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Research article

Developmental Changes in Presentation Rate Effect on Auditory Event-Related Potential through Childhood to Adulthood

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Abstract. In adults, the rate of stimuli presentation has been shown to play a critical role for the event related potentials (ERP): its components become larger as presentation rate decreases. But there are few works evaluating developmental changes of this ERP modulation that might provide insights into basic forms of learning. The current study aims to examine the developmental changes in the effect of the presentation rate on ERP. Participants (N = 48) of four age groups (2–7, 8–11, 12–17 and 18–35 years old) were presented with auditory tone (1000 Hz) at three different stimulus onset asynchrony (SOA): 0.9, 1.8, and 3.6 s. During stimuli presentation 28-channels electroencephalogram (EEG) was recorded. Amplitude of ERP components increased with SOA prolongation. However, this effect was differently pronounced in each of the age groups, depending on the component and cortical site. N1P1 amplitude was increased from 0.9 to 1.8 s SOA in two oldest groups (12–17 years old and adults) predominantly at fronto-central sites. Similar increase demonstrated P2N1 component but starting from younger group (8–11 years old). Only the adult group was characterized by a significant increase in N1P1 and P2N1 amplitudes with SOA increase from 1.8 to 3.6 s. Thus, the effect of presentation rate on ERP is not fully mature even at adolescence and depends on the component with P2N1 amplitude showing modulations at younger age.

Key words: auditory event-related potentials, stimulation rate, stimulus onset asynchrony, sensory-specific adaptation, development, children, maturation

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Introduction

Event related potentials (ERPs) are a convenient tool for assessing the processing of auditory information in the brain. They are registered independently of the participant's attention and activity. All of this makes ERPs a useful tool for assessing auditory processing in children.

In adults, a complex of components P1, N1, P2, and N2 is traditionally distinguished in ERP, with a clear prevalence of the N1 and P2 components (Ruchat et al., 2002; Sharma et al., 1997). Their prominence depends on the parameters of sound presentation and the duration of the stimulus onset asynchrony (SOA) plays a critical role for the N1 and P2 components amplitude: it becomes larger as presentation rate decreases (Pereira et al., 2014). N1 amplitude continued its growth beyond 12 seconds SOA (Jaffe-Dax et al., 2017; Sams et al., 1993). It was proposed that auditory stimuli leave in the auditory system a trace (sensory or echoic memory) based on the neuronal adaptation process that affects the processing of the subsequent stimuli (Lu et al., 1992; Sams et al., 1993). The stimuli specific adaptation of neurons and sensory memory is important for isolating individual sound units from the stream or, conversely, integrating auditory information over time, which is important for processing complex auditory scenes (Ulanovsky et al., 2004). In Lu et al., 1992 the memory duration for stimuli loudness, measured in psychophysical experiments correlated with the decay of the neuronal activation trace in primary auditory cortex assessed by magnetoencephalographic technique, linking behavioural and neurophysiological data. Neuronal activation of previous stimuli fades with time and can be disregarded with long SOA. So increasing N1 and P2 with increasing SOA reflects release from neuronal adaptation (Jaffe-Dax et al., 2017). Alteration in the memory trace decay may influence other aspects of cognitive processing. For example, adult dyslexics have faster decay of auditory trace at neurophysiological level that corresponds with reduced influence of previous stimuli on the next pseudoword pronunciation at behavioral level (Jaffe-Dax et al., 2017; Lieder et al., 2019). Autism spectrum disorders are characterized by an atypically prolonged adaptation process in the auditory system, which correlates with ASD symptom severity (Lieder et al., 2019; Millin et al., 2018). Such enlarged lifetime for neuronal representations provides opportunity for linking unrelated processes and might interfere with optimal cognitive functioning. Thus, neurophysiological characteristics of sensory-specific adaptation can also serve as neuromarkers of a wide range of developmental disorders (Guiraud et al., 2011; Jaffe-Dax et al., 2017; Millin et al., 2018).

Sensory memory continues to develop throughout childhood as well as a development of the cortex continues until adulthood. However, the auditory memory trace decay time during typical developmental childhood was not systematically studied.

Table 1

Characterisation of studies that have examined age-related aspects of the effect of SOA duration on ERP components

Study	Groups (N)	Stimuli type	Paradigm	SOA conditions (s)	Recording setup	Results (as SOA increases)
Paetau et al., 1995	0.9 ISI: 3-15 years-old (N=5) adults (N=9) 1.2, 2.4 s ISI: 3-15 year-old (N=15)	Tones pairs (1000 Hz, 0.05 s, intra-pair gap of 0.025 s); Pseudowords (two syllables) Intensity: ~80 dB SPL	Pseudowords and tone pairs presented alternately within same block	ISI (offset to onset) 0.9, 1.2, 2.4 s	24–channel dc-SQUID right - left hemisphere effect consider	*Amplitude:* N1m↑
Čeponiene al., 1998	Main study: 7-9 years-old (N=10) Control 7-9 years-old (N=7)	Standard: tones (1000 Hz, 0.1 s) Deviant: tones (1100 Hz, 0.1 s) Intensity: 75 dB SPL	Oddball paradigm (only standard considered) and deviant-alone condition	Main study: 0.45, 0.8 and 1.5 s (constant within one block) Control study: 2.1 s Deviants-alone: ~ 4.493 s	F3, C3, P3, T3 referenced to left mastoid; F4, C4, P4, T4 referenced to right mastoid	*Amplitude:* P100: 0.45 to 0.8 s ns; 0.8 to 1.5 s ↓; 1.5 to 2.1 s ↓; N160: 0.4 not defined; 0.8 to 1.5 s ↑; 1.5 to 2.1 s and to 4.493 ↑ N250: 0.3 to 1.4 s ns; 1.4 to 2.1 s ns; *Latency:* P100 ns; N160↓; N250 ns
Čeponiene al., 2002	9-year-old (N=8) adults (N=8 and 9)	Tones (500 Hz, 0.1 s) Intensity: 65 dB SPL	Oddball paradigm (only standard considered)	short: 0.7 s, long: varying between 2.8 s and 7.7 s	21 electrodes; two mastoids re-referenced off-line	*Amplitude:* P1: 9 y.o. ↓; adults ns; N1: adults ↑ (and more posterior in the long SOA); P2: adults ↑; N2: 9 y.o. & adults ns; *Latency:* P1: 9 y.o. ↓; adults ns; N1: adults ns P2: adults ↑; N2: 9 y.o. ns
Gilley et al., 2005.	3-4 year-old (N=10) 5-6 year-old (N=8) 7-8 year-old (N=11) 9-10 year-old (N=9) 11-12 year-old (N=12) adults (N=10)	Natural speech syllable [uh] of 0.23 s Intensity: 70 dB SPL	Stimuli presented in a trains of with defined decreasing SOAs patterns	0.59, 0.79, 1.23 and 2.23 s	Cz as the active electrode; referenced to the right mastoid	*Amplitude:* P1↑ (3-4 and 5-6 groups); N1 ns; P2 ns N1P2↑ (after 7 years)) *Latency:* P1↓ (more in younger); N1 ns; P2 ns
Sussman et al., 2008	8 year-old (N=10) 9 year-old (N=7) 10 year-old (N=12) 11 year-old (N=10) 16 year-old (N=10) adults (N=12)	Tones (880 Hz, 0.05 s) Intensity: 75 dB SPL	Oddball paradigm (only standard considered)	0.2, 0.4, 0.6 and 0.8 s	32-channel; reference: tip of the nose but only Fz, Cz, and Pz analyzed	*Amplitude:* P1↓; N1↑ (only in adults); P2↑ (Cz, Pz); N2↑ (Fz) *Latency:* P1↓ (no age interaction); N1 ns; P2↑; N2 ns

Neurophysiological studies showed that development of the auditory cortex takes a long time. The maturation of thalamocortical afferents to deeper layers of the cortex lasts until 5 years of age, and the maturation of commissural and associative axons that provide connections between different parts of the auditory cortex lasts until 11–12 years of age (Moore, 2002; Moore & Guan, 2001). Throughout childhood, maturation of synaptic efficiency and myelin formation of axons occurs (Eggermont & Ponton, 2003). The maturation of the auditory cortex is also reflected in cortical auditory potentials, which undergo significant changes as

the child grows. As in adults, prominence of ERP components depends on the interstimulus interval parameters in childhood (Sussman et al., 2008). The N1 component has been shown to be weakly expressed in children under 6–9 years of age when stimuli were presented at high presentation rate, but it was more pronounced when a longer SOA was used (Bruneau et al., 1997; Čeponiene et al., 2002; Gilley et al., 2005; Wunderlich et al., 2006). However, there are few works evaluating age-related changes of different ERP components to stimuli presented with different SOAs. Most of them studied mainly the N1 component and covered just limited age range or only fast or slow stimulus presentation rate (Čeponiene et al., 1998, 2002; Gilley et al., 2005; Paetau et al., 1995; Sussman et al., 2008). The main details and results of studies considering the SOA duration effect in children are presented in Table 1. The study of the modulation of the main ERP components by different presentation rate and at different ages will provide a more complete understanding of the maturation of the auditory system, the processes of sensory adaptation and associated cognitive processes.

It should also be noted that most studies of auditory event-related potentials look at a limited number of cortical regions, predominantly central, and in some cases temporal. Nevertheless, it has been shown that the strength of components and its maturation changes depends on topography (Bishop, 2007; Gomes et al., 2001; Ponton et al., 2000). Thus, we believe it is important to investigate the effects of SOA in a wide range of cortical sites.

In the present study in order to assess the age-related dynamics of the amplitude and latency of the P1, N1, P2, N2 components depending on the rate of presentation, we investigated auditory ERPs to stimuli with SOA 0.9, 1.8 and 3.6 s in children aged 2 to 17 years and in adults.

Procedure and methods

Participants. *Children group:* 32 children aged from 2.5 to 16.9 (mean age = 8.8 ± 3.5) years without neurological, psychiatric disorders, mental and speech delays, or hearing problems according to parental reports. Partially data from this sample was reported in our previous work (Kostanian et al., 2023), as this group was used as a control for Rett syndrome group.

The participants were split into four groups: group 1 – children aged from 2.5 to 7 years old (N = 11, females = 6), group 2 – children aged from 8 to 10 years old (N = 12, females = 10), group 3 – adolescents aged from 11 to 17 years old (N = 9, females = 7)

Adult group: 15 neurotypical adults aged from 21 to 27 (females = 10, mean age 24.3 ± 1.7). All participants did not report any neurological, psychiatric or hearing disease, brain injuries and had not taken any medicine in the six months before the study.

The research procedure met the standards for research from the Helsinki Declaration of 1975 (Protocol 1 from 01.15.2020) and was approved by the ethical committees of IHNA and Nph RAS (Protocol no. 2 at April 30th, 2020) and Sirius University of Science and Technology amendment from April 15th, 2021. Team representative explained the procedure to participants before the study. Adults

participants signed informed consent. For children participants informed consent was obtained from the parents or legal representatives. Children have given verbal consent to participate. Participants were allowed to withdraw from the experiment at any time.

Stimuli. Pure tone at 1000 Hz, with a duration of 0.1 seconds and loudness of 65 db sound pressure level (SPL), was presented in three experimental blocks, each with a different stimulus onset asynchrony (SOA): 0.9, 1.8, and 3.6 s. Stimuli with each SOA type were presented in a separate block. For the 1.8 and 3.6 s SOA conditions, each tone was presented 150 times, while for the 0.9 s condition, it was presented 300 times. The large number of trials for the 0.9 s SOA condition was a precaution to get sufficient number of epochs for averaging when epochs with motion and other artifacts are excluded and to be able to run other types of analysis. For the current analysis only the first 150 artifact-free epochs from 0.9 s SOA condition were used to equate with other SOA conditions.

Procedure. Participants sat in comfortable chairs in a soundproof room. They listened to binaural sounds through headphones and watched a silent movie of their choice on a screen. They were asked to ignore the sounds and not move. Short breaks were given at the request of the participants. During these breaks, participants could change their position

EEG recordings: Electroencephalographic data were recorded using the NeuroTravel system with 28-scalp electrodes arranged according to the international 10–20 system guidelines ('Fp1', 'Fp2', 'F3', 'Fz', 'F4', 'F7', 'F8', 'Fc3', 'Fcz', 'Fc4', 'C3', 'Cz', 'C4', 'Cp3', 'Cpz', 'Cp4', 'P3', 'Pz', 'P4', 'Tp7', 'Tp8', 'T3', 'T4', 'T5', 'T6', 'O1', 'Oz', 'O2'). Linked earlobe electrodes were used as reference, and AFz as ground, and 0.01-70 Hz online filters were applied. The data were sampled at 500 Hz. The electrode impedances were below 10 kΩ.

Data processing. EEG was filtered with 1-30 Hz offline filters. Bad channel interpolation was applied if it was necessary. Automatic raw data inspection with ±400 μV thresholds was used for rejecting EEG segments with large artefacts, then for artifact rejection the independent component analysis (ICA) was performed. The data was segmented into epochs starting 0.2 s before a stimuli onset and lasting 0.6 s after the onset. Automatic rejection of the bed segments with signals more than ±100 μV was applied. Auditory event-related potentials (ERPs) were obtained by averaging good epochs for each condition separately. ERPs were baseline corrected to –0.2 s prestimulus intervals.

Due to age-specific dynamics in latency (Čeponiene et al., 1998, 2002; Gilley et al., 2005; Sussman et al., 2008), the mean amplitude values were calculated in different time windows for each group. The FCz channel was chosen for latency measure, as according to the literature the auditory cortex response is optimally measured in this area (Ruhnau et al., 2011). The latencies of the components were estimated as a maximum amplitude of a given component at the grand average ERP for each age group and condition separately. Since the latencies did not substantially vary between SOA conditions, average between SOA condition latency values were considered. The latencies of the components for each group are reported in Table 2.

Mean amplitude of ERP components were calculated by the time window surrounding group grade average peak latencies within 30 ms for P1, N1, P2 as these peaks are rather sharp and within 50 ms for N2. To eliminate the effects caused by the contamination of the components, the next stage of analysis considers peak to peak amplitudes. Peak to peak amplitudes for N1P1, P2N1 and N2P2 were calculated as the absolute difference in amplitude between two peaks.

Table 2

Latencies (in s) of the ERP components for each group

Age – Group	ERP components			
	P1	N1	P2	N2
2-7 years	(0.11, 0.11, 0.12) 0.115	(0.15, 0.15, 0.15) 0.15	(0.19, 0.19, 0.18) 0.185	(0.29, 0.29, 0.27) 0.28
8-10 years	(0.08, 0.08, 0.08) 0.08	(0.13, 0.12, 0.12) 0.125	(0.17, 0.18, 0.18) 0.175	(0.27, 0.26, 0.28) 0.27
11-17 years	(0.08, 0.07, 0.07) 0.075	(0.11, 0.11, 0.11) 0.11	(0.16, 0.18, 0.18) 0.17	(0.27, 0.27, 0.28) 0.27
Adults	(0.04, 0.04, 0.04) 0.04	(0.11, 0.11, 0.11) 0.11	(0.15, 0.17, 0.17) 0.16	(0.3, 0.31, 0.31) 0.305

Note: Upper row in parenthesis reported latency values of ERP components (P1, N1, P2, N2) in different SOA conditions (0.9, 1.8, 3.6 s), lower row reported average latency values, used for amplitude estimation.

The effects associated with the stimuli presentation rate and age group were investigated by repeated-measures analysis of variance (RM ANOVA) separately for amplitudes of the P1, N1P1, P2N1 and N2P2 components for each channel. Statistical analysis was performed using Python 3 with the pandas and Pingouin (Vallat, 2018) packages. RM ANOVA included Age-Group and SOA factors as well as their interaction. Age-Group was the between-subjects factor (four levels 2–7 years, 8–10 years, 11–17 years and Adults) and SOA was the within-subjects factor (three levels 0.9 s, 1.8 s and 3.6 s). Assuming independence of measures at each channel, bonferroni correction was applied.

Results

The grand-averaged ERPs in response to tones presented with different SOAs showed the expected pattern of identifiable P1, N1, P2 and N2 components at least at three of four age groups. In the youngest group (2–7 years) the N1 component was not clearly pronounced, therefore the P2N1 peak to peak amplitude was also reduced. Figure 1 demonstrates event related potentials (ERPs) in different SOA conditions in four age groups. Cz channel was chosen as most representative for the auditory evoked response and observed effects (you can see other channels in the supplementary materials).

Figure 2 represents the topography of the F-scores obtained in whole-channels ANOVA for the main effect of Age-Group and SOA and Age-Group by SOA interaction.

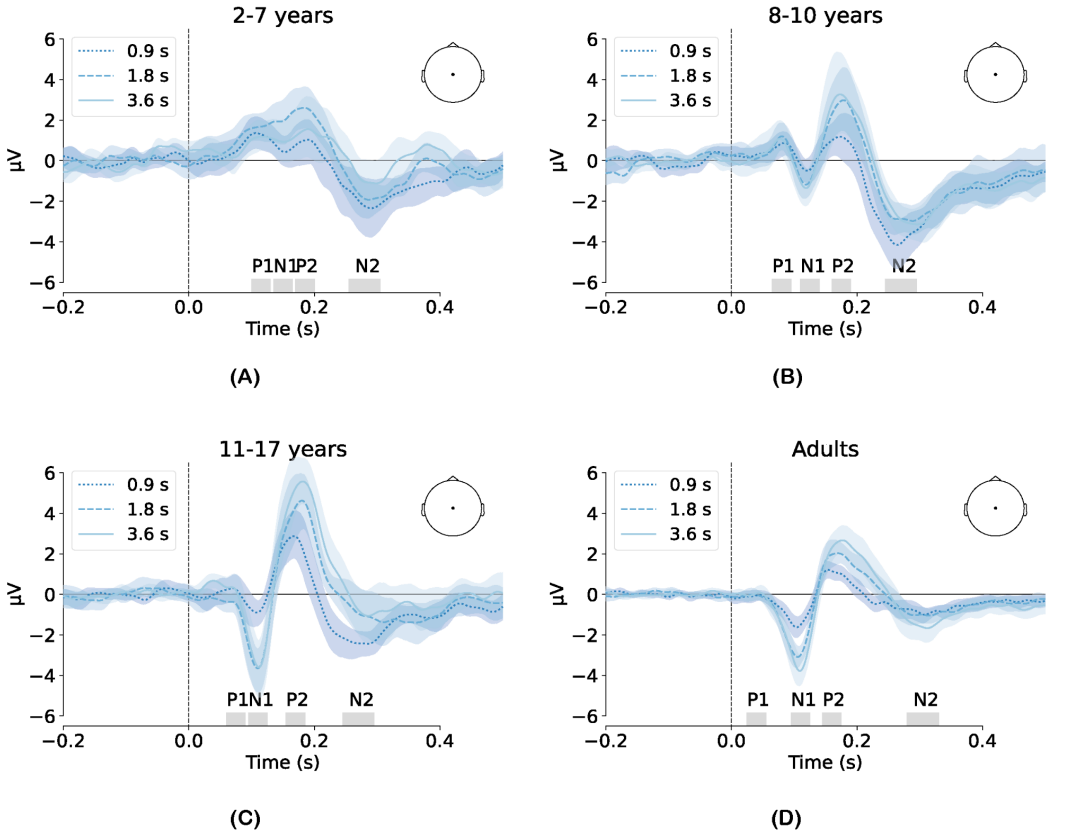

Figure 1. Auditory evoked potentials (ERPs) in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions(Cz channel) in different age groups: (A) 2 -7 years, (B) 8-10 years, (C) 11-17 years (D) Adults. Gray bars indicate time intervals, in which mean amplitude of the components was extracted. Shading corresponds to 95% confidence level

Age-Group effect. All of the measured components demonstrated the Age-Group effect. This effect was mostly pronounced for P1 and P2N1 amplitudes (15 and 12 significant channels, respectively). For the N1P1 component, the Age-Group effect was observed only for the Cz channel. N2P2 demonstrates Age-Group effect only in right temporal channels (T4, TP8, T6).

SOA effect. General SOA effect was observed for all of the components. P1 amplitude demonstrates SOA effect only in right temporal channels (T4, TP8). For N1P1 and P2N1 components SOA effect was more generalized (23 and 26 channels with significant effects, respectively). For N2P2 amplitude SOA effect was observed in 3 centro-parietal channels (Cz, CPz, Pz). The observed effect was related to decrease in the amplitude of components at a short SOA condition (0.9 s).

Age-Group by SOA interaction. P1 shows interaction effects for the T4 channel. P1 amplitude significantly differentiated 0.9 s and 3.6 s SOA conditions, but only in the youngest age group (2–7 years) (Figure 3).

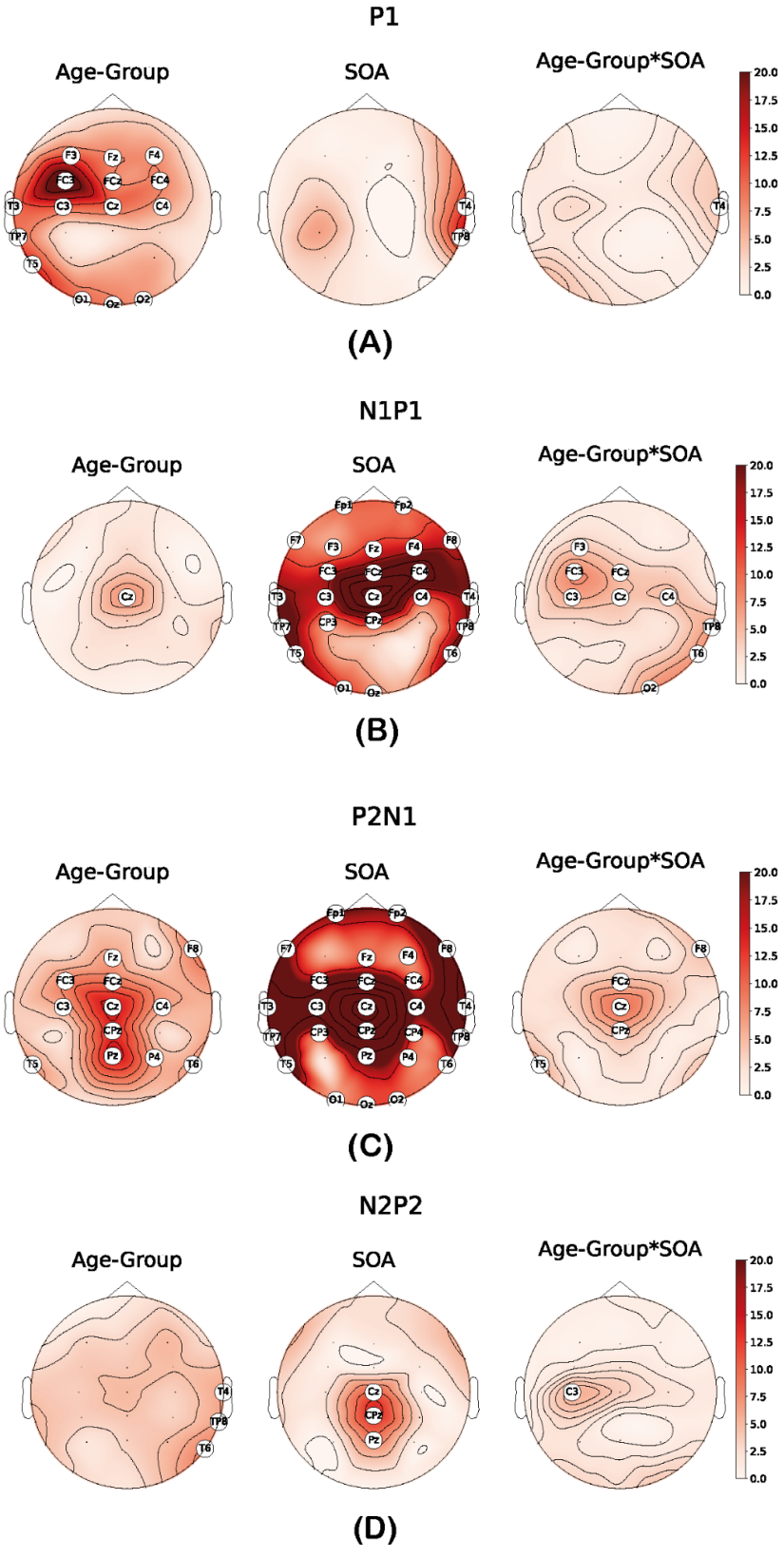

Figure 2. F-scores topography for Age-Group, SOA and Age-Group*SOA interaction effects for each component (A) P1, (B) N1P1, (C) P2N1, (D) N2P2. White dots highlight channels with significant effects ($p<0.05$, bonferroni correction)

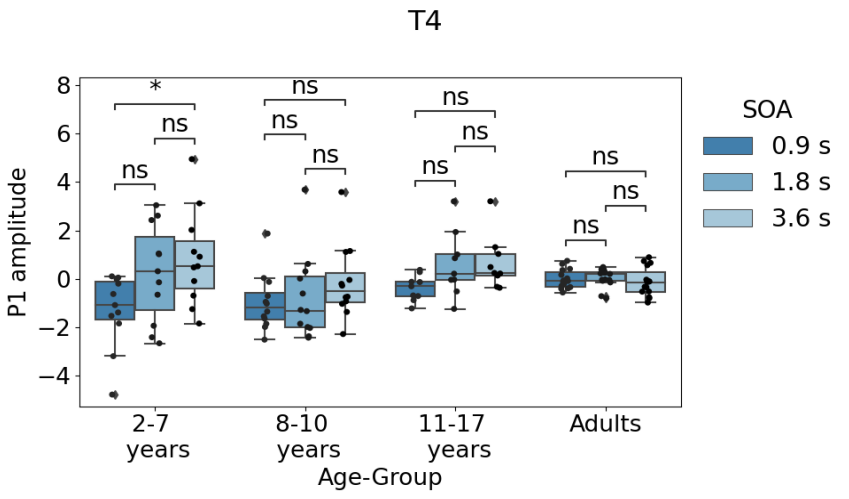

Figure 3. Boxplots representing distributions of P1 amplitudes at T4 channel in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions in different age groups. Post-hoc statistical analysis results are presented (paired T-test, asterisks (*) highlight a significant difference between conditions with $p<0.05$)

For N1P1 significant Age-Group by SOA interaction was observed for 9 channels. In the 2–7 years group the N1P1 amplitude decreases in 3.6 compared to 0.9 s SOA condition in O2 channel. There were no SOA effects for the 8–10 years group. This effect begins to be pronounced in a group of adolescents (11–17 years). In particular, in six of the channels (FC3, Cz, C3, FCz, T6, TP8) there was an increase in the amplitude of this component with enlargement of the SOA from 0.9 s to 1.8 s. No differences between 1.8 s and 3.6 s SOA conditions were observed in this group. In the adult group, SOA effects were observed in all of the significant channels. At the same time, a significant response increase from 1.8 s to 3.6 s SOA conditions was observed in Cz, C3, T6 and TP8 channels (Figure 4).

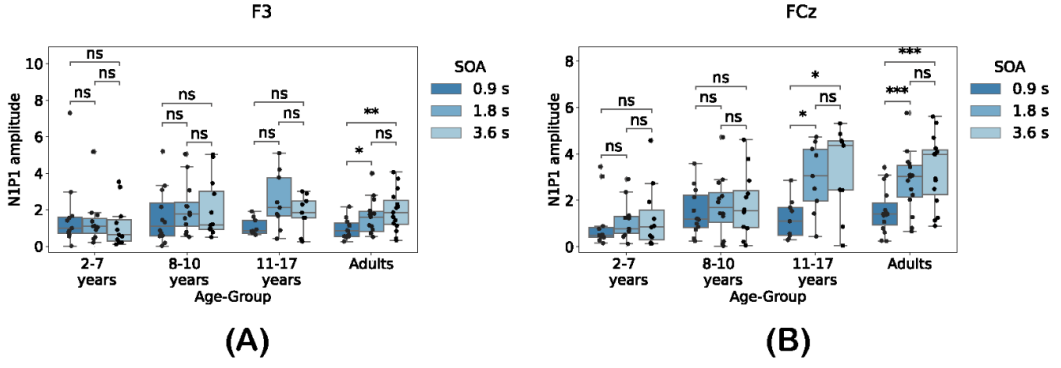

Figure 4. Boxplots representing distributions of N1P1 amplitudes at channels with significant effects in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions in different age groups. Post-hoc statistical analysis results are presented (paired *T*-test, asterisks (*) highlight a significant difference between conditions with $p < 0.05$)

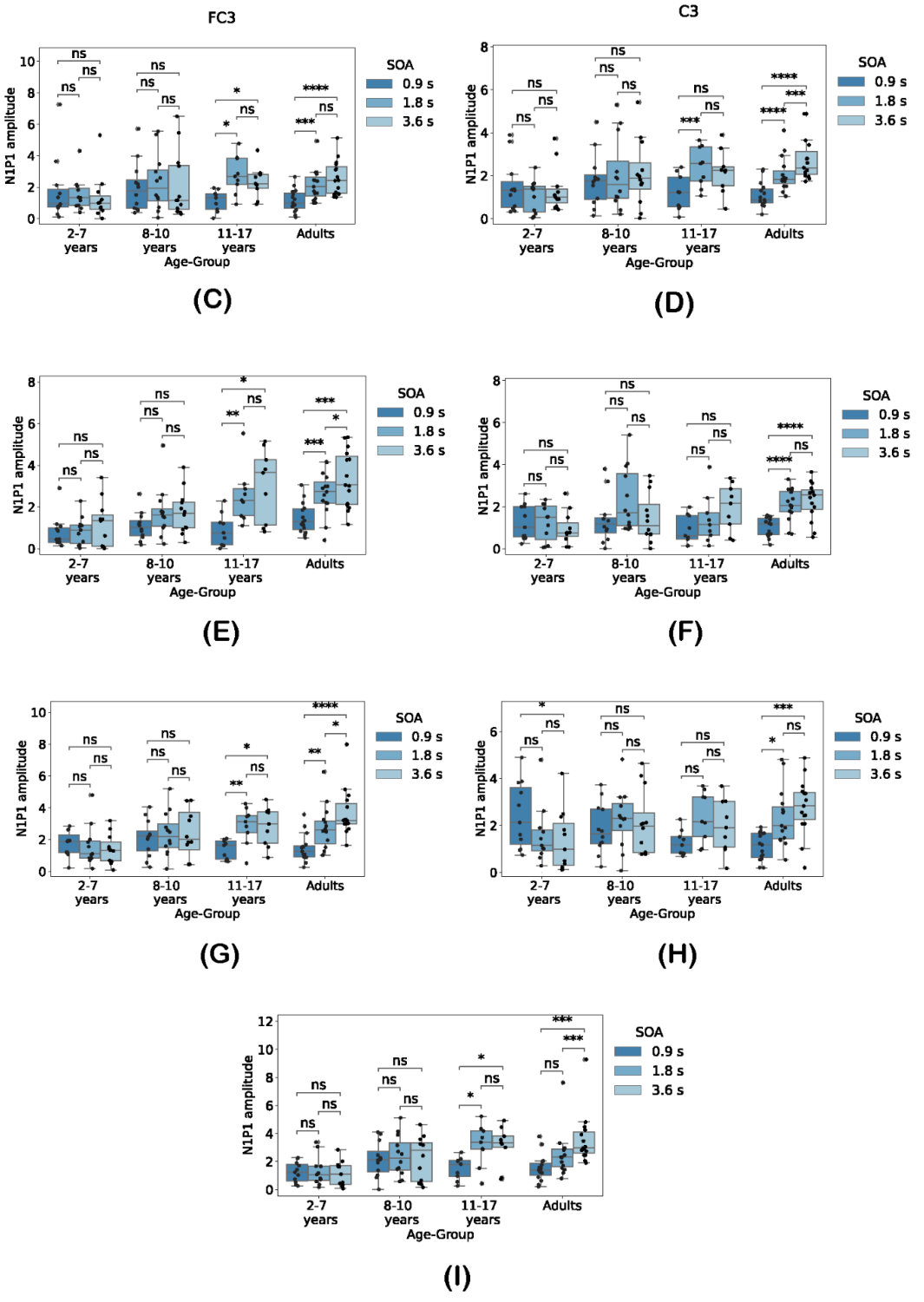

Figure 4 (***ending***)**.** Boxplots representing distributions of N1P1 amplitudes at channels with significant effects in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions in different age groups. Post-hoc statistical analysis results are presented (paired *T*-test, asterisks (*) highlight a significant difference between conditions with $p < 0.05$)

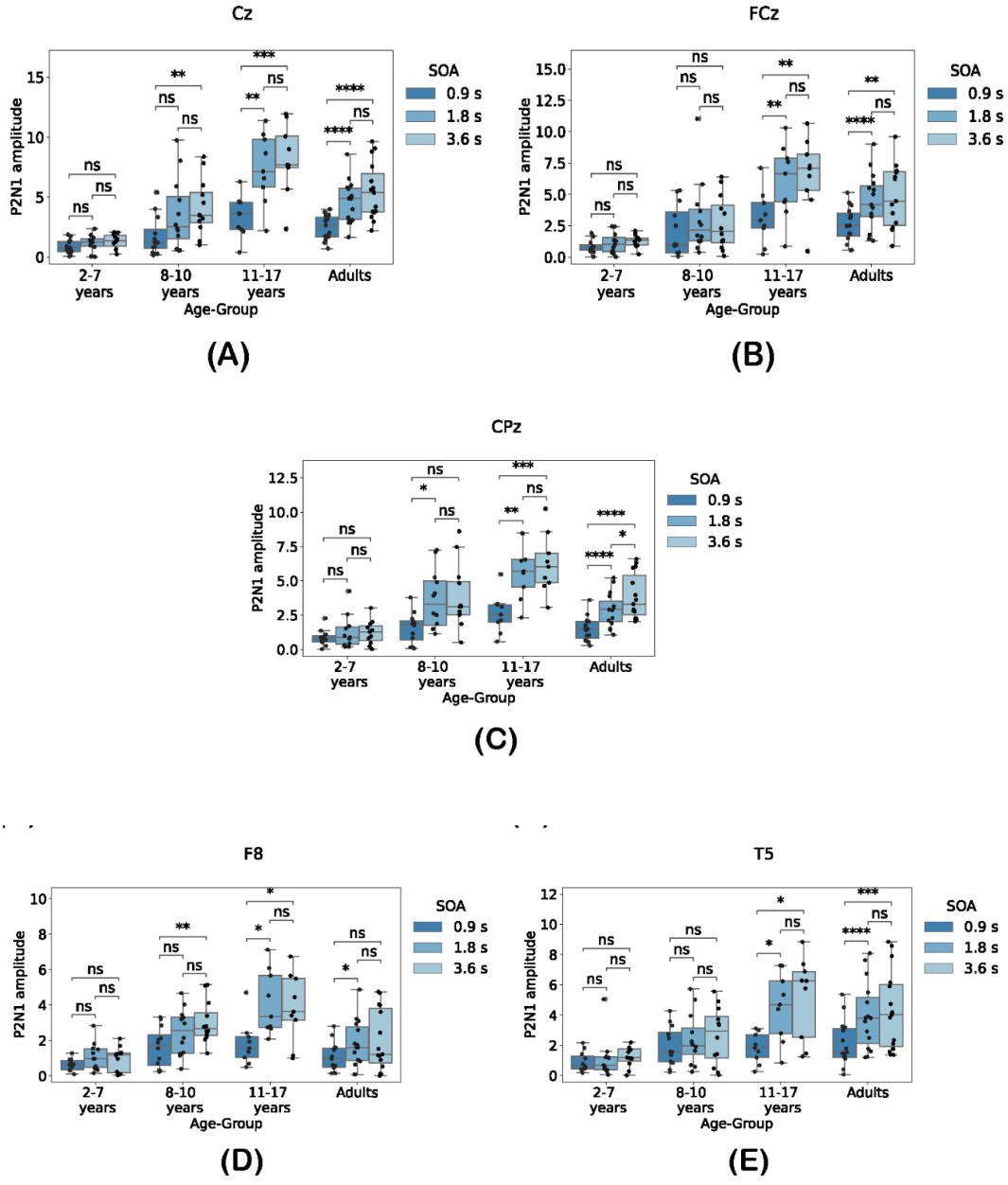

Figure 5. Boxplots representing distributions of P2N1 amplitudes at channels with significant effects in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions in different age groups. Post-hoc statistical analysis results are presented (paired *T*-test, asterisks (*) highlight a significant difference between conditions with $p < 0.05$)

For P2N1 significant Age-Group by SOA interaction was observed for 5 channels (Cz, FCz, CPz, F8, T6). The youngest group (2–7 years) demonstrates no SOA effects. In 8–10 years group SOA effect was observed in F8 and Cz channels as significant increase of the response for 3.6 s SOA condition in relation to 0.9 s condition, and in CPz channel as enhanced response for 1.8 s SOA compared to 0.9 s condition. The adolescent and adults group (11–17 years) demon-

strates significant increase in P2N1 amplitude for 1.8 and 3.6 s SOA conditions compared to 0.9 s SOA condition in all of the significant channels. However, only the adult group showed significant increase in the P2N1 amplitude in the 3.6 s condition, compared to the 1.8 s condition and that was in CPz channel (Figure 5).

N2P2 shows interaction effects for the C3 channels. This component demonstrated significant amplitude decrease from 1.8 to 3.6 s conditions in 2–7 years group (Figure 6).

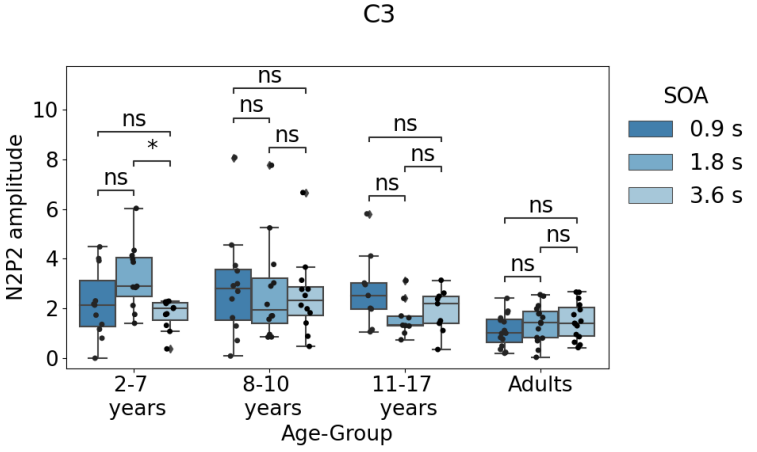

Figure 6. Boxplots representing distributions of N2P2 amplitudes at C3 channel in 0.9 s, 1.8 s and 3.6 s onset asynchrony (SOA) conditions in different age groups. Post-hoc statistical analysis results are presented (paired *T*-test, asterisks (*) highlight a significant difference between conditions with $p < 0.05$)

Discussion

In the present study, we investigated the adaptation of the main components of auditory ERPs to different stimulus presentation rates over a wide age range across groups of children from 2 to 17 years of age and adults. The presentation rate effects were observed for the P1, N1, P2 and N2 components, but these effects were age-specific. Presentation speed effects were smallest in the youngest age group, and increased with age group. The N1P1 and P2N1 were more sensitive for SOA prolongation and developmental enlargement of the amplitude of the response as well as its modulation by SOA were more pronounced.

Developmental changes in ERP configuration. While we identified the main ERP components in all experimental groups, the N1 and P2 components were noticeably reduced in the youngest group (2–7 years) consistent with previous studies (Bishop et al., 2007; Čeponiene et al., 2002). In this group ERP had a weak bifurcation similar to N1 on the P1N2 shoulder. Previous works showed that N1 was not consistently observed before the age of 7–8 years (Bishop et al., 2007; Čeponiene et al., 2002; Ruhnau et al., 2011; Sussman et al., 2008). It is important to note that even though the N1 component was clearly identifiable on the younger group's grand average response, individual ERPs of this group were characterized by high variability. The P2 component due to underdevelopment of N1 in childhood often merges with the greatly pronounced P1 component (Ponton & Eggermont, 2001). Some studies identify it as a discrete component only in children

older than 8 and in more long SOA conditions (Čeponiene et al., 2002; Gilley et al., 2005; Sussman et al., 2008).

Age-related changes were also seen as a decrease in the amplitude of the P1 response. This component almost vanished in the adult group. Decreasing the P1 amplitude with the increase of age corresponds to data revealed by Wunderlich et al., 2006 and Sussman et al., 2008. We also found a significant decrease of the N2 component in adults in the right temporal area compared to children groups. Similar age-related changes in N2 were noted in the works of Bishop et al., 2007 and Wunderlich et al., 2006.

Latency shifting is also a well-known age-related change in ERP configuration. Changes in ERP latency are associated with processes of axon myelination and maturation of synaptic mechanisms (Eggermont, 1988). Ponton et al., 2000 showed that the P1 and N1 latency decreased with age while the P2 latency did not change significantly. In contrast, the N2 component increased in latency as a function of age. In our study latency effects were not assessed statistically but at the level of group grand average response followed the previously described pattern: the latency of P1, N1 and P2 shortened from the 2–7 years group to adults, and N2 latency increased in adults.

Age effect in ERP modulation by SOA. In contrast to the present study, previous research examining age-related changes in ERP components at different SOAs have used either age-restricted samples, limited SOAs, or considered only the N1 component.

In our study the presentation rate effect on P1 amplitude was only observed in the youngest group (2–7 years) in one right temporal channel, where the amplitude became more positive as the SOA lengthened. In our previous work (Kostanian et al., 2023) we did not observe any SOA effects for P1 amplitude in the group of typically developing children, but in this work we considered only the FCz channel and not studied age effects. The observed effect in the current study might be due to the contamination of the components P1 and N1, as they are not yet fully differentiated in young children. In a study by Čeponiene et al., 1998, the frontal-central P1 and temporal P100/130 components in T3 and T4 sites decreased with increasing SOA in 7–9 year old children. It is difficult to say what our differences in results are related to, but it may be related to loudness, which was higher in Čeponiene (75 dB vs. our 65 dB).

In our study, N1P1 components demonstrated an increase with SOA prolongation only for 11–17 years and adults, it rose as SOA increased. In the adult group amplitudes of this component demonstrated significant differences between all of three experimental SOA conditions. This effect was observed in the fronto-central region with left-sided predominance and in the right inferior temporal, parietal and occipital regions. However, in adolescence, this component differed only between the 0.9 s and both longer SOA conditions and this effect was pronounced at fewer regions than in adults. Previous studies were generally consistent with these results as N1 SOA effect was reported mostly at older age (Sussman et al, 2008; Ceponiene et al 2002). For example, Čeponiene al., 2002 found SOA effect on N1 amplitude for adults but not 9 years old children, which is due to the poor promi-

nence of N1 in the shorter SOA conditions. However, some effect of SOA on N1 in younger children were found (Gilley et al 2005, Ceponiene 1998), that might be also due to the higher stimulus intensities used at these studies, as the SOA effect was shown to increase as sound intensity increases (López-Caballero et al., 2023).

For P2N1, the presentation rate effect in our study was observed for all but the youngest group: the amplitude in the 0.9 s condition was lower than in the both longer SOA conditions. In children 8–10 years of age, the effect was observed in the central electrodes; in adolescents it had a wider topography. In adults, the effect was most pronounced, and in addition, there were significant differences between the 1.8 s and the 3.6 s conditions in the centro-parietal sites. Previous works in adults showed that the amplitude of P2 significantly increased until 6 s and even 9.5 s SOA conditions (Jaffe-Dax et al., 2017; Pereira et al., 2014), thus our data is consistent with them. We have shown for the first time that the P2 SOA effect is present in children older than 7 years of age, and it appears that this P2 presentation rate effect saturates rather quickly at about 1.8 seconds.

Some studies (Paetau et al., 1995; Gilley et al., 2005) used speech sounds to estimate SOA effect. Comparable results to studies using tones as stimuli (Čeponiene et al., 1998, 2002; Sussman et al., 2008) suggest similar adaptation processing mechanisms for speech and non-speech stimuli.

Difficulties in reliable detection of the N1 и P2 components at short intervals complicated the investigation of the full developmental trajectory of these components. In Sussman (2008) and Čeponiene (2002) studies N1 component was considered only in adult groups. Čeponiene (1998) did not estimate N1 amplitude in short (0.45 s) condition. The P2 component in the Susman study was not measured in the 8 years group. To overcome this limitation, our study considered mean (average within a limited time window) rather than peak amplitude values. This approach allowed us to estimate SOA effects for a wide range of SOAs and age groups, revealing for the first time the P2 increase with SOA changes from 0.9 s to longer intervals, for the children starting from 8 years of age.

The results of our study that cover the age range from 2 years old up to young adulthood demonstrate that the effects of presentation rate on children up to the age of seven are absent for all major ERP components, with the exception of temporal P1. In children aged 8 to 10, these effects are observed only for the P2 component. In adolescents, as in adults, they are observed for the N1 and P2 components, but only between 0.9 and 1.8 s SOAs. It is important to note that the strength of SOA effects increases with age, and their topography expands. This may be related to brain maturation processes and changes in the generators of ERP components with age.

The effects of stimuli presentation rate are believed to be based on processes of stimulus-specific neuronal adaptation (Lu et al., 1992; Sams et al., 1993). Our data show that ERP amplitude reaches its maximal level or at least stops enlargement at shorter SOA in children than in adults, indicating quicker release from adaptation. At the behavioural level, this may be indirectly supported by evidence of smaller working memory capacity in children. It might also be linked to development of timing skills in children as they undertake substantial changes during the transaction from childhood into adolescence (Portnova et al., 2022).

Limitation, future directions. Some limitations should be mentioned for our study. Because of the weakness of some components in some age groups, amplitude estimations were made in averaged time windows. This approach does not allow us to assess peak amplitude and latency at the individual level. A more detailed analysis of peak latencies would allow better understanding of the source of some of the age-related and SOA effects. Additionally, we cannot exclude the possibility that in children the rate of recovery from adaptation has a nonlinear character and further increases in N1 and P2 amplitude will be observed at longer SOA than in the present study. To answer these questions it is necessary to use an additionally longer SOA than in the present work. However, adding such conditions would significantly increase the time required to study and make it difficult to handle, especially for younger children.

Conclusion

To sum up, our study showed that age-related changes in the stimulus specific adaptation, presumably related to sensory or echoic memory, continue throughout childhood until adulthood. In particular, we revealed that modulation of ERP by SOA duration becomes pronounced after 8 years of age and appears as changes in P2N1 amplitude in the central and right frontal area. Significant differences between 1.8 and 3.6 s SOA conditions occur only in the adult group and are more pronounced for the amplitude of the N1P1 component. These findings are important both for expanding fundamental knowledge about the functional organization of the auditory system and cognitive processes, and for practice. It is necessary to take into account that the detected features of sensory input depend on the speed of stimulus presentation and age of the receiver. This knowledge might be implicated in educational programs, e.g. by adjusting the optimal interstimulus interval for presenting stimuli in different perceptual and cognitive tasks. In the future, provided neurophysiological correlates might be also used to track a shift from typical developmental trajectory and creation of the new correctional and diagnostic approaches.

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Исследовательская статья

Возрастные изменения влияния скорости предъявления стимулов на конфигурацию слуховых вызванных потенциалов: от детства к взрослости

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Аннотация. Известно, что у взрослых частота предъявления стимулов имеет большое влияние на конфигурацию вызванных потенциалов (ВП): амплитуда их компонентов увеличивается по мере снижения частоты предъявления стимулов. Однако работ, оценивающих возрастные изменения этой модуляции ВП, мало. Цель данного исследования – изучить возрастные изменения влияния скорости предъявления слуховых стимулов на ВП. Участникам ($N = 48$), разделенным на четыре возрастные группы (2–7, 8–11, 12–17 и 18–35 лет), предъявлялся слуховой тон (1000 Гц) при трех различных условиях интервала от начала предъявления одного стимула до начала предъявления следующего стимула: 0,9 с, 1,8 с и 3,6 с. Во время предъявления стимулов электроэнцефалограмма (ЭЭГ) регистрировалась с помощью 28 каналов. Установлено, что амплитуда компонентов ВП увеличивалась при удлинении интервала. Однако этот эффект был по-разному выражен в каждой из возрастных групп в зависимости от компонента и участка коры. Амплитуда N1P1 увеличивалась с условия 0,9 с до условия 1,8 с в двух самых старших группах (12–17 лет и взрослые) преимущественно во фронто-центральных отделах. Аналогичное увеличение демонстрировал компонент P2N1, но эффект скорости презентации начинал наблюдаться с более младшей группы (старше 8–10 лет). Только для взрослой группы было характерно значительное увеличение амплитуд N1P1 и P2N1 при удлинении интервала с 1,8 до 3,6 с. Таким образом, эффект скорости предъявления стимулов

на ВП не является полностью сформированным даже в подростковом возрасте и зависит от компонента ВП, при этом амплитуда P2N1 демонстрирует модуляцию в более молодом возрасте.

Ключевые слова: слуховой вызванный потенциал, частота презентации, интервал между стимулами, сенсорно-специфическая адаптация, развитие, дети, созревание

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