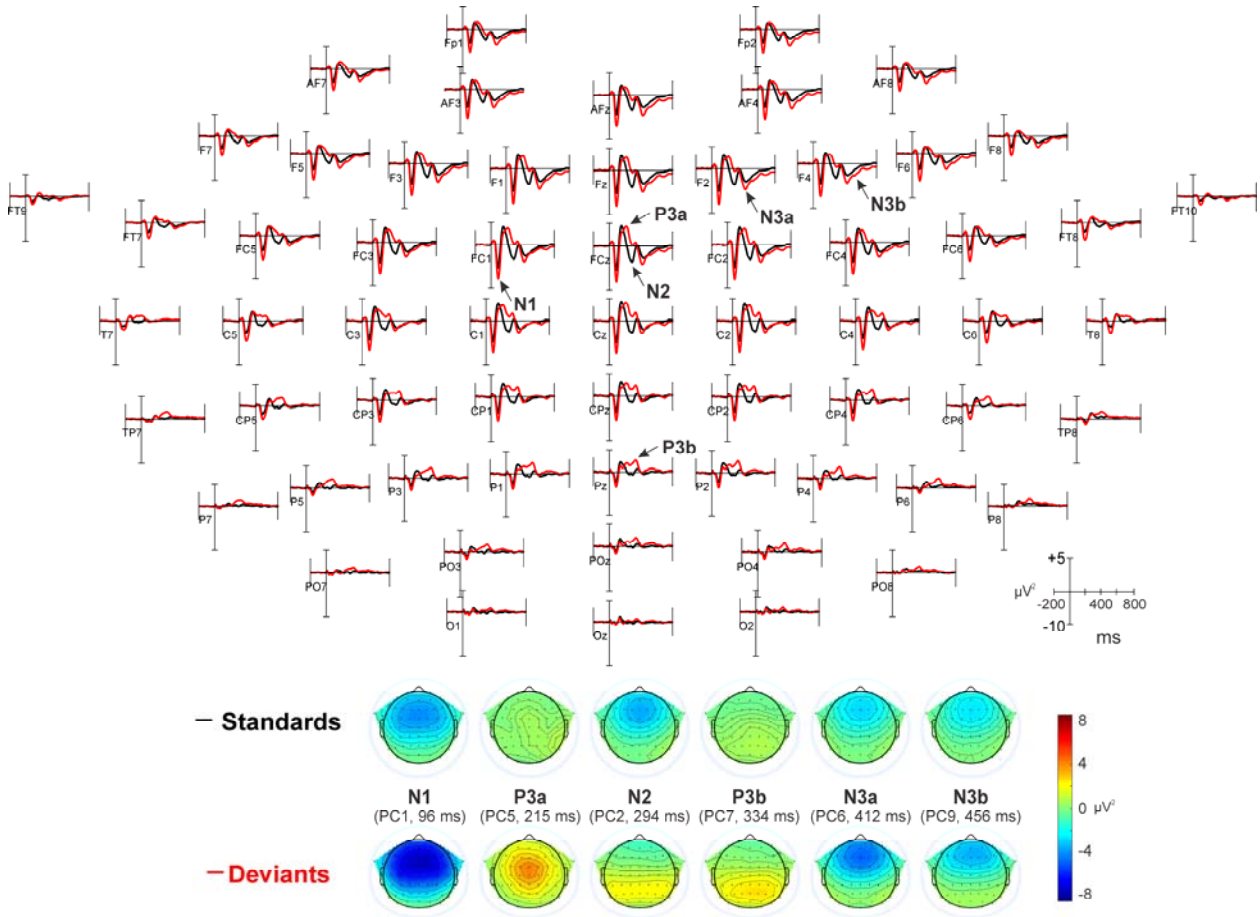

Figure 10. Top: ERP waveforms averaged for all five experiments, referred to average mastoids, for standards (black) and deviants (red). In Experiments I – IV two deviants were taken together. Most prominent peaks are indicated by arrows (P2, which did not differ between standards and deviants, is not shown). Bottom: topographical distributions for PCA data.

Discussion

To our best knowledge, this is the first systematic investigation of the N3 wave in the oddball paradigm. Being the first, it has largely explorative character. We conducted a large number of analyses, some of which with up to five factors. Such analyses can yield false positive results. We are aware both of this problem and of the fact that it still does not have a satisfactory solution. Thus we admit that some effects described in the Results section may later turn out to be spurious. Nevertheless, we have several

reasons to be convinced that at least the most important findings are reliable. First, many of them attained very high significance level such as $p \leq .001$, which would survive any reasonable correction. But the second reason for our optimism is much more important that the formal level of significance: the major effects are consistently *replicated* from experiment to experiment notwithstanding considerable differences between experimental conditions. This pleasantly surprising replicability already starts with the results of PCA. The latencies of principal components found in different experiments differed, on average, in 4.8 ms; the latencies of the components reflecting N3 (the target component of the present study) differed in 2.7 ms only. In other words, the structure of ERP was virtually identical in all experiments.

What can be said about N3 on the basis of the presented data? First, it is not an artifact. In our experiments all kinds of artifacts were carefully excluded. Participants listened to stimuli with eyes closed, which strongly diminish ocular effects. Bad channels were replaced by interpolation, non-EEG components were removed by means of the ICA, and finally, a few remaining artifact trials were excluded from averaging. Also in the literature we could find no indication of the artificial nature of N3.

Second, the PCA showed that N3 consistently entailed two subcomponents only weakly correlating with each other, with peak latencies of 412-416 and 456-459 ms, respectively. All experimental effects, however, were virtually the same on the two subcomponents and revealed only quantitative differences (mostly, the earlier subcomponent exhibited stronger effects than the later one). Both subcomponents also had the same topography.

This similarity (or even near-identity) of the earlier and the later N3 subcomponents can be compared with the significant differences between them and the other ERP negativities. Although a superficial glance might see a similar distribution of N3 and N1 (negativities in frontal areas, positivities in temporo-parietal areas), their direct comparative analysis demonstrated substantial differences. The negative-to-

positive transition was much more anterior for N3 than for N1. The maximum of frontal negativity was more lateralized for N1 than N3. The distribution of N1 CSD is compatible with a fronto-temporal sink, whereas the distribution of N3 CSD is rather in line with a midfrontal sink[1].

Third, N3 is obviously not a mismatch negativity. CSD data of the MMN demonstrate a completely different topographical distribution (see, for example, [Deouell et al., 1998, Fig. 3b; Shalgi and Deouell, 2007, Fig. 5; Kayser and Tenke, 2015, Fig. 12]). Another argument against the concept of N3 as a kind of the MMN is the fact that the number of stimuli in the present experiments was rather small. N3 could be seen even after as few deviants as 20-50, while MMN experiments usually need hundreds of deviants to demonstrate substantial effects.

Fourth, the decrease of the N3 amplitude in the posterior direction might remind on the Positive Slow Wave (PSW, e.g., [Squires et al., 1975; Sutton and Ruchkin, 1984; Dien, 1998; Spencer et al., 1999; Dien et al., 2004]) that can also have negative amplitudes in frontal leads. However, the analysis of the data with average mastoid reference (Figure 10) shows that contrary to the PSW, N3 do not change its polarity in the sagittal dimension, i.e., it remains negative also in the parietal areas. This is in line with CSD data indicating that the electrical source of the N3 dipole is not parietal but posterior temporal, in the vicinity of the mastoid electrodes. Furthermore, Kayser et al. [1998] identified N3 and the PSW as two different principal components.

Fifth, N3 does not appear to depend on the alleged significance of stimuli, at least within the range tested in this study. Tones, which elicited N3, had been previously related to participants' own names in Experiments II and IV, to emotional stimuli in Experiment V, to unconceivable sounds in Experiment III, and were completely neutral in Experiment I. No substantial differences between the results of these

[1] As all stimuli in our experiments had a duration of 200 ms, one might even ask whether N3 was an N1 of the *offset* response. But this hypothesis is, of course, wrong just because other studies (including our own [Kotchoubey and Pavlov, 2017]) used stimuli of broadly varying duration, yet the peak latency of N3, according to the corresponding figures, was always between 400 and 450 ms from stimulus *onset*.

experiments were observed, and statistical comparisons did not reveal any significant effects of the factor Experiment. Likewise, N3 did not substantially depend on the number of stimuli in the oddball experiment, as it was equally recorded in two- and three-stimuli paradigms.

Sixth, N3 demonstrated an oddball effect, being larger to deviants than standards. In contrast to N1 and P3, this effect was rather manifested as a Stimulus x Topography interaction than as a main effect of Stimulus. This means that N1 was consistently more negative, and P3a and P3b were more positive to deviants than standards, although these amplitude increases also had their local maxima for each component. N3, in contrast, was typically more negative to deviants than standards over anterior regions but more positive to deviants than standards over posterior regions.

Table III shows an overview of the presence of N3 in the previously published passive oddball reports. Only those studies are included in the Table in which ERP waveforms at least at the three midline sites (Fz, Cz, Pz) were clearly presented. We do not include the numerous MMN passive oddball experiments, because these experiments usually show only an MMN-P3a complex but no P3b. We are interested, in contrast, in a late negativity after P3b.

Table III. Experiments designated as “passive oddballs”, in which figures do or do not show an N3 following P3b.

	Study	**Stimulus modality**	**Instruction**	**N3 yes/no**	**comment**
1	Polich 1987	auditory	To daydream	No	
2	Polich 1989 Exp1	auditory	To ignore	Yes	
3	Polich 1989 Exp2	auditory	To ignore	No	Stimulus sequences were not random, i.e., deviants might be predicted
4	Polich 1989 Exp2	auditory	To solve a puzzle	No	

5	O'Donnell 1992	visual	To relax and ignore	Yes	N3 in participants from 20 to 31 years old, no N3 in older participants
6	Polich 1994	auditory	Just to listen	Yes	
7	Oades 1995a	auditory	Unclear	Yes	
8	Oades 1995b	auditory	Unclear	Yes	
9	Lang 1997	visual	to pay attention w/out a task	No	In the corresponding active experiments participants had to pay attention *and count* rare stimuli
10	Lang 1997	auditory	to pay attention w/out a task	No	
11	Bennington 1999	visual	Just to observe	No	Deviants might be predicted like in Polich 1989 above (No. 3, 4)
12	Bennington 1999	auditory	Just to listen	Yes	
13	Zenker 1999	auditory	Just to listen	Yes	Participants were children from 6 to 14, N3 observed only in the oldest group
14	Potts 2004	visual	Just to observe	Yes	
15	Holeckova 2006	auditory	Watching TV	Yes	Stimuli were own name and other names
16	Barry 2006	auditory		Yes	
17	Bostanov 2006	auditory	Just to listen	Yes	Confirmed by a time-

					frequency analysis
18	Brinkman 2008	auditory	Viewing a cartoon	Yes	N3 larger in children than adults
19	Wronka 2008	auditory	Visual task	No	Three-stimuli paradigm
20	Barry 2006	auditory		Yes	
21	Eichenlaub 2012	auditory	Viewing a video	No	Stimuli were own name and other names
22	Fischer 2014	auditory	Viewing a video	Yes	Three-stimuli paradigm, N3 in both healthy subjects and (delayed) in schizophrenic patients
23	Erlbeck 2014	auditory	Just to listen	Yes	
24	Erlbeck 2014	auditory	Visual task	No	
25	Kayser 2014	auditory	No particular task	Yes	
26	Kühnis 2014	auditory	Visual task	No	Vowels presented to musicians and non-musicians
27	Marhöfer 2014	visual	Visual distraction	No	Four-stimuli oddball (3 deviants)
28	Brown 2015	auditory	Solving a puzzle	No	
29	Brown 2015	auditory	Reading a book	No	

30	Rogenmoser 2015	auditory	Visual task	No	Auditory stimuli difficult to distinguish
31	Justen 2016	auditory	No particular task	Yes	Simple tones
32	Justen 2016	auditory	No particular task	No	Individual finger snapping sounds

References to the table: [Barry et al., 2006; Barry et al., 2009; Bennington and Pollich, 1999; Bostanov and Kotchoubey, 2006; Brinkman and Stauder, 2008; Brown et al., 2015; Eichenlaub et al., 2012; Erlbeck et al., 2014; Fisher et al., 2014; Holeckova et al., 2006; Justen and Herbert, 2016; Kayser et al., 1997; Kühnis et al., 2014; Lang et al., 1997; Marhöfer et al., 2014; O'Donnell et al., 1997; Oades et al., 1995a; Oades et al., 1995b; Polich, 1987; Polich, 1989; Polich and McIsaak, 1994; Potts, 2004; Rogenmoser et al., 2015; Wronka et al., 2008; Zenker and Barajas, 1999].

The table lists a total of thirty-two experimental conditions, N3 being observed in seventeen of them. Of these 17, the condition described as “passive” included an additional distraction task in three experiments (viewing a cartoon in [Brinkman and Stauder, 2008], viewing a video in [Holeckova et al., 2006; Fisher et al., 2014]. Of the three “exceptional” experiments, Holeckova et al. [2006] presented participants’ own names and similar names as the to-be-ignored auditory stimuli, and one may suppose that the distraction task did not completely distract participants from processing of the highly significant words. Participants of Brinkman & Stauder [2008] were adolescents, and those of Fischer et al. [2014], schizophrenic patients. However, in the latter study N3 was also observed in the control group. In the other 14 experiments showing N3, the authors implicitly [Oades et al., 1995a, b] or explicitly (the rest of

studies) indicated the lack of any task: participants should simply view visual stimuli or listen to auditory ones.

Of the 15 data sets that do *not* show an N3, active distraction was used in nine experiments. Moreover, the participants in both visual and auditory experiments of Lang et al. [1997] were instructed to actively follow stimuli, albeit without a motor task. Polich [1987] instructed his subjects to daydream, which may or may not be regarded as distraction. In his later works [Polich, 1989] a passive condition without any distraction was presented twice, and N3 was recorded in Experiment 1 but not in Experiment 2. The sequence of auditory stimuli in this study and in Bennington and Polich [1999] was not random or pseudorandom, but consisted of trains of 10 stimuli, and deviants could only appear in the second halves of the trains. This means that subjects could guess deviants more or less successfully. In an extreme case, after a series of 9 standards, the deviant could even be predicted for sure. Finally, Justen and Herbert [2016] showed an extremely large N3 in a completely passive condition with tonal stimuli, but very small (if any) in the same oddball with sounds of snapping fingers. Again, such sounds (also including snapping of a participant's own fingers) are highly idiosyncratic.

Of course, passive conditions present a principal psychological problem, because the less exact is participants' instruction, the less do we really know what they were doing during stimulation. This general difficulty is also valid for the experiments summarized in Table III. However, the above analysis results in a rough differentiation between the two kinds of conditions that can be designated as "passive-attentive", in which participants' attention was directed either to a different class of stimuli or to the presented stimuli (but without any response requirement), and "passive-inattentive", in which participants' attention was allowed to freely move around. If this classification is correct, N3 was shown in 14 "passive-inattentive" conditions and 3 "passive-attentive" conditions. No N3 was shown in 4

“passive-inattentive” conditions and 11 “passive-attentive” conditions. The resulting difference is highly significant (Fisher Exact Test: p = .0036).

This leads to a hypothesis that N3 may be a result of an “intermediate” (i.e., not too deep, not too superficial) level of stimulus processing. Generally, slow cortical negativities reflect preparatory activation of apical dendrites, and thus anticipatory “warming up” large populations of cortical neurons [Rockstroh et al., 1989; Birbaumer et al., 1990; Mitzdorf, 1991; Kotchoubey, 2006]. Because such preparation to a further activity is usually followed by the phase of the realization of the prepared potential, the vast majority of cortical ERPs can be presented as a sequence of negative-positive complexes (i.e., cycles of preparation and realization [Kotchoubey, 2006]. There are, however, exceptional cases in which a cortical response ends with a negative rather than positive phase, such as the occipital P1-N1 complex of visual ERPs or the N400 to semantically unrelated word pairs (e.g., table-mouse) presented on the background of related word pairs (e.g., cat-mouse). Such an ending negativity is usually a sign of preparation that did not find its realization [Kotchoubey, 2006].

According to this hypothesis, N3 should be elicited neither during a goal-directed activity, when the eliciting stimulus is regarded as task relevant, and the corresponding task is performed, nor in a situation of attentional distraction when the stimulus is regarded as fully irrelevant and processing resources are directed to a completely different task. In the former case, there are two options. With simple task-relevant stimuli, such as in most oddball experiments, the processing is closed with a P3b whose meaning has been a subject of long discussions [e.g., Johnson, 1986; Donchin and Coles, 1988; Verleger, 1988; Verleger et al., 1994; Kotchoubey et al., 1997]. Very complex or highly emotional stimuli elicit, beyond this, late slow wave activity that reflects sustaining processing. In the latter case again two options appear: if an irrelevant stimulus matches the current expectancies, its processing ends with the P2 component some 180-250 ms after stimulus onset; if it mismatches the expectancies, the complex of

the MMN and P3 follows. None of these four cases requires an additional negative deflection N3 after P3b.

The situation is different when the stimulus is regarded by the brain as potentially relevant, yet its supposed relevance cannot be linked to any adaptive activity. It appears to mean something, but no behavioral consequence can be inferred from this meaning. The result is processing that commences but does not come to a closure.

If this hypothesis is true, further experiments can be designed to vary the precision of the meaning of presented stimuli. A starting paradigm may be the design of Erlbeck et al. [2014] who employed an active condition (attention to the stimuli), a distraction condition (attention away from the stimuli), and a passive condition (free attention). However, future experiments on this basis should employ more gradual conditions, such as attentional tasks with high and low motivation (assuming that motivation mobilizes attentional resources), instructions to pay attention to the stimuli without any overt or covert response, or just presentation of stimuli without any instruction. Also, though Erlbeck et al.'s [2014] results *appear* to be in line with the above hypothesis (i.e., N3 appears in the passive condition only), nothing can be said with confidence because the authors were almost exclusively interested in the MMN.

Moreover, if this hypothesis is true, N3 might also be found in some *active* oddball experiments, e.g., when the experimental instruction was (intentionally or not) formulated with less precision. For example, N3 in active oddball was recorded in Polich (1989) and Kayser et al. (1998), but not in Erlbeck et al. (2014) and Brown et al. (2015). However, this hypothesis can only be tested in a systematic review of the voluminous literature about ERPs in active oddball, which goes far beyond the present study.

Acknowledgement

The study was supported by the German Research Society (Deutsche Forschungsgemeinschaft), Grant KO-1753/13.

REFERENCES

Barry RJ, Rushby JA, Smith JL, Clarke AR, Croft RJ. (2006): Dynamics of narrow-band EEG phase effects in the passive auditory oddball task. Europ J Neurosci 24:291-304.

Barry RJ, Rushby JA, Smith JL, Clarke AR, Croft RJ. (2009): Brain dynamics in the auditory oddball task as a function of stimulus intensity and task requirements. Internal J Psychophysiol 73:313-325. doi: 10.1016/j.ijpsycho.2009.05.003.

Bennington JY, Polich J. (1999): Comparison of P300 from passive and active tasks for auditory and visual stimuli. Internal J Psychophysiol 34:171-177.

Birbaumer N, Elbert T, Canavan AGM, Rockstroh B. (1990): Slow potentials of the cerebral cortex and behavior. Physiol Rev 70:1-41.

Bostanov V, Kotchoubey B. (2004): Recognition of affective prosody: Continuous wavelet measures of event-related brain potentials to emotional exclamations. Psychophysiol 41:259-268.

Bostanov V, Kotchoubey B. (2006): The t-CWT: A new ERP detection and quantification method based on the continuous wavelet transform and Student's t-statistics. Clin Neurophysiol 117:2627-2644.

Brinkman MJ, Stauder JE. (2008). The development of passive auditory novelty processing. Internat J Psychophysiol 70:33-39. doi: 10.1016/j.ijpsycho.2008.04.005.

Brown SBRE, Van der Wee NJA, Van Noorden MS, Giltay EJ, Neuwenhuis S. (2015): Noradrenergic and cholinergic modulation of late ERP responses to deviant stimuli. Psychophysiol 52:1620–1631.

Courchesne E, Hillyard SA, Courchesne R. (1975): Stimulus novelty, task relevance and the visual evoked potential in man. EEG Clin Neurophysiol 39:131-143.

Deouell LY, Bentin S, Giard MH. (1998): Mismatch negativity in dichotic listening: Evidence for interhemispheric differences and multiple generators. Psychophysiol 35:355-365.

Dien J. (2010): The ERP PCA Toolkit: An open source program for advanced statistical analysis of event-related potential data. J Neurosci Meth 187:138–145. https://doi.org/10.1016/j.jneumeth.2009.12.009.

Dien J, Spencer KM, Donchin E. (2004). Parsing the "Late Positive Complex": Mental chronometry and the ERP components that inhabit the neighborhood of the P300. Psychophysiol 41(5):665-678.

Donchin E. (1981): Surprise! … Surprise? Psychophysiol 18:493-513.

Donchin E, Coles MGH. (1988): Is the P300 component a manifestation of context updating? Behav Brain Sci 11:357-374.

Duncan-Johnson CC, Donchin E. (1977): On quantifying surprise: The variation of event-related potentials with subjective probability. Psychophysiol 14:456-467.

Eichenlaub JB, Ruby P, Morlet D. (2012): What is the specificity of the response to the own first-name when presented as a novel in a passive oddball paradigm? An ERP study. Brain Res 1447:65-78. doi: 10.1016/j.brainres.2012.01.072.

Erlbeck H, Kübler A, Kotchoubey B, Veser S. (2014): Task instructions modulate the attentional mode affecting the auditory MMN and the semantic N400. Front Hum Neurosci 8:654.

Falkenstein M, Hohnsbein J, Hoormann J. (1994): Effects of choice complexity on different subcomponents of the late positive complex of the event-related potential. EEG Clin Neurophysiol 92:148-160.

Fisher DJ, Smith DM, Labelle A, Knott VJ. (2014): Attenuation of mismatch negativity (MMN) and novelty P300 in schizophrenia patients with auditory hallucinations experiencing acute exacerbation of illness. Biol Psychol 100:43-49.

Holeckova I, Fischer C, Giard MH, Delpuech C, Morlet D. (2006): Brain responses to a subject's own name uttered by a familiar voice. Brain Res 1082:142-152.

Horn JL. (1965): A rationale and test for the number of factors in factor analysis. Psychometrika 30:179–185. https://doi.org/10.1007/BF02289447.

Johnson R. (1986): A triarchic model of P300 amplitude. Psychophysiol 23:367-384.

Justen C, Herbert C. (2016): Snap your fingers! An ERP/sLORETA study investigating implicit processing of self- vs. other-related movement sounds using the passive oddball paradigm. Front Hum Neurosci 10:465.

Kayser J, Tenke CE. (2006): Principal components analysis of Laplacian waveforms as a generic method for identifying ERP generator patterns: I. Evaluation with auditory oddball tasks. Clin Neurophysiol 117:348–368. https://doi.org/10.1016/j.clinph.2005.08.034.

Kayser J, Tenke CE. (2015): Issues and considerations for using the scalp surface Laplacian in EEG/ERP research: A tutorial review. 97:189-209.

Kayser J, Tenke CE, Croppmann CJ, Alschuler DM, Fekri S, Ben-David S, Corcoran CM, Bruder GE. (2014): Auditory event-related potentials and alpha oscillations in the psychosis prodrome: Neuronal generator patterns during a novelty oddball task. Internat J Psychophysiol 91:104-120.

Kayser J, Tenke CE, Nordby H, Hammerborg D, Hugdahl K, Erdmann G. (1997): Event-related potential (ERP) asymmetries to emotional stimuli in a visual half-field paradigm. Psychophysiol 34:414-426.

Kayser J, Tenke CE, Bruder GE. (1998). Dissociation of brain ERP topographies for tonal and phonetic oddball tasks. Psychophysiol 35:576-590.

Kotchoubey B. (2006): Event-related potentials, cognition, and behavior: A biological approach. Neurosci Biobehav Rev 30:42-65.

Kotchoubey B, Grözinger B, Kornhuber AW, Kornhuber HH. (1997): Electrophysiological analysis of expectancy: P3 in informed guessing. Internat J Neurosci 91:105-122.

Kotchoubey B, Kaiser J, Bostanov V, Lutzenberger W, Birbaumer N. (2009): Recognition of affective prosody in brain-damaged patients and healthy controls: A neurophysiological study using EEG and whole-head MEG. CABN 9:153-167.

Kotchoubey B, Lang S, Bostanov V, Birbaumer N. (2003): Cortical processing in Guillain-Barre syndrome after years of total immobility. J Neurol 250:1121-1123.

Kotchoubey B, Pavlov YG. (2017): Name conditioning in event-related brain potentials. Neurobiology of Learning and Memory, doi: https://doi.org/10.1016/j.nlm.2017.09.009

Kühnis J, Elmer S, Jäncke L. (2014): Auditory evoked responses in musicians during passive vowel listening are modulated by functional connectivity between bilateral auditory-related brain regions. J Cogn Neurosci 26:2750-2761. doi: 10.1162/jocn_a_00674.

Lang S, Kotchoubey B, Lutz A, Birbaumer N. (1997): Was tut man, wenn man nichts tut? Kognitive EKP-Komponenten ohne kognitive Aufgabe. Z Exper Psychol XLIV:138-162.

Luck SJ, Gaspelin N. (2017): How to get statistically significant effects in any ERP experiment (and why you shouldn’t). Psychophysiol 54:146–157.

Marhöfer DJ, Bach M, Heinrich SP. (2014): Faces are more attractive than motion: evidence from two simultaneous oddball paradigms. Doc Ophthalm 128:201-209. doi: 10.1007/s10633-014-9434-1.

Mitzdorf U. (1991): Physiological sources of evoked potentials. EEG Clin Neurophysiol Suppl. 42:47-57.

Oades RD, Dittmann-Balcar A, Zerbin D. (1995a): The topography of 4 subtraction ERP-waveforms derived from a 3-tone auditory oddball task in healthy young adults. Internat J Neurosci 81:265-281.

Oades RD, Zerbin D, Dittmann-Balcar A. (1995b): The topography of event-related potentials in passive and active conditions of a 3-tone auditory oddball test. Internat J Neurosci 81:249-264.

O'Donnell BF, Friedman S, Swearer JM, Drachman DA. (1992): Active and passive P3 latency and psychometric performance: Influence of age and individual differences. Internat J Psychophysiol 12:187-195.

Palmer JA, Kreutz-Delgado K, Makeig S. (2012): AMICA: An adaptive mixture of independent component analyzers with shared components. Swartz Center for Computational Neuroscience, University of California San Diego, Tech. Rep. https://sccn.ucsd.edu/~jason/amica_a.pdf.

Polich J. (1987): Comparison of P300 from a passive tone sequence paradigm and an active discrimination task. Psychophysiol 24:41-46.

Polich J. (1989): P300 from a passive auditory paradigm. EEG Clin Neurophysiol 74:312-320.

Polich J, McIsaak HK. (1994): Comparison of auditory P300 habituation from active and passive conditions. Internat J Psychophysiol 17:25-34.

Potts GF. (2004): An ERP index of task relevance evaluation of visual stimuli. Brain Res 56:5-13.

Rockstroh B, Elbert T, Canavan A, Lutzenberger W, Birbaumer N. (1989): Slow Cortical Potentials and Behavior. Baltimore: Urban & Schwarzenberg.

Rogenmoser L, Elmer S, Jäncke L. (2015): Absolute pitch: evidence for early cognitive facilitation during passive listening as revealed by reduced P3a amplitudes. J Cogn Neurosci 27:623-637.

Shalgi S, Deouell LY. (2007): Direct evidence for differential roles of temporal and frontal components of auditory change detection. Neuropsychologia 45:1878–1888.

Spencer KM, Dien J, Donchin E. (1999). A componential analysis of the ERP elicited by novel events using a dense electrode array. Psychophysiol 36:409-414.

Squires NK, Squires KC, Hillyard SA. (1975). Two varieties of long-latency positive waves evoked by unpredictable auditory stimuli in man. EEG Clin Neurophysiol 38: 387-401.

Sutton S, Braren M, Zubin J, John ER. (1965): Evoked-potential correlates of stimulus uncertainty. Science, 150:1187-1188.

Sutton S, Ruchkin DS. (1984). The late positive complex: advances and new problems. Ann NY Acad Sci 425:1-23.

Verleger R. (1988): Event-related potentials and cognition: A critique of the context updating hypothesis and an alternative interpretation of P3. Behav Brain Sci 11:343-356.

Verleger R, Jaskowsky P, Wauschkuhn B. (1994): Suspense and surprise: On the relationship between expectancies and P3. Psychophysiol 31:359-369.

Wronka E, Kaiser J, Coenen AM. (2008): The auditory P3 from passive and active three-stimulus oddball paradigm. Acta Neurobiol Exper 68:362-372.

Zenker F, Barajas JJ. (1999): Auditory P300 development from an active, passive and single-tone paradigms. Internati J Psychophysiology, 33:99-111.