Biological Psychology 159 (2021) 108024

Contents lists available at ScienceDirect

Biological Psychology

journal homepage: www.elsevier.com/locate/biopsycho

Pre-attentive auditory change detection for rapid auditory transient combinations: Insight from age-related processing changes

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ARTICLE INFO

Keywords:
N1
Mismatch negativity (MMN)
Aging
Temporal window of integration (TWI)
Temporal grouping
Statistical regularity
Latent inhibition

ABSTRACT

The N1 event-related potential (ERP) enhancement to auditory transients preceded briefly by another transient has been interpreted as a reflection of latent inhibition, or alternatively, as a superimposing mismatch negativity (MMN) to rare transient event combinations. In a previous study (Volosin, Gaál, & Horváth, 2017a), when rare glides preceded frequent gaps by 150 ms in continuous tones, gap-related N1 was enhanced in younger adults while P2 was attenuated both in younger and older adults, which could be parsimoniously explained by MMN overlap which was delayed with aging. The present study replicated and extended these results with a condition in which the roles of the two event types were reversed. Transients separated by 150 ms elicited delayed MMN in older adults, supporting the MMN interpretation over the latent inhibition account. Furthermore, the divergence of N1 and MMN elicitation patterns demonstrated the independence of N1 and MMN.

1. Introduction

Adaptation requires that the organism detects and responds to changes of the environment. The human sensory systems feature several mechanisms of change detection. In the auditory domain acoustic changes are processed pre-attentively, which allows us to safely ignore and focus on task-relevant parts of our environment. Rare, sudden changes in the acoustic background – potentially carrying information relevant to our survival – are still processed, and draw our attention to these changes (Schröger, 1997). Auditory change detection processes have been related mainly to two auditory event-related potentials (ERPs): the N1 and the mismatch negativity (MMN). Whereas N1 is elicited simply by acoustic transients (e.g., on- and offsets, glides, gaps in continuous tones, Näätänen & Picton, 1987; Weise, Schröger, & Horváth, 2018; Winkler, 2007), MMN is elicited by rare violations of simple (e.g., tone repetition) or complex (e.g., feature contingencies, Bendixen, Prinz, Horváth, Trujillo-Barreto, & Schröger, 2008) auditory regularities. Whereas N1 seems to reflect auditory feature detection, MMN is theorized to reflect deviations from a pre-attentively maintained model of the auditory environment (Näätänen & Winkler, 1999; Winkler, 2007). When a tone is repeated, the N1 elicited by the second tone is typically smaller (Imada, Hari, Loveless, McEvoy, & Sams, 1993; Näätänen & Picton, 1987), which has been interpreted as stimulus-specific adaptation (or refractoriness) of the neural elements responding to the given sound features (Budd, Barry, Gordon, Rennie, & Michie, 1998). Surprisingly, however, several studies demonstrated N1 amplitude *enhancements* when the repetition occurred within 400 ms (Budd & Michie, 1994; Sable, Low, Maclin, Fabiani, & Gratton, 2004). To explain this between-tone interval dependent pattern, it was suggested that the enhancement reflected the interaction of a rapid general sensitization and a strong, but delayed inhibitory process (*latent inhibition* explanation, McEvoy, Levänen, & Loveless, 1997; Sable et al., 2004). Based on post-hoc observations showing that the negative ERP enhancement not only affected the N1, but also shifted the temporally close P2 in the negative direction, two recent studies (Volosin et al., 2017a; Wang, Mouraux, Liang, & Iannetti, 2008) suggested that the enhancement might be caused by an overlapping negative ERP, possibly an MMN. To provide further evidence for this post-hoc interpretation, the goal of the present study was to replicate and extend the study of Volosin et al. (2017a) with a clear hypothesis on the presence of a similar MMN effect in a plausible variation of the previously administered paradigm.

To provide a reasonable alternative to the *latent inhibition* explanation, the *MMN-based interpretation* of N1 amplitude enhancements for

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https://doi.org/10.1016/j.biopsycho.2021.108024
Received 17 July 2020; Received in revised form 2 December 2020; Accepted 12 January 2021
Available online 15 January 2021
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repetitions occurring within 400 ms must explain why the closely following auditory event would elicit an MMN. In the study by Wang et al. (2008) interstimulus intervals (ISIs) preceding repetitions were random between 100 and 1000 ms. Based on this, Wang and colleagues suggested (among other alternatives) that repetitions with preceding ISIs shorter than 200 ms deviated from a regularity representation predicting a more typical – longer – ISI (see also Sable et al., 2004).

In the study by Volosin et al. (2017a) continuous tones alternating between two pitches by rare, brief glides were presented to participants in a passive arrangement (continuous stimulation paradigm, Horváth & Winkler, 2010). Beside rare glides, tones also contained short gaps following glides in 150 ms, 250 ms, 650 ms or in longer periods. Because of the limited number of transients and the continuous presentation of the tone, this type of stimulation allows a better assessment of between-transient relationships than paradigms presenting discrete sounds of various durations separated by silent intervals. In younger adults, gaps preceded by rare glides in 150 ms resulted in N1 enhancement (i.e., a more negative amplitude) followed by a P2 attenuation (i.e., less positive amplitude), whereas in older adults only a P2 attenuation was present. This pattern can be parsimoniously described in terms of a negative waveform overlapping the N1 and P2 latency ranges, which is delayed in older adults. Because numerous studies found that MMN is delayed in older adults (Alain, McDonald, Ostroff, & Schneider, 2004; Bertoli, Smurzynski, & Probst, 2002; Cooper, Todd, McGill, & Michie, 2006; Schroeder, Ritter, & Vaughan, 1995), this is in line with the assumption that this negativity is presumably an MMN elicited by the rare combination of closely following transients.

In addition to its parsimony, the MMN-based explanation seems plausible because numerous studies support the notion that pre-attentive auditory processes group closely (within 150−250 ms) following auditory events together (Grimm, Roeber, Trujillo-Barreto, & Schröger, 2006; Tervaniemi, Saarinen, Paavilainen, Danilova, & Näätänen, 1994; Winkler, 2007; Yabe et al., 1998). When presenting rapid trains of discrete tones, rare sound omissions elicit MMN only when the between-onset interval is lower than about 175 ms (Yabe et al., 1998, 2001). Also, when rare tones occur only together (i.e., in pairs) in a repetitive tone sequence, they only elicit one MMN (related to the first tone of the pair), when the between-onset interval is lower than about 250 ms (Czigler & Winkler, 1996; Sussman, Winkler, Ritter, Alho, & Näätänen, 1999; Sussman et al., 2002; Winkler, Czigler, Jaramillo, Paavilainen, & Näätänen, 1998), but two successive MMNs, when the between-onset interval is longer. Importantly, when tone sequences included both single and paired deviants (Czigler & Winkler, 1996; Sussman et al., 2002) or when deviant pairs violated two distinct regularities (Oceák, Winkler, & Sussman, 2008; Sussman et al., 1999; Winkler & Czigler, 1998), two successive MMN responses were registered, suggesting that during temporal grouping, the information on the second event is not lost (Oceák et al., 2008). Beside ERPs, the temporal window of integration (TWI) can be reflected in several perceptual phenomena as well: for example, participants tended to overestimate tone durations of very short sounds ($<$ 130 ms; Efron, 1970) and sounds shorter than 200 ms were perceived louder compared to sounds beyond 200 ms (loudness summation: Cowan, 1987; Oceák, Winkler, Sussman, & Alho, 2006; Zwislocki, 1969). The studies referred to above indicated that the duration of the TWI was about 150−250 ms in younger adults.

In summary, it seems plausible to interpret the results of Volosin et al. (2017a) as a reflection of the pre-attentive processing of rare or unexpected combinations of auditory transients. According to this interpretation, a frequent auditory transient (a gap in a continuous tone) was pre-attentively grouped with a rare transient (a glide in the continuous tone) when the glide preceded the gap by 150 ms, and due to the infrequency of this combined stimulus (in comparison to that of separately presented gaps), an MMN was elicited.

Although attractive, this post-hoc hypothesis relies on a single direct experimental result, and thus needs further support. The goal of the present study was to provide further evidence for this hypothesis in two ways. First, we aimed to replicate the original findings with a targeted design: In one condition, beside rare glides and frequently occurring, but temporally distant gaps, the present study featured only glide-gap pairs with 150 ms separation (i.e., no glide-gap separations of 250 and 650 ms occurred). This conceptual replication would strengthen the evidence that the original ERP results were not caused by chance. Second, we put this hypothesis to the test in a plausible variation of original paradigm: we hypothesized that if the measured effect was indeed an MMN reflecting the pre-attentive detection of a rare transient combination, then the effect should also appear if the roles (probabilities) of the two event types were reversed. That is, because exchanging the two transient types in the continuous tone would not affect their regular co-occurrence pattern, a similar pattern of MMN elicitation is to be expected. Thus, in a second condition we presented continuous tones with frequent glides and rare gaps, and expected that an MMN would be elicited by glides following rare gaps in 150 ms, and that this MMN would also be delayed in older adults independently of N1. The introduction of this reversed condition also allowed to assess the presentation frequency-related modulation of the auditory ERPs elicited by the gaps and glides.

2. Methods

2.1. Participants

Fifty-two healthy adults (26 in the older and 26 in the younger group) participated in the experiment, but due to excessive number of EEG artifacts, data from two participants were not used in the analyses. Thus, 25 younger (mean age = 22 years, SD = 2.02, 13 females, two left-handed) and 25 older adults (mean age = 68 years, SD = 3.24, 16 females, one left-handed) remained in the final sample. The younger adult group was recruited by a student part-time job agency, whereas older adults were selected from the department's participant data base. Both groups were remunerated for their participation. All participants gave written informed consent after the experiment was explained to them. The project was approved by the United Ethical Review Committee for Research in Psychology (Hungary) and all experiments were conducted in accordance with the Declaration of Helsinki.

All participants reported normal or corrected-to-normal vision, normal hearing and the absence of psychiatric and neurological diseases. The experimental session started with the measurement of pure tone thresholds for both ears in the 250–2000 Hz range (SA-6 audiometer, Mediroll, Debrecen, Hungary) as this range covered the frequency spectrum of the stimuli. Threshold differences between the ears did not exceed 15 dB for any of the participants; however, they were systematically higher in the older than in the younger adult group (see Table 1). To compensate between-subject differences in hearing thresholds, the intensity of the tones presented during the experiment (see below) were individually adjusted to 50 dB above the 75 % hearing threshold as measured in the Single Interval Adjustment Matrix procedure (Kaernbach, 1990; Shepherd, Hautus, Stocks, & Quek, 2011).

2.2. Stimuli and procedure

After measuring the hearing thresholds and mounting the electrodes,

Table 1
Group-average hearing thresholds (dB SPL) and corresponding standard deviations between 250 and 2000 Hz.

Group	250 Hz	500 Hz	1000 Hz	2000 Hz
Younger	13.8 (SD = 4.21)	8.8 (SD = 4.79)	4.9 (SD = 3.34)	10.1 (SD = 4.59)
Older	20.2 (SD = 5.54)	15.6 (SD = 6.54)	13.6 (SD = 4.9)	19.7 (SD = 5.74)
	$t(44.816) = 4.596, p < .001$	$t(43.991) = 4.19, p < .001$	$t(42.381) = 7.333, p < .001$	$t(45.783) = 6.528, p < .001$

participants were seated in a comfortable chair in a dimly lit, acoustically isolated and electrically shielded room. Before starting the experiment, a short, 1–2 minutes long block with various eye-movements (as described by Schlögl et al., 2007) was recorded. During the experiment, a self-selected, subtitled movie was presented. Instead of the original sound of the movie, 321 s long continuous tones were played through headphones (HD-600, Sennheiser, Wedemark, Germany). Each long tone corresponded to an experimental block, and participants were instructed to relax and watch the movie while ignoring the auditory stimulation. Tones were generated by Csound (version 6.09.1, www.csounds.com) with a sampling rate of 44.1 kHz and consisted of the fundamental, second and third harmonics (the first one was missing) presented with equal amplitudes. Tones were alternating between two base frequencies (220 Hz and 277 Hz) by quick, 10 ms long glissandos (glides) and they also contained short, 10 ms long silent periods with 10−10 ms linear rise and fall times (gaps).

The presentation probability of glides and gaps was manipulated blockwise: in "Frequent Gaps" blocks gaps were presented frequently, and glides rarely, whereas in "Frequent Glides" blocks glides were presented frequently, and gaps rarely (Fig. 1). The continuous tones were divided into 1300 ms segments. Rare events could occur at the timepoints separating the 1300 ms segments, with a probability of 1/7. Randomization was biased in order to allow only separations of at least with 3900 ms between rare events (on average, there were 36 rare events per block). Frequent events were randomly inserted 150 ms after the potential timepoints of rare events with a probability of 50 %. That is, rare and frequent events either occurred in event-pairs separated by 150 ms (glide-gap or gap-glide pairs) or without any event preceding them within at least 1150 ms (single gap or single glide).

Altogether 14 blocks (tones) were presented during the experiment. The first half of the experiment (1-7th blocks) consisted of Frequent Glides blocks for 50 % of the participants while the other 50 % started with Frequent Gaps blocks.

2.3. EEG recording and pre-processing

The continuous EEG was recorded with a sampling rate of 500 Hz using an ActiCHamp (Brain Products GmbH, Gilching, Germany) amplifier in the 0−140 Hz range. 61 active electrodes were mounted on an elastic cap (ActiCAP slim) arranged according to the extended 10–20 % system (Nuwer et al., 1998). Two additional active electrodes were placed at the mastoids. The reference and ground electrodes were placed at the tip of the nose and at the forehead at electrode position Fpz on the cap, respectively. The vertical electro-oculogram was calculated offline by subtracting the signal of an additional electrode placed under the left eye from the Fp1 electrode. The horizontal electro-oculogram was measured online as the difference signal between two external passive electrodes attached near the outer canthi of the eyes.

The continuous EEG was filtered offline. After using a 1 Hz highpass finite impulse response (FIR) filter (Kaiser-windowed sinc filter, beta of 4.53, 2929 coefficients; 0.5 Hz transition bandwidth, stopband attenuation at least 50 dB), the eye movement correction procedure introduced by Schlögl et al. (2007) was applied. After that, the corrected EEG data was filtered again with a 30 Hz lowpass FIR filter (Kaiser-windowed sinc filter, beta of 4.53, 2929 coefficients, 0.5 Hz transition bandwidth, stopband attenuation at least 50 dB).

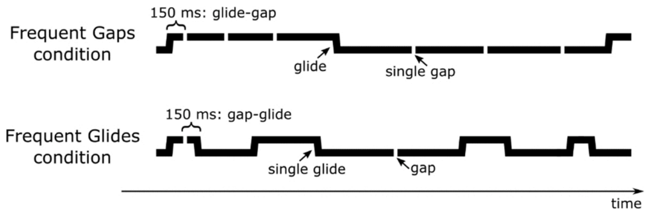

Fig. 1. The schematic design of the study. The order of Frequent Gaps and Frequent Glides conditions were counterbalanced between participants. In the Frequent Gaps condition, glide-gap pairs consisted of a gap preceded by a rare glide in 150 ms while in the Frequent Glides condition, gap-glides pairs consisted of a glide preceded by a rare gap in 150 ms.

800 ms long epochs were extracted, including a 150 ms long interval preceding the potential onset time of a frequent event separately for each event type. In the Frequent Gap condition, "glide-gap", "single gap", and "single glide" events were extracted, whereas in the Frequent Glides condition "gap-glide", "single glide", and "single gap" events were selected. Frequent events preceded by a rare one in 150 ms (glide-gap and gap-glide event-pairs) are labeled *paired,* whereas those without such immediately preceding rare events are labeled *single* events in the following. To eliminate ERPs overlapping those related to paired events, a further correction procedure was applied: For each frequent event type, ERP epochs featuring the same physical stimulation except for the frequent event were extracted. These *correction* ERPs were subtracted from the corresponding paired ERPs to yield an estimate of the ERP related to the frequent event, and these difference ERPs are referred to as *corrected* waveforms (see Figs. 2, 4 and 6) That is, for example, the Frequent Gaps condition single glide ERP was subtracted from the glide-gap ERP to yield a corrected glide-gap ERP. Also, for the Frequent Gaps condition single gap ERP, an ERP segment without a rare glide anchored to the timepoint at which frequent gaps could have had potentially occurred, but had not, was subtracted to yield a corrected single gap waveform. For the comparison of the physically identical single events (single glide or single gap) between conditions, no correction was applied due to the lack of temporal overlap with any other stimulus type. Epochs with a signal range exceeding 100 μV in any channels were rejected from processing. Individual ERPs were separately averaged in the two groups, conditions (Frequent Gaps/Frequent Glides) and event types (single/paired).

2.4. Statistical analysis – ERP amplitudes

Following the processing and statistical procedures of Volosin et al. (2017a), and because N1 originates from different sources (Näätänen, 1982), all peak latency and amplitude measurements were performed separately at two clusters of electrodes in both conditions: the fronto-central cluster ("FCz cluster" as the average of FCz, Cz, Fz, FC1, and FC2 signals, the corresponding electrodes are highlighted in Figs. 3–6) and the mastoid cluster ("M cluster" as average of the left and right mastoid signals, the corresponding electrodes are highlighted in Figs. 3–6). As gap- and glide-related ERPs required slightly different analyses, they are presented separately. For both stimulus types, first the analyses on single vs. paired events are described, second, comparisons of rare vs frequent single events are presented.

2.4.1. Frequent Gaps condition

N1 and P2 ERPs were identified for single (single gap) and paired (glide-gap) events separately in the group-average corrected waveforms. Individual ERP amplitudes were calculated as signal averages in 20 ms long windows centered at the identified latencies at the FCz, and at the M cluster (for N1) for single gaps. N1 and P2 amplitudes at the FCz cluster were submitted to Group (younger/older) × Type (single/paired) ANOVAs. To investigate the positive N1 aspect (typically observable at the mastoid sites with nose-referenced recordings (Vaughan & Ritter, 1970), an ANOVA with the same structure was conducted for amplitudes at the M cluster. Because N1 and P2 may be different in the two groups per se, group-normalized N1 and P2 amplitudes were also submitted to Welch's t-tests. Because the variability of ERP amplitudes between groups is large, the amplitudes were normalized by dividing individual amplitudes with the corresponding group-averaged single waveform (single gap or single glide) amplitudes for each group in each condition. This comparison assessed whether the magnitude of ERP modulation – in terms of proportions – differed between groups.

For each participant, MMN amplitudes were calculated for corrected

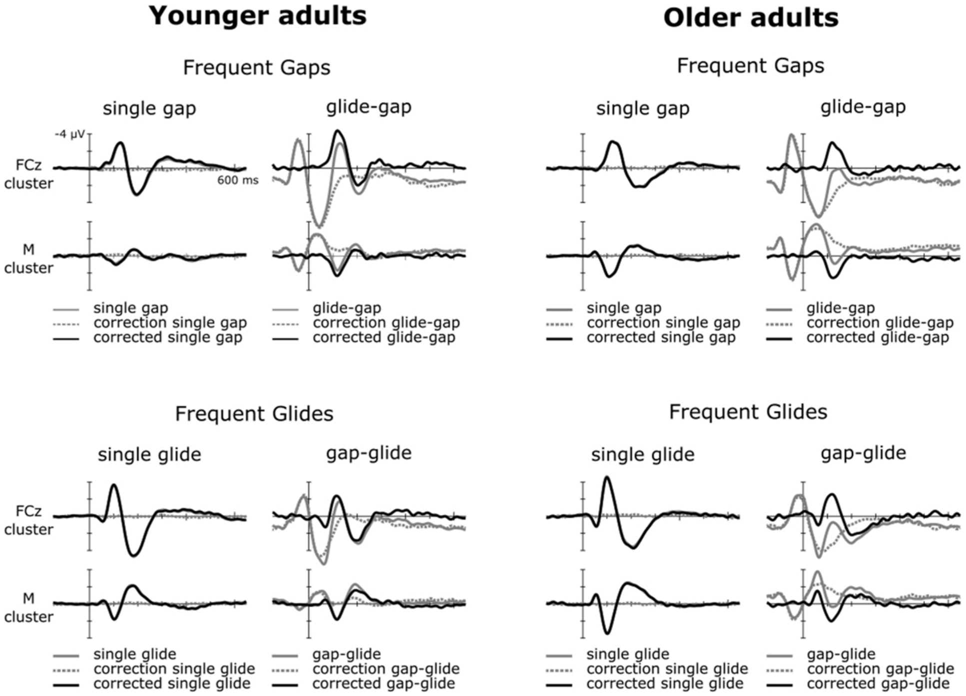

Fig. 2. Group-mean corrected ERP waveforms (black solid lines) resulting from the subtraction of correction waveforms (grey dashed lines) from paired (glide-gap and gap-glide) and single waveforms (grey solid lines) in Frequent Gaps and Frequent Glides conditions for gaps (top row) and glides (bottom row) at the FCz and M electrode clusters.

"(glide-gap)-minus-(single gap)" difference waveforms utilizing a 20 ms long window around the group-average peak at the FCz and M cluster, separately in the two groups and conditions. Amplitudes were compared between the groups by Welch's t-tests separately at the FCz and M clusters. Although we expected a single peak in each group and condition, ERPs in the Frequent Gaps condition showed two fronto-central peaks in the younger adults whereas only one was elicited in the older group. Therefore, both peaks in the younger adult group were compared separately with the single peak in older adults.

To investigate the effect of Presentation Frequency (frequent/rare) on the ERPs elicited by the same events, ERPs elicited by single gaps in Frequent Gaps (frequent) and Frequent Glides (rare) conditions were compared. As single gaps were not closely preceded by any other event, no correction for overlapping waveforms was necessary. For each participant N1 amplitudes for single gaps were calculated as the average signal in 20 ms long windows centered at the peak latencies identified at the FCz, and at the M cluster for the group-average single gap-related ERPs in the Frequent Gaps condition separately for younger and older adults. Amplitudes were submitted to Group (younger/older) × Presentation Frequency (frequent/rare) ANOVA separately for FCz and M cluster.

2.4.2. Frequent Glides condition

As a visible latency difference was present between single (single glide) and paired (gap-glide) events (see Fig. 6) in the Frequent Glides condition, N1 and P2 amplitudes were identified separately for gap-glide and single glides in the group-average corrected waveforms, and averaged individually in 20 ms long windows at FCz and at M cluster (for N1). Individual N1 and P2 ERPs calculated at FCz cluster were submitted to Group (younger/older) × Type (single/paired) ANOVAs. For the mastoid inversion of N1, signals calculated at averaged mastoids were submitted to Group (younger/older) × Type (single/paired) ANOVAs as well.

Second, similarly to Frequent Gaps, group-normalized N1 and P2 amplitudes were compared in order to assess whether the amplitude modulation differed between older and younger adults. As in normalization process in Frequent Gaps condition, individual amplitudes were divided by group-averaged single glide waveform amplitudes in each group and were submitted to Welch's t-tests.

Regarding MMN, both older and younger adults exhibited one well definable peak at "(gap-glide)-minus-(single glide)" waveform. Amplitudes were calculated in 20 ms long windows around group-averaged peaks at FCz and M clusters and submitted to Welch's t-tests separately at two clusters.

Similarly to the Frequent Gaps condition, the effect of Presentation Frequency (frequent/rare) was assessed by comparing ERPs to single glides presented in Frequent Glides (frequent) and Frequent Gaps (rare) conditions (without overlap correction). For each participant, N1 amplitudes for single glides were calculated as the average signal in a 20 ms long window centered at the peak latencies identified in the group-average ERP waveform at the FCz, and at the M clusters in the Frequent Glides condition separately for both age groups. Amplitudes were submitted to Group (younger/older) × Presentation Frequency (frequent/rare) ANOVA separately for FCz and M cluster.

2.5. Statistical analysis – ERP latencies

2.5.1. Frequent Gaps condition

Part of the present experiment is a conceptual replication of the study by Volosin et al. (2017a). For the replication, MMN latencies were analyzed in both conditions. Gap-related MMN latencies were compared at the FCz and M clusters by the jackknife method combined with a fractional area technique (Kiesel, Miller, Jolicœur, & Brisson, 2008). As two fronto-central MMN peaks were present in the younger adult group, these were compared separately to the peak latency measured in the older adult group. For details on voltage boundaries for the MMN latency comparisons, see Table 2.

2.5.2. Frequent Glides condition

Because N1s elicited by the gap-glides were apparently, and unexpectedly, delayed in comparison to single glides (in contrast to Frequent Gaps), N1 latencies were also analyzed. Latencies were measured at the

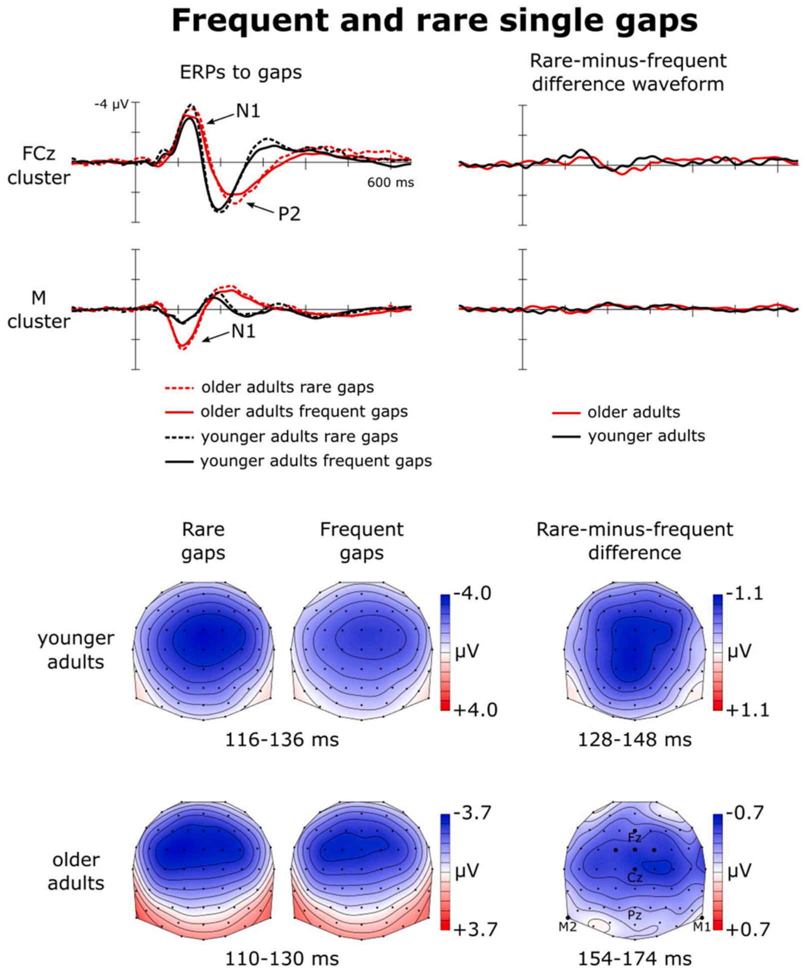

Fig. 3. Group-mean ERPs to rare and frequent gaps (top left); the corresponding rare-minus-frequent difference waveforms (top right); and the corresponding N1 and rare-minus-frequent difference topographies (bottom) in the two groups. The time intervals under the topographies reflect the intervals (±10 ms centered at the peak latency) used in the statistical analyses. The electrodes forming FCz cluster and M cluster are highlighted by thicker points.

FCz and M clusters utilizing a jackknife method combined with a fractional area technique (Kiesel et al., 2008). The glide-related N1 latencies were characterized by the timepoints halving the area under the ERP curve bounded by -0.5 µV at the FCz and 0.5 µV at the M cluster in both groups. MMN latencies were submitted to Group (younger/older) × Electrode (FCz cluster/M cluster) ANOVAs. For MMN voltage boundaries were defined individually, see Table 2. Furthermore, visual inspection showed that MMN was preceded by a positive deflection with inverted polarity at mastoids. As a further post-hoc analysis, the latency of this deflection (characterized by the timepoints halving the area between 50 and 150 ms with cutoff at -0.5 µV at FCz cluster and with cutoff at 0.5 µV at M cluster in both groups) was submitted to a Group × Electrode ANOVA.

For the jackknife-based latency analyses, unadjusted degrees of freedom, but adjusted F-, t- and p-values are reported. For the ANOVAs, generalized eta squared (η^2_G) effect sizes are reported (Bakeman, 2005; Olejnik & Algina, 2003). All statistical tests were conducted by R version 3.4.1 (R Core Team, 2017).

3. Results

The epoch numbers remaining after artifact rejection for single and paired events and for correction epochs are presented in Table 3. In the following, the analyses of ERPs obtained in the Frequent Gaps and Frequent Glides conditions are presented separately.

3.1. Frequent Gaps condition

Both corrected single gaps and glide-gaps elicited a negative and positive deflection (N1 and P2) at fronto-central leads. The negative peak showed inverted polarity at the mastoids in both groups (Fig. 2, top row). In the younger adult group, ERP responses to single gaps peaked at 128 ms at the FCz cluster and reached their maximal amplitudes at 100 ms in the mastoid signal (N1). P2 peaked at 196 ms. The (glide-gap)-minus-(single gap) difference waveforms in younger adults (Fig. 4, top right) exhibited two negative fronto-central deflections, with peaks at 112 and 164 ms. For single gaps, the N1 peaked at 126 ms at the FCz cluster and 108 ms at the M cluster (in the non-corrected group-average waveform) in the Frequent Gaps condition. The rare-minus-frequent single gap difference exhibited a negative deflection peaking at 138 ms at the FCz cluster with a smaller and a larger polarity reversal at the mastoids peaking at 114 and 146 ms, respectively (Fig. 3).

In the older adult group, N1 peaked at 122 ms in the fronto-central areas and at 108 ms at mastoids; P2 reached its maximum at 220 ms. Although the (glide-gap)-minus-(single gap) waveform exhibited several local extrema, their relatively small amplitudes did not allow a clear separation, therefore only the largest one was included in the analysis (peaking at 252 ms fronto-centrally and at 230 ms in the mastoid signal).

For single gaps, the N1 peaked at 120 ms at the FCz cluster and 110 ms at the M cluster (in the non-corrected group-average waveform) in the Frequent Gaps condition. The rare-minus-frequent single gap difference exhibited a negative deflection characterized a smaller and a

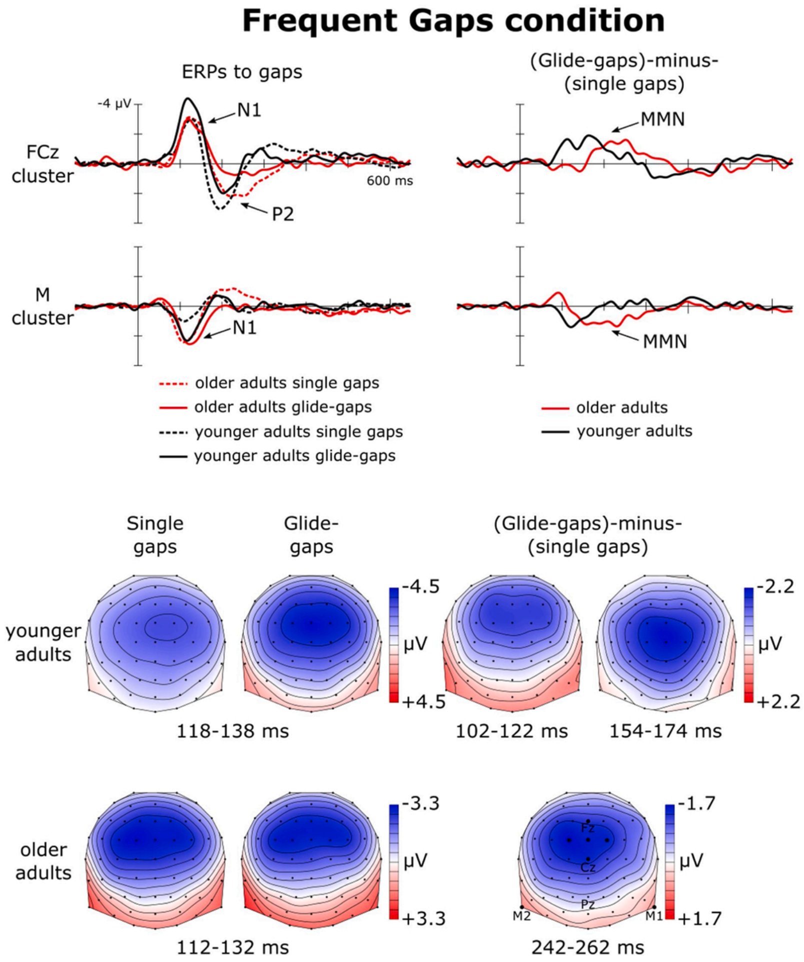

Fig. 4. Group-mean corrected ERPs (top left) and (glide-gap)-minus-(single gap) difference waveforms (top right); and the corresponding N1 and MMN topographies (bottom) in the two groups in the Frequent Gaps condition. In the younger adult group, the (glide-gap)-minus-(single gap) difference waveform exhibited two peaks, which peaked significantly earlier compared to the MMN peak of older adults. The time intervals under the topographies reflect intervals of the amplitudes (±10 ms centered at the peak latency) for the statistical analyses. The electrodes forming the FCz and M clusters are highlighted by thicker points.

larger negative peak at 136 and 164 ms the FCz cluster. The positive deflection at the mastoids also exhibited two positive local peaks: the larger one peaked at 112 ms and the smaller one peaked at 148 ms (Fig. 3).

3.1.1. *Amplitudes*

3.1.1.1. *Paired vs single gaps*

3.1.1.1.1. *N1.* The Group × Type ANOVA of the N1 amplitudes measured at the FCz cluster showed a significant Type main effect (F(1, 48) = 5.482, p = .023, η^2_G = .017), showing that glide-gaps elicited larger (more negative) amplitudes than single gaps. Group did not have a significant effect (F(1, 48) = 1.099, p = .3, η^2_G = .019). More importantly, the Group × Type interaction was significant (F(1, 48) = 9.721, p = .003, η^2_G = .029). Follow up t-tests showed that while in the younger adults group glide-gaps elicited larger (more negative) N1s than single gaps (t(24) = −3.623, p = .001), in the older group no difference was present between the two types of stimuli (t(24) = .59, p = .56). In line with that, the group-normalized N1 amplitudes also suggest that amplitude modulation was significantly stronger in the younger than in the older adult group: t(39.533) = −2.295, p = .027.

At the M cluster, results mirror the FCz cluster: no significant Group effect was present (F(1, 48) = 3.93, p = .053, η^2_G = .17), but the main effect of Type (F(1, 48) = 5.541, p = .023, η^2_G = .024) and Group × Type interaction (F(1, 48) = 15.426, p < .001, η^2_G = .067) were significant. Single gaps in the younger adult group elicited smaller responses than glide-gaps (t(24) = 4.532, p < .001) while no difference was present in the older adults group: t(24) = 1.091, p = .286. The group-normalized amplitudes suggest that amplitude modulation was significantly stronger in the younger than in the older adult group (t(27.348) = −4.298, p < .001).

3.1.1.1.2. *P2.* The Group × Type ANOVA of the gap-related P2 amplitudes showed significant Group (F(1, 48) = 6.003, p = .018, η^2_G = .081) and Type (F(1, 48) = 28.052, p < .001, η^2_G = .143) main effects, suggesting that amplitudes were larger in the younger than in the older adult group, and that amplitudes were larger for single gaps than for glide-gap events. The Group × Type interaction was not significant (F(1, 48) = .25, p = .619, η^2_G = .001).

3.1.1.1.3. *MMN.* In the younger adult group, the corrected (glide-gap)-minus-(single gap) difference waveform exhibited two clear fronto-centrally negative peaks. In the older adult group, although several local peaks were present, these were less clear-cut than in the younger adult group, thus the largest peak was used to characterize this deflection (Fig. 4, top right). The between-group comparisons of peak amplitudes showed no significant differences at the FCz cluster (first peak: t(46.64) = −.082, p = .935; second peak: t(42.428) = −.726, p = .472), or at the M cluster either: t(46.773) = .024, p = .981.

3.1.1.2. *Rare vs frequent gaps*

3.1.1.2.1. *N1.* For the FCz cluster, the Group × Presentation Frequency ANOVA showed a significant Presentation Frequency main effect, suggesting more negative amplitudes when gaps were rare (i. e., in the Frequent Glides condition) compared to when they were frequent (i. e. in the Frequent Gaps condition, see Fig. 3, left): F(1, 48) = 11.543, p = .001, η^2_G = .017. Neither the Group main effect (F(1, 48) = .006, p = .939, η^2_G < .001), nor the Group × Presentation Frequency interaction: F (1, 48) = 2.293, p = .136, η^2_G = .004 was significant.

At the M cluster, the Group × Presentation Frequency ANOVA

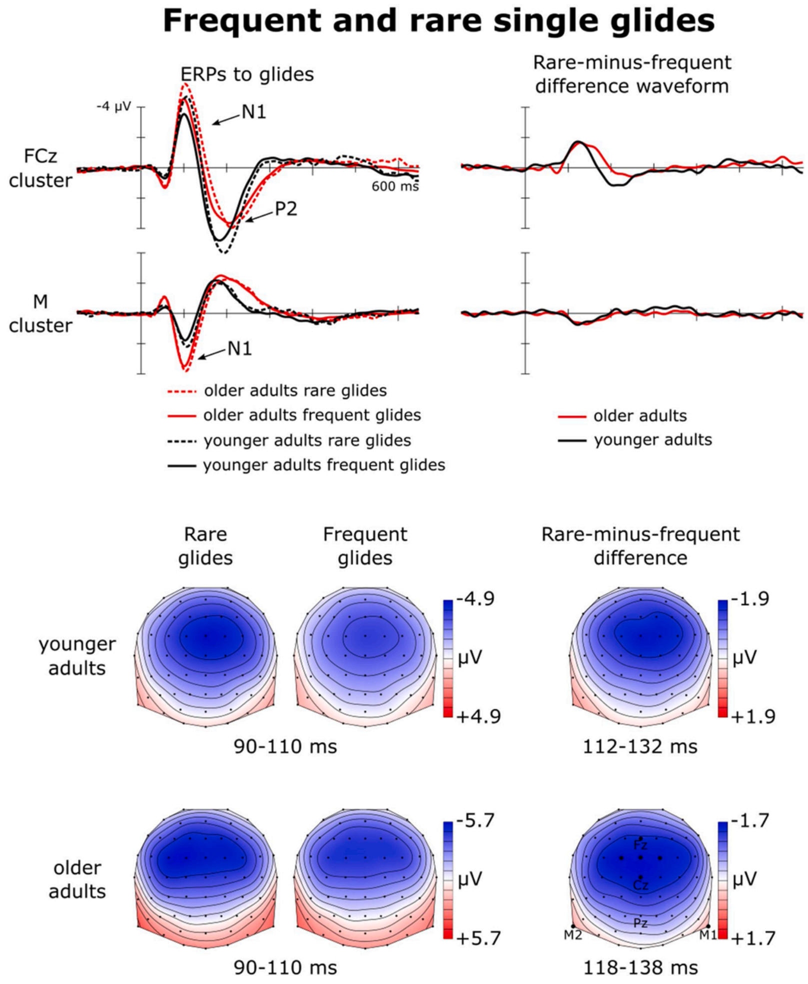

Fig. 5. Group-mean ERPs to glides (top left); the corresponding rare-minus-frequent glide difference waveforms (top right); and the corresponding N1 and rare-minus-frequent difference waveform topographies (bottom) in the two groups. The time intervals under the topographies reflect intervals (±10 ms centered at the peak latency) used in the statistical analyses. The electrodes forming FCz cluster and M cluster are highlighted by thicker points.

revealed a significant Group main effect: $F(1, 48) = 19.200$, $p < .001$, $\eta^2_G = .265$, indicating more negative amplitudes in the older adults. Neither the Presentation Frequency main effect ($F(1, 48) = .957$, $p = .333$, $\eta^2_G = .002$), nor the Group × Presentation Frequency interaction ($F(1, 48) = .812$, $p = .372$, $\eta^2_G = .002$; Fig. 3, right) was significant.

3.1.2. *Latencies*

The Group × Electrode ANOVA of the MMN latencies involving the first MMN peak in the young adult group showed a significant Group main effect ($F(1, 48) = 154.34$, $p < .001$, $\eta^2_G = .999$), and a significant Group × Electrode interaction ($F(1, 48) = 5.526$, $p = .023$, $\eta^2_G = .974$). This shows that MMN was delayed in the older adult group both at the FCz cluster ($t(31.965) = -10.383$, $p < .001$) and at the M cluster ($t(33.698) = -6.121$, $p < .001$), but the delay was more marked at fronto-central sites.

The Group × Electrode ANOVA of the MMN latencies involving the second MMN peak in the young adult group showed a significant Group ($F(1, 48) = 71.493$, $p < .001$, $\eta^2_G = .997$) and Electrode ($F(1, 48) = 12.441$, $p < .001$, $\eta^2_G = .988$) main effect, suggesting delayed MMN in older adults and also at the FCz cluster. The Group × Electrode interaction was not significant, however ($F(1, 48) = .305$, $p = .583$, $\eta^2_G = .677$).

3.2. *Frequent Glides condition*

Single glide and gap-glide events elicited ERP waveforms with a biphasic negative-positive waveform in both groups (Fig. 6, top left). In the younger adult group, N1 peaked at 100 ms fronto-centrally, and at 102 ms in the mastoid signal. P2 peaked at 182 ms at the FCz cluster. Unexpectedly, and in contrast with the Frequent Gaps condition, the (gap-glide)-minus-(single glide) difference waveform (Fig. 6, top right) exhibited a positivity preceding the MMN. The positivity peaked at 86 ms, the MMN at 158 ms at the FCz cluster. Both deflections reversed polarity at the mastoids (peaking at 92 ms and 152 ms, respectively). Regarding non-corrected waveforms, N1 to single glides reached its maximal amplitude at 100 ms at the FCz cluster and the polarity reversal peaked at 102 ms at mastoids (Fig. 5, left). The rare-minus-frequent glides difference exhibited MMN peaking both at FCz cluster and mastoid signal and at 122 ms (Fig. 5, right).

In the older adult group, N1 peaked at the FCz and the M cluster at 100 and at 102 ms, respectively; P2 reached its maximal amplitude at 210 ms. In the (gap-glide)-minus-(single glide) difference waveform a positive and a negative (MMN) deflection were present. The positivity peaked at 94 ms both at the FCz cluster and at the mastoids. The negative, fronto-central aspect of MMN peaked at 166 ms, while the positive aspect peaked at 158 ms (M cluster). The ERP waveforms and the corresponding scalp topographies are presented in Fig. 6. The non-corrected waveforms of single glides reached their maximal amplitudes at 100 ms at the FCz cluster and 102 ms at the M cluster (N1). When subtracting frequent glides from rare glides, a clear MMN was present peaking fronto-centrally at 128 ms in accompanied with a polarity reversal at mastoids peaking at 142 ms as depicted in Fig. 5.

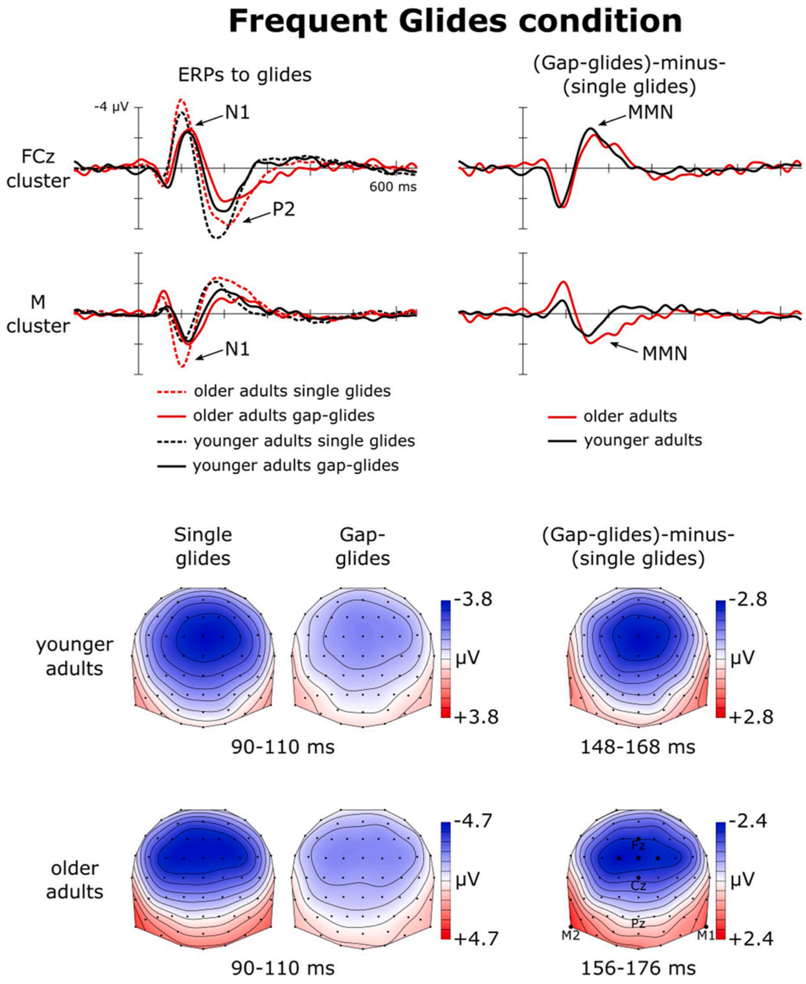

Fig. 6. Group-mean corrected ERPs (top left) and the corresponding corrected (gap-glide)-minus-(single glide) waveforms (top right) at the FCz electrode cluster and the mean mastoid signal in the Frequent Glides condition, and the corresponding ERP amplitude topographies in the N1 (bottom, left) and MMN (bottom, right) intervals. The time intervals are centered at the latency of the respective gap-glide-elicited peak and denote the time windows in which statistical analysis was performed. The electrodes forming FCz cluster and M cluster are highlighted by thicker points.

Table 2
The boundaries for defining peak latencies using the jackknife procedure combined with fractional area technique for younger and older adults in the two conditions. Note that in the Frequent Gaps condition, two MMN peaks were observable in the younger group at the FCz cluster.

Frequent Gaps condition		
	younger adults	older adults
FCz cluster	< −1.2 μV (1st peak); < −1.7 μV (2nd peak)	< −.5 μV
M cluster	> 1.2 μV	> .5 μV
Frequent Glides condition		
	younger adults	older adults
FCz cluster	< −1.2 μV	< −.5 μV
M cluster	> 1.2 μV	> .5 μV

3.2.1. Amplitudes

3.2.1.1. Paired vs single glides

3.2.1.1.1. N1. At the FCz cluster, the Group × Type of ANOVA yielded a significant Type (F(1, 48) = 29.208, $p < .001$, $\eta^2_G = .118$) main effect, showing that amplitudes were reduced for glides following gaps. Neither the Group main effect (F(1, 48) = .930, p = .34, $\eta^2_G = .015$) nor the Group × Type interaction (F(1, 48) = 1.21, p = .277, $\eta^2_G = .005$) were significant. The normalized amplitudes did not significantly differ between groups: t(38.233) = −.088, p = .929.

At the M cluster, significant Group main effect (F(1, 48) = 9.911, p = .003, $\eta^2_G = .119$), Type main effect (F(1, 48) = 9.61, p = .003, $\eta^2_G = .065$) and Group × Type (F(1, 48) = 10.777, p = .002, $\eta^2_G = .072$) interaction was found. Follow-up t-tests showed that in the older group, single glides elicited higher amplitudes compared to gap-glides (t(24) = −4.346, $p < .001$). In contrast, no amplitude difference was present between gap-glide and single glide amplitudes: t(24) = .135, p = .893. The group-normalized amplitudes also indicate a stronger amplitude reduction in the older than in the younger adult group when glides were preceded by rare gaps: t(32.658) = −2.794, p = .009.

Table 3
The mean number of epochs (with standard deviations) utilized for ERP analysis for paired and single events, including correction waveforms.

	younger adults				older adults			
	paired	paired correction	single	single correction	paired	paired correction	single	single correction
Frequent Gaps	121 (4.37)	121 (6.78)	714 (4.4)	295 (22.3)	121 (5.9)	121 (6.7)	705 (35.6)	295 (22.3)
Frequent Glides	122 (4.69)	122 (4.74)	711 (27.2)	362 (19.5)	120 (7.6)	120 (8.41)	702 (43.2)	365 (25.4)

3.2.1.1.2. P2. At the fronto-central cluster, significant Group (F(1, 48) = 4.25, p = .045, $\eta^2_G = .064$) and Type (F(1, 48) = 96.983, p < .001, $\eta^2_G = .309$) main effects were found, showing that higher amplitudes in the younger than in the older adult group, and for gap-glides compared to single glides. The Group × Type interaction was not significant, however (F(1, 48) = 1.26, p = .267, $\eta^2_G = .006$).

3.2.1.1.3. MMN. MMN amplitudes did not significantly differ between the two groups in the Frequent Glides condition (neither at the fronto-central: t(46.64) = −.082, p = .935, nor at the mastoid leads: t (45.35) = −1.022, p = .312).

3.2.1.2. Rare vs frequent glides

3.2.1.2.1. N1. At the FCz cluster, the Group × Presentation Frequency ANOVA showed significant Presentation Frequency main effect: F(1, 48) = 29.555, p < .001, $\eta^2_G = .036$, reflecting enhanced amplitudes to rare glides compared to the frequently presented ones. The main effect of Group (F(1, 48) = 1.901, p = .117, $\eta^2_G = .036$) and the Group × Presentation Frequency interaction (F(1, 48) = .037, p = .848, $\eta^2_G < .001$) was not significant.

At the M cluster significant Group (F(1, 48) = 14.697, p < .001, $\eta^2_G = .218$) and Presentation Frequency (F(1, 48) = 5.952, p = .018, $\eta^2_G = .011$) main effects were present, indicating that N1 amplitudes measured at mastoids were larger in the older compared to the younger adults and also enhanced for rare glides compared to the frequent ones. The Group × Presentation Frequency interaction was not significant, however: F(1, 48) = .093, p = .761, $\eta^2_G < .001$.

3.2.2. Latencies

3.2.2.1. N1. As N1 latencies seemed to differ between single glide and gap-glide events, N1 peak latencies were compared with a jackknife procedure combined with a fractional area technique separately in the fronto-central and the averaged mastoid signal via Group × Type ANOVAs. The significant Type main effect at both electrode sites (FCz cluster: F(1, 48) = 32.169, p < .001, $\eta^2_G = .988$; M cluster: F(1, 48) = 22.626, p < .001, $\eta^2_G = .991$) suggests that responses to glides preceded by rare gaps were similarly delayed in both groups. No other effects were significant at the FCz cluster (Group: F(1, 48) = .667, p = .797, $\eta^2_G = .382$; Group × Type: F(1, 48) = .001, p = .975, $\eta^2_G = .003$) or in the averaged mastoid signal (Group: F(1, 48) = .016, p = .9, $\eta^2_G = .1$; Group × Type: F(1, 48) = .361, p = .551, $\eta^2_G = .631$).

3.2.2.2. Positivity preceding MMN. Second, because the visual inspection of the difference waveform (Fig. 6, top right) revealed that MMN was preceded by a positive fronto-central peak, with a simultaneous negativity at mastoids, we submitted these latencies to a Group × Electrode ANOVA as well. Only a significant Group main effect was found: F(1, 48) = 4.604, p = .037, $\eta^2_G = .962$, showing delayed responses in the older adults. The Electrode main effect (F(1, 48) = .25, p = .619, $\eta^2_G = .62$) and the Group × Electrode interaction (F(1, 48) = 1.778, p = .189, $\eta^2_G = .921$) was not significant.

3.2.2.3. MMN. The Group × Position ANOVA of the MMN latencies yielded a significant Group main effect only (F(1, 48) = 19.82, p < .001, $\eta^2_G = .99$), showing that MMN peaked later in the older than in the younger adult group. Neither the Position main effect (F(1, 48) = 1.472, p = .231, $\eta^2_G = .909$) nor the Group × Position interaction were significant (F(1, 48) = .795, p = .377, $\eta^2_G = .843$).

4. Discussion

The goal of the present study was to replicate results of Volosin et al. (2017a) according to which the delayed pre-attentive processing of rare combinations of consecutive auditory transients as reflected by MMN would be modulated independently of the N1 and delayed in older adults. The results of the Frequent Gaps condition strongly supported our hypotheses: As in the previous study, gaps following rare glides in 150 ms (glide-gaps) led to a negative ERP displacement encompassing the N1 and P2 time intervals in the younger adult, but affecting only the P2 interval in the older adult group. The fronto-centrally negative displacement was accompanied by a positive displacement in the mastoid signal, showing two distinct peaks in the younger adult and one peak in the older adult group. The significant delay in the older adult group (both fronto-centrally and in the mastoid signal) in comparison to both peaks in the younger group and the MMN delay in absence of N1 modulation also supports our previous results.

Although we hypothesized that similar results will be obtained in the Frequent Glides condition, in which the glide and gap roles (presentation frequencies) were reversed, in this condition slightly different effects could be observed. Despite that a negative deflection for glides preceded by rare gaps in 150 ms was present as in the Frequent Gaps condition with a later peak in the older adult group, the N1 elicitation pattern differed from that in the Frequent Gaps condition. First, an N1 adaptation or refractoriness effect (Budd et al., 1998; Zhang, Eliassen, Anderson, Scheifele, & Brown, 2009) was observed: glides following rare gaps (gap-glides) elicited lower (less negative) N1 amplitudes than glides without an immediately preceding gap, independently of age. Second, responses to these glides were not only smaller but they were also significantly delayed in both age groups. Importantly, the fronto-central N1-effect observable as a positivity in the (gap-glide)--minus-(single glide) waveforms was delayed in the older adult group in comparison to that in the younger adult group. This age-related N1-effect delay may also contribute to the observed delay of the following negativity (MMN), because it may overlap the initial part of the negative waveform. That is, the positive N1-effect might have artificially increased the observed MMN latency in the older adult group by removing the initial part of the MMN. That is, in contrast to the Frequent Gaps condition, a strong conclusion regarding the age-related delay of the MMN cannot be drawn. The results, nonetheless, clearly show that the processing of glides preceded by rare gaps was delayed in the older adult group as reflected by the positive N1-effect and possibly by the MMN. All in all, these results are compatible with the notion that the detection of rare acoustic transient combinations is delayed in older adults.

Whereas the frequent transients in the gap-glide and glide-gap pairs resulted in similar MMN responses, N1 was modulated differently in the two conditions. For glide-gap pairs (Frequent Gaps condition), in comparison to ERPs elicited by single gaps, the gap-related ERP was enhanced in the N1 interval in the younger adult group while the ERP was seemingly not affected in the older adult group, replicating the results of Volosin et al. (2017a). In contrast, for gap-glide pairs (Frequent Glides condition), glide-related N1 amplitudes were lower than those elicited by single glides in both age groups, and they were also significantly delayed. That is, whereas rare glides do not seem to substantially affect N1 elicitation by subsequent gaps, rare gaps strongly reduced, and delayed the N1 elicited by subsequent glides. Moreover, irrespectively of the direction of N1 modulation, P2 amplitudes were attenuated in both conditions to co-occurring transients. This pattern of results contradicts the latent inhibition explanation, according to which auditory events closely following another auditory event would elicit an enhanced N1 ERPs due to a general sensitization which is not suppressed yet by a delayed inhibition of auditory processing (Sable et al., 2004). The pattern of results makes it more likely, that the N1 enhancement typically found in younger adults for an auditory event quickly following another auditory event is due to the presence of MMN reflecting the pre-attentive detection of a statistical irregularity (Volosin et al., 2017a; Wang et al., 2008).

The asymmetric N1 adaptation or -refractoriness pattern is likely caused by the gaps exciting a larger population of neural afferents, which strongly overlaps afferents involved in the processing of glides. Even though sound amplitudes were decreased and increased in 10 ms linear ramps, gaps probably still resulted in substantial excitation across the whole frequency spectrum (spectral splatter, see e.g., Moore, 2008). Despite the spectral complexity of the tone (but only three harmonics within the 220−1108 Hz range), glissandos apparently did not result in a similar, widely spread afferent activation. This explanation fits previous findings showing processing differences between glides and gaps, observable already in brainstem potentials (Arlinger et al., 1982; Arlinger & Jerlvall, 1981).

The comparison of physically identical rare and frequent single events showed the expectable results. Rare gaps and glides elicited ERPs which were fronto-centrally more negative in the N1 interval than those elicited by the physically identical frequent counterparts, irrespectively of age (Figs. 3 and 5). The delayed peak of the corresponding rare-minus-frequent differences suggest that the differences may receive contributions from an infrequency-related N1-enhancement and an MMN. This finding is in line with previous studies featuring both discrete oddball (Näätänen, 1982; Ruhnau, Herrmann, & Schröger, 2012; Schröger & Wolff, 1998) and continuous stimulation paradigms (Horváth, 2014; Volosin, Grimm, & Horváth, 2016). These presentation frequency-related results are also in contrast with the results observable for the paired rare event combinations, for which MMN and N1 modulations showed a diverging pattern.

Beside supporting the MMN-based explanation over the latent inhibition explanation, the results also contribute to the debate on the independency of MMN and N1. In short, two theories were put forward on the origins of the MMN waveform. The *predictive model based theory* (see e.g. Näätänen, Kujala, & Winkler, 2011) suggests that MMN reflected a distinct set of processes which allow the auditory system to encode auditory stimulation regularities (Cowan, Winkler, Teder, & Näätänen, 1993; Näätänen, 1990; Näätänen et al., 2011) and maintain predictions by the hierarchical comparison of bottom-up sensory input and top-down expectations (see also Friston, 2005). In case of discrepancy, MMN emerges as an error signal (Campbell, Winkler, & Kujala, 2007; Näätänen et al., 2011; Symonds et al., 2017). This interpretation is supported by studies showing MMN elicitations not only by simple physical changes like frequency, duration, intensity (Giard et al., 1995), or gaps inserted to the middle of the tones (Näätänen, Pakarinen, Rinne, & Takegata, 2004), but also changes in complex, non-repetitive, but statistically regular sound patterns (for a summary see Winkler, 2007) including repetitions in random sequences (e.g., Horváth & Winkler, 2004), violations of feature contingencies (e.g., Bendixen et al., 2008), or even language- (Hasting, Kotz, & Friederici, 2007; Pulvermüller et al., 2001) and music-related (Koelsch & Siebel, 2005) regularities.

The *neural adaptation theory* suggests that MMN is a result of latency- and amplitude modulation of the N1 response occurring because of stimulus-specific adaptation processes. This modulation is caused by a release from neural adaptation when a change occurs in repetitive or constant stimulation (Budd et al., 1998; Pérez-González & Malmierca, 2014; Zhang et al., 2009), resulting in larger neural response by the fresh afferents of the non-adapted neurons (Jääskeläinen et al., 2004; May & Tiitinen, 2007, 2010). In these terms, the MMN waveform is an N1-effect driven by adaption patterns of N1-specific neurons (Fishman, 2014; May & Tiitinen, 2010; Symonds et al., 2017).

The present results strongly support the independence of the two responses, that is, the predictive model-based interpretation of MMN. First, although the observable topographical distributions both of N1 and MMN are mostly fronto-central, their time course differ (e.g., Horváth, Winkler, & Bendixen, 2008; Symonds et al., 2017). Second, as the difference waveforms show (Figs. 4 and 6), in the Frequent Glides condition the overlapping MMN started beyond single-glide-related N1 in both groups while in the Frequent Gaps condition it started in the time course of N1 in the younger adults. Moreover, that the N1 amplitudes changed in opposite directions in the two conditions, while MMN did not (for similar effects with intensity deviants see Winkler et al., 1998), further strengthens the assumption that N1 and MMN should be regarded as two independent components (Campbell & Winkler, 2006; Symonds et al., 2017; Takasago et al., 2020).

The N1-effect for gap-glide transient pairs (Frequent Glides condition) was an unexpected, but (post-hoc) theoretically easily interpretable finding. That this N1-effect was significantly delayed in the older adult group can be explained in several ways. First, it is well possible that this age-related delay of the positive ERP effect reflects the overlap from the following negative MMN: if MMN onset is earlier in younger adults, then it may reduce the apparent latency of the positive N1 effect. In this case the corresponding interpretation is that in the older adult group the processing of rare transient combinations is delayed in comparison to the younger adult group, the same as for the pattern observed in the Frequent Gaps condition. Second, gaps might have caused stronger neural adaptation (or refractoriness) in the older than in the younger adult group. Although the present data cannot provide direct neural evidence on the mechanism underlying this effect, the analyses of group-normalized N1-effects showing stronger relative amplitude reductions in the older adult group are compatible with such explanations. Thus, the age-related N1-effect delay may be also attributed to age-related differences in gap processing as several studies found lower ERP amplitudes in older adults (Volosin, Gaál & Horváth, 2017b; Alain et al., 2004; Harris, Wilson, Eckert, & Dubno, 2012; Volosin et al., 2017a), but note that no age-related differences were demonstrated in latencies (Bertoli et al., 2002; Harris et al., 2012). At this point, it cannot be decided which interpretation is correct, but both points to delayed processing in the older adult group.

The present and the previous study (Volosin et al., 2017a) essentially exploited that MMN was delayed in older adults (Alain et al., 2004; Bertoli et al., 2002; Cooper et al., 2006; Schroeder et al., 1995) to disambiguate the interpretation of the waveforms. The nature of these MMNs, however, is different from those typically utilized to investigate age-related changes in auditory processing. The majority of such studies present repeating sequences of discrete tones, in which MMN is elicited by occasional violations of the repetition regularity. Such violations may occur either at tone onset (e.g., frequency deviants), or slightly later for duration deviants (Cooper et al., 2006; Schroeder et al., 1995) or silent gaps inserted into the tones (Alain et al., 2004; Bertoli et al., 2002). As transient-detection per se is a fundamental index of temporal processing, MMN delays in such paradigms may be interpreted as age-related changes in this processing aspect. In contrast, the MMNs elicited in the present and previous study reflect that the auditory system represented the duration of the constant tone interval typically following a transient, which was “cut short” for infrequent transient combinations. That is, MMN was elicited in relation to a frequent transient which closely followed a rare transient, suggesting that the two transients were processed together as a rare, compound event type. This interpretation fits numerous studies on the temporal window of integration (a characteristic 150−250 ms interval in which the auditory system integrates acoustic information). The duration of TWI does not seem to substantially change with older age for discrete tones (Horváth, Czigler, Winkler, & Teder-Sälejärvi, 2007) while marginal prolongation was found with aging for vowel stimuli (Saija, Başkent, Andringa, & Akyürek, 2019).

The present paradigm may also serve as a simple model for investigating age-related changes in speech processing. Although no speech stimuli were utilized, the temporal structure of the transients may capture some relevant speech aspects. Transients similar to the glides and gaps used in the present study are essential in segmenting speech input (Weise, Bendixen, Müller, & Schröger, 2012). Gaps play significant role in perceiving temporal acuity of speech voice onset times (Phillips, Taylor, Hall, Carr, & Mossop, 1997; Zaehle, Jancke, & Meyer, 2007) and discriminating speech from background noise (Kopp-Scheinpflug, Sinclair, & Linden, 2018). Glides are crucial in discriminating stop consonants (Bishop, Adams, Nation, & Rosen, 2005) or vowels (Padgett, 2008). The 150 ms temporal separation of the glide-gap and gap-glide pairs is reasonably close to the time course of syllables (Pellegrino, Coupé, & Marsico, 2011; Summers, Bailey, & Roberts, 2012). The age-related delay in the ERP response to the compound transient event –

and thus a delay in the processing of such segmentation cues - is probably present for speech transients as well, which may make speech understanding more difficult for older adults. Such a processing delay is unlikely to be manifested in deteriorated detection thresholds (Harris, Eckert, Ahlstrom, & Dubno, 2010; Humes, Kewley-Port, Fogerty, & Kinney, 2010; Pichora-Fuller, 2003), and may remain hidden when the situation allows the use of compensation strategies (based on enhanced attention; Herrmann & Johnsrude, 2020; Lustig, Hasher, & Zacks, 2007; Zanto & Gazzaley, 2014).

In summary, the present study successfully replicated and extended the results of Volosin et al. (2017a), supporting the hypothesis that the N1 enhancement typically found in younger adults for an auditory event quickly following another event is not due to latent inhibition, but to the presence of an MMN reflecting the pre-attentive detection of a rare compound auditory event. For gap-glide pairs, the pattern of results also points to an age-related processing delay, but does not allow the exact determination of the locus of the effect, which may involve processes reflected by the MMN as well as the N1. The age-related delay of the MMN allowed us to separate the contributions of N1 and MMN, which was not possible in previous studies conducted with the participation of younger adults only. The diverging N1 and MMN modulation patterns support their independence, and suggests that N1 modulation to acoustic events following rare transients might be a result of an overlapping MMN rather than latent inhibitory processes. Our results also contribute to the better understanding of age-related deterioration of consecutive auditory transients which plays a fundamental role in speech understanding and listening abilities. Moreover, although the present paradigm utilizes less rich and complex stimulation than speech, it might be useful to map how younger and older adults process temporal relations in speech.

Declaration of Competing Interest

All authors report no actual or potential conflicts of interest.

Acknowledgements

The study has been implemented with the support provided from the National Research, Development and Innovation Fund of Hungary financed under the K funding scheme (Project no. 119587). We thank István Winkler for suggesting to replicate our previous study with reversing the role of transients. We are also grateful to Zsuzsanna D'Albini for collecting the data.

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