Evaluation of frontocentral and temporal auditory evoked potentials in children with auditory processing disorder

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Abstract

Early identification of auditory processing disorder (APD) is critical to mitigate its negative impact on academic performance. However, substantial variability in cortical auditory responses among children with APD has limited the identification of a consistent neurophysiological biomarker. This variability may be partially stem from methodological limitations including use of non-ecologically valid listening conditions and stimuli with limited temporal variation. Background noise and speech stimuli with distinct temporal dynamics, such as short and long voice onset time (VOT) may provide a more sensitive framework for evaluating auditory processing impairments. More importantly, no previous study has investigated the T-complex in APD. The T-complex, which matures earlier than Frontocentral responses (P1, N1, P2, N2), may represent a promising candidate for early identification of APD. Furthermore, even in typical populations, T-complex has not been examined in response to VOT contrasts in the presence of noise. The present thesis therefore evaluated both frontocentral and T-complex responses in quiet and noisy conditions, using various stimuli in normal-hearing adults and typically developing (TD) children, followed by children with APD. The aim was to characterize typical patterns of both frontocentral and T-complex in noisy conditions and determine how these responses are altered in APD. In adults, noise induced enhancements of responses to speech, possibly reflecting the increased engagement of top-down processing during speech perception in noise. The rightward lateralization was also observed in noise, suggesting adaptive cortical mechanism to maintenance auditory processing efficiency. Although TD children showed prolonged latency and suppressed amplitude of AEPs, there was no noise-induced enhancement and rightward lateralization patterns. Such finding presumably can indicate developmental maturation of auditory cortical adaptation. Despite of developmental differences in speech-in-noise processing, both adults and TD demonstrated a rightward lateralization of VOT representation indexed by the T-complex, suggesting that hemispheric asymmetry for temporal processing is established in childhood. Children with APD exhibited significant difference in AEPs between APD and TD groups, highlighting the impairments of auditory processing in children with APD at cortical levels. Also, there was not a strong pattern of lateralization of temporal processing in APD group. Given the early maturation of T-complex and consistent lateralized processing of temporal features across development, these findings support its potential as a stable biomarker of temporal auditory processing and a promising candidate for the early identification and intervention of APD.

Acknowledgment and Dedication

I dedicate this dissertation, with the utmost respect and gratitude, to the memory of my beloved father, Dr. Ali Mohammad Ahmadi. His unwavering encouragement, profound faith in my abilities, and constant emotional support have shaped not only my academic journey but also the person I have become. Whether during his lifetime or in his absence, his presence has remained a guiding force in my life. The strength, motivation, and sense of purpose that sustained me throughout this endeavor are deeply rooted in the love and support I received from him. This achievement stands as a testament to his enduring influence and belief in me.

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Acronyms

ABR: Auditory Brainstem Response

ADHD: Attention deficits hyperactivity disorder

AEPs: auditory evoked potentials

Ag/AgCl: Silver chloride

ALRs: auditory late responses

AMRs: auditory middle responses

ANOVA: Analysis of Variance

APD: Auditory processing disorders

APDQ: Auditory processing domain questionnaire

ACPT: Auditory continuous performance test

Electrocochleography: ECochG

dB: decibels

EEG: electroencephalogram

Hz: Hertz

IHCs: Inner hair cells

Mismatch Negativity: MMN

μV: microvolts

ms: milliseconds

LI: Language impairment

OAE: Otoacoustic emissions

OHC: Outer hair cells

SNR: signal-to-noise ratio

SPL: Sound pressure level

VOT: Voise onset time

TD: Typically developing

Chapter 1. Introduction

1.1. Definition and profile of Auditory processing disorder

Auditory processing refers to the set of neural operations that enable the interpretation of acoustic signals after they enter the central auditory nervous system (BSA, 2018). When these processes do not function efficiently, individuals may struggle to understand or interpret auditory information, even though their peripheral hearing abilities remain intact. This condition is commonly described as Auditory Processing Disorder (APD) (AHSA, 2005). In this view, APD reflects impairments with auditory (sensory-neural) origins and is often conceptualized as a bottom-up disorder meaning that the primary difficulty is presumed to arise within the auditory system itself. However, a second perspective classifies APD primarily as a top-down disorder (Ferguson & Moore, 2014; Moore et al., 2010; Moore & Hunter, 2013). From the top-down perspective, APD is attributed to higher-order cognitive processing (e.g., attention, working memory and language), and cognitive processing impairments are considered key contributors to common symptoms such as difficulties in understanding speech in noise (Ferguson & Moore, 2014; Moore et al., 2010; Moore & Hunter, 2013). More recently, APD has also been discussed within an integrative framework that acknowledge the combination of bottom-up auditory processing and top-down cognitive-linguistic factors (BSA, 2018). According to this perspective, the etiology of APD is thought to involve dysfunction in neural systems within both the afferent (bottom-up) and efferent (top-down) of the auditory nervous pathways, as well as the neural mechanisms that support top-down modulation of auditory processing (BSA, 2018).

1.2. Symptoms of APD

Children with APD often present with everyday listening difficulties, particularly in environments where auditory signals are masked by background noise, competing speakers, or reverberation (Eranovic et al., 2022; Neuman et al., 2010; O'Hara & Mealings, 2018; Prodi & Visentin, 2022). Such challenges can affect communication, learning, and social participation, especially in school settings where spoken information is the primary mode of instruction (Alanazi, 2023; Bamiou et al., 2001; Canale et al., 2014; Cestnick & Jerger, 2000; Del Zoppo et al., 2015; Drosos, 2024; Hunsaker, 1983). APD may manifest in a variety of auditory behaviors, such as difficulties with temporal processing, sound discrimination, binaural integration, and extracting speech from noisy background (Alanazi, 2023; Dillon & Cameron, 2021a). According to ASHA, 2005, synthesis, comprehension and interpretation of auditory-verbal inputs are dependant on auditory processing. Impaired processing of temporal cues of

speech features such as rapid and slow changes in formants may result in poor listening and speech perception difficulties (Hestvik et al., 2023; Tallal & Piercy, 1973).

Considering the detrimental effects of APD on language acquisition, learning, and education, early identification and intervention of APD are strongly recommended (ASHA, 2005).

1.3. Evaluation and identification of Auditory Processing Disorder

The evaluation of APD traditionally includes performance-based auditory tests that assess specific auditory mechanisms, such as dichotic listening, temporal resolution, and speech perception in noise. However, the interpretation of these behavioral assessments can be complicated by non-auditory factors, such as attention (Dillon & Cameron, 2021a; Gyldenkærne et al., 2014), linguistic ability (Silman et al., 2000), and motivation (Geffner & Ross-Swain, 2014). For this reason, there has been increasing emphasis on incorporating objective physiologic (behavioral evaluations of APD have been inserted in Appendix) and electrophysiologic tests measures into APD assessment (Alanazi, 2023; Dillon & Cameron, 2021a; Ferre, 1997; Maggu & Overath, 2021).

1.3.1. Auditory physiological response

Auditory physiological responses provide information mainly from lower level of auditory system and include**:** Otoacoustic emissions (OAE) (Guinan, 2006; Shera, 2004; Veuillet et al., 1991), Electrocochleography (ECochG) (Dallos et al., 1972; Ding et al., 2002), which assess the activities of outer hair cells (OHCs) and inner hairs cells (IHCs). Also, Otoacoustic emissions suppression is another physiological response that is designed to assess the effect of inhibitory efferent system on cochlea function. It is recognized that the inhibitory effect may facilitate the comprehension of speech in demanding listening conditions (Chintanpalli et al., 2012; Morlet et al., 2019; Veuillet et al., 1991).

1.3.2. Auditory neurophysiological responses

At subcortical and cortical levels, auditory function can be assessed through recording auditory evoked potentials (AEPs) (Luck, 2012). AEPs refer to the auditory neuro-electrical activities that provide a non-invasive means of examining how sound is encoded at different stages along the auditory pathway, with high temporal resolution (Burkard et al., 2007; Kraus & Nicol, 2009; Larson et al., 2014). There are three typical AEP responses based on response latency (timing): the short (occurs during the first 10 milliseconds, ms, after the stimulation), the middle (recorded between 10 and 80ms) and the late latency responses (occurs between

80ms to 250ms) (Kraus & McGee, 1992). AEPs with short latency measure activity from subcortical areas (Anderson & Kraus, 2010; Russo et al., 2004). Middle-latency auditory evoked potentials (MLAEPs or MLR) are electrophysiological responses to sound occurring 10–80ms post-stimulus, originating from the thalamocortical pathway and auditory cortex (Goldstein & Rodman, 1967). Long latency responses signify auditory processing at the primary and secondary auditory cortex (Kraus & McGee, 1992; Näätänen & Picton, 1987; Wolpaw & Penry, 1975).

An objective test battery, referred to as *site-based APD evaluation*, has been proposed to assess auditory processing using the physiological and neurophysiological measures (Maggu & Overath, 2021) (Tabel 1). While this set offers valuable insights into the locus and nature of deficits within the auditory pathway, it seems to primarily focus on subcortical and brainstem auditory processing, so there is indeed a necessity to assess auditory processing at the cortical levels. In addition to subcortical measures, neurophysiological measures examining cortical responses could provide a more comprehensive understanding of auditory processing deficits, especially in individuals with APD.

Table 1. *Objective test battery for a site-based APD evaluation.*

Anatomical site	Test
Outer Hair Cells (OHCs)	Otoacoustic Emissions (OAE); Cochlear microphonics
Inner hair cells	Summating potential (SP) in electrocochleography (ECochG)
Ribbon synapses	Wave I Auditory Brainstem Response (ABR)
Brainstem (cochlear nucleus, superior olivary colliculi, lateral lemniscus, inferior colliculi)	ABR waves (II–V)
Efferent system (olivocochlear bundle to OHCs)	Contralateral suppression of OAE
Low spontaneous rate auditory nerve fibers	Middle Ear Muscle Reflex (MEMR)

1.4. Utility of the objective measures in identification of APD at cortical auditory levels

The following section presents a literature review of auditory cortical responses in normal-hearing populations and in individuals with APD, highlighting existing limitations, research gaps, and the focus of the current thesis.

1.4.1. Cortical AEPs responses

There are two types of cortical AEPs: obligatory and cognitive (Hall, 2007). Cognitive AEPs vary as a function of the listener's attention and performance on cognitive tasks performed while responses are recorded (Näätänen, 2000; Picton, 1992). In contrast, obligatory

AEPs are those whose presence, latency, and amplitude are determined primarily by the acoustic characteristics of the stimulus (Lütkenhöner & Steinsträter, 1998; Näätänen & Picton, 1987; Picton et al., 1999). Although these responses do not require the listener's attention to elicit, they can be impacted by attention. N1 is often larger when participant is attending to the stimuli. The larger auditory evoked potential in active paradigm versus passive paradigm suggesting that introducing attention to the target may reduce the suppression of the early cortical representation of the target leading to increase amplitude of auditory responses (Price & Bidelman, 2021; Zhang et al., 2014).

The current thesis has focused mainly on obligatory AEPs to evaluate the cortical auditory processing while minimizing the influence of top-down factors. At the cortical levels, obligatory AEPs can be categorized, based on their scalp topography, into two main distributions: frontocentral and temporal responses. The characteristics and utility of frontocentral and temporal cortical AEPs for examining auditory processing are elaborated in detail in the following section.

1.4.1.1. Definitions and features of frontocentral AEPs

The P1, N1, P2, and N2 responses are the frontocentral components of long latency AEPs, represent synchronous neural activation of structures mainly in the primary auditory cortex and the secondary associated areas of the temporal lobe (Eggermont & Ponton, 2002; Hall, 2007; Luck, 2012; Näätänen & Picton, 1987; Picton et al., 1999). P1, N1 and P2 encode the acoustical features such as frequency and timing (Čeponienė et al., 2005; Liégeois-Chauvel et al., 1994; Lightfoot, 2016; Näätänen & Picton, 1987; Schaefer, 2021). N2 it is associated with sensory auditory processing, also can be modulated by attention, perception, discrimination, and recognition of sounds (Cone-Wesson & Wunderlich, 2003; Luck, 2012; Näätänen et al., 1982).

In adult, the dominant characteristics of obligatory auditory cortical responses is N1 and P2, while in young children (under age 9), P1 and N2 are the dominant waves of cortical response (Ceponiene et al., 2002; P. M. Gilley et al., 2005; Sussman et al., 2008; Tomé et al., 2015). P1 and N2 waves reduce in amplitudes, along with a reduction in latency of P1, as children mature through infancy to adolescence (V. L. Shafer et al., 2015; Sharma et al., 1997; Wunderlich et al., 2006).

Multiple studies in normal populations have documented that the characteristics of frontocentral responses would be modified by changing the experiment parameters such

presenting non-speech versus speech stimuli with different spectral and temporal features (Kaplan-Neeman et al., 2006; Meehan et al., 2024; Näätänen & Picton, 1987; Sharma & Dorman, 1999; Sharma et al., 2000; Steinschneider et al., 1999; Wagner et al., 2016) and/or applying the background noise during the recording (Androulidakis & Jones, 2006; Billings et al., 2013; Curtis J. Billings et al., 2009; Dias et al., 2021; Duarte et al., 2022; Duquette-Laplante et al., 2024; Kaplan-Neeman et al., 2006; Papesh et al., 2015; Russo et al., 2009; Wang & Xu, 2021; Katherine A. Whiting et al., 1998a).

Among the parameters modulating the auditory brain responses, the current thesis focuses on the effect of noise on AEPs, because the difficulty understanding speech in noise is one of the most common reported symptoms in children with APD (Drosos, 2024; Majak et al., 2023; O'Hara & Mealings, 2018). In addition, examining the impact of noise on cortical neurophysiological processing of speech may help to better characterize speech perception difficulties across different levels of the auditory cortex including primary and secondary auditory cortex (Näätänen & Picton, 1987; Katherine A Whiting et al., 1998; Wolpaw & Penry, 1975).

Noise generally leads to longer latency and smaller amplitudes of auditory responses such as N1 and P2 (Androulidakis & Jones, 2006; Billings et al., 2013; Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006; Russo et al., 2009; Katherine A. Whiting et al., 1998a). However, the modulation of amplitude has not always been suppression, and some studies observed larger amplitude of N1 in noise, compared to quiet (Alain et al., 2009; Duquette-Laplante et al., 2024; Papesh et al., 2015; Parbery-Clark et al., 2011). The methodological differences have been used to account for the discrepancies in N1 findings (sBillings et al., 2009; Parbery-Clark et al., 2011). The enhanced N1 was explored in studies employing binaural stimuli presentations at relatively fast rates (Alain et al., 2009; Parbery-Clark et al., 2011), whereas smaller N1 amplitude in noise was reported in studies using monaural presentations at slower rates (Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006). However, this distinction is not fully supported, as Papesh et al. (2015) reported larger N1 in noise using both monaural and binaural conditions (Papesh et al., 2015). Therefore, more research is needed to untangle the enhancement vs reduction in noise.

Duquette-Laplante et al., 2024, recently, suggested that stimulus type is another factor influencing modulation of the N1 amplitude in noise. Specifically, N1 enhancement was only observed when speech stimuli was presented in white noise compared to non-speech stimuli. The authors proposed that the larger N1 amplitude for speech in noise reflects differences

in acoustic salience relative to the masking noise: speech was more salient than tones in white noise, thereby eliciting a larger N1 response (Duquette-Laplante et al., 2024). However, because most of the AEPs waves were not identifiable in 8-talker babble noise condition, it was not possible to compare responses between non-speech stimuli and speech stimuli under that type of noise (Duquette-Laplante et al., 2024). As Freyman et al. (2004) suggested, informational characteristics of the masker beyond six talkers are diminished, creating a constant steady state noise with minimal lexical context and thus becoming more similar to a strong energetic masker. Babble noise with fewer talkers, for example 4-talker babble, appears to be more suitable informational masking for AEP research, as several studies have reported identifiable waveforms under this condition (Benítez-Barrera et al., 2021; Billings et al., 2011; B. R. Zendel et al., 2015). Moreover, 4-talker babble noise is an informational mask, while white noise is an energetic mask. Energetic or acoustical masking refers to masking the acoustical features of speech with overlapping frequency content of noise such as broad-band noise or speech-shaped noise (Douglas S Brungart et al., 2001). Informational masking, such as 4-talker babble noise, reflects the competing speech sounds that not only physically interfere with the target speech but also impair the seperation of the sound of the speaker from the background speech (Douglas S Brungart et al., 2001). It is assumed that informational masking is more detrimental to speech understanding than energetic masking (Douglas S Brungart et al., 2001), due to failures of subject's attention leading to lower ability to attend to and segregate out the target speech in the presence of other similar speech sounds (Barbara G Shinn-Cunningham, 2008). Further studies on frontocentral cortical AEPs using 4-talker babble noise in addition to white noise, are needed to examine the effect of noise type and stimulus type (speech and non-speech stimuli) on cortical responses.

1.4.1.2. Frontocentral responses, results in clinical populations and limitations

Numerous studies have reported atypical patterns in the latencies and amplitudes of cortical AEPs, including P1, N2, P2, and N2, in children with APD (Koravand et al., 2017; Liasis et al., 2003; Morlet et al., 2019; Purdy et al., 2002). For example, a prolonged N2 latency (Koravand et al., 2017), and larger N2 and P2 amplitude (Morlet et al., 2019) have been reported in children with APD, compared to typically developing (TD) children. These differences suggest that children with APD may present a developmental delay and/or atypical cortical auditory processing, potentially reflecting altered neural transmission and processing within the auditory pathways (Morlet et al., 2019). Moreover, differences in the AEPs between the groups are particularly notable in the presence of the noise (Hussain et al., 2022; Liasis et

al., 2003; Ramadan Hassaan, 2015; Sharma et al., 2014). A study from Hussain in 2022 revealed that children with APD showed a larger N2 amplitude than TD group, while no significant between-group difference were found for P1 amplitude and N2 latency. In noise, however, the APD group demonstrated larger P1 and N2 amplitudes as well as a prolonged P1 latency relative to TD children (Hussain et al., 2022). Collectively, these findings support the view that auditory processing in children with APD is less efficient under noisy listening conditions than in typically developing peers (Anderson, Skoe, et al., 2010; Cunningham et al., 2002; Russo et al., 2005). Presumably, children with APD need more time to process acoustic stimuli and have limited access to the resources involved in sound processing in noise than TD children (Hussain, 2022; Liasis, 2003).

Although multiple studies have evaluated the effects of APD on frontocentral responses, no reliable or consistent electrophysiological biomarker of APD has yet been established. In other words, there is a high degree of variability observed in the pattern of frontocentral responses among children with APD that make it difficult to draw clear patterns or conclusions (Macaskill et al., 2022). For example, some studies have reported no significant differences in the latencies or amplitudes of frontocentral components between children with APD and their TD peers (Fox-Thomas, 2003; T. S. Mattsson et al., 2019).

According to a review article on the application of cortical auditory evoked potentials (AEPs) in the identification of APD, the variability in findings may be partially attributed to methodological heterogeneity (Macaskill et al., 2022). One methodological factor contributing to these inconsistencies is the limited number of studies in APD that have investigated the auditory cortical responses under ecologically valid listening conditions, such as in the presence of background noise. There are many studies examining AEPs in noise that have been conducted in typically developing (TD) children (Almeqbel & McMahon, 2015; Anderson, Chandrasekaran, et al., 2010; Benitez-Barrera et al., 2021; J. Cunningham et al., 2001; Gustafson et al., 2019; J.-H. Han et al., 2020; K. A. King et al., 2008; Salisbury et al., 2002; Ubiali et al., 2025; Ubiali et al., 2016; Zhang et al., 2018) which advanced the understanding of the neurophysiological mechanisms underlying speech perception in noisy environments. However, only a limited number of AEPs in noise studies have been conducted in children with APD (Hassaan, 2015; Hussain et al., 202; Morlet et al., 2019). Moreover, these mentioned studies haves applied only one single type of background noise, such as white noise or babble noise, which limits the ability to compare the effects of acoustical and informational masking and generalize findings across different listening environments (Hussain et al., 2022; Morlet et

al., 2019). Comparing auditory cortical responses across different types of background noise could yield important insights into the neurophysiology of auditory processing in noise. Additional studies investigating frontocentral AEPs under diverse noise and speech timing conditions are warranted to signify a more complete characterization of auditory processing and improve consistency in the literature.

Another methodological source of inconsistency in the APD population may be the limited variation in stimuli used across studies. Most investigations in this population have employed stimuli such as pure tones or speech tokens with similar fast temporal–spectral characteristics, such as /da/ or /ba/ and /ga/ (Hussain et al., 2022, Fox-Thomas, 2003; Koravand et al., 2017, Mattsson et al., 2019, Morlet et al., 2019, Macaskill et al., 2022) and barely investigated AEPs in response to more complex stimuli like speech sounds with varying temporal-spectral features, such as differences in voice onset time (VOT) (Hestvik et al., 2023). In natural speech, voice onset time (VOT) functions as a temporal cue for differentiating voiced from voiceless stop consonants (Sharma et al., 1997). Because consonants convey much of the information necessary for understanding word meaning, accurate neural encoding of the temporal features underlying consonant perception is critical for effective speech understanding in noisy listening conditions (Warrier et al., 2004). Therefore, understanding how noise modulates the cortical representation of various temporal speech cues, such as voice onset time (VOT), in children with APD is particularly important, given the frequently reported difficulties these children experience when understanding speech in everyday noisy environments (Clayson et al., 2019; Cunningham et al., 2002; Hestvik et al., 2023).

The last but important reason for inconsistent AEPs patterns in APD may lie in this fact that most of the these studies seem to look only at frontocentral responses (Ahmadi et al., 2024; Macaskill et al., 2022). To the best of our knowledge, auditory temporal responses or T-complex has not been investigated in children with APD. The following has reviewed the features of this response and it's potential applicability in the identification of auditory processing impairments at cortical levels.

1.4.1.3. Definitions and features of Temporal responses

T-complex is another type of late AEPs originating from the temporal lobe that can be measured at electrodes T7 and T8 electrodes (Bruneau et al., 2003; Wolpaw & Penry, 1975, 1977b). The three distinct deflections include: Na-a negative deflection that peaks at about 50ms, Ta-a positive deflection observed between 70 and 125ms, and Tb-a negative deflection

observed between 125 and 200ms after stimulation (McCallum & Curry, 1980; Näätänen & Picton, 1987; Wolpaw & Penry, 1975).

The T-complex and frontocentral responses differ in their cortical origins, dipole orientations (Figure 1 (Cohen & Halgren, 2003)), and maturational trajectories (Ponton et al., 2002; Wolpaw & Penry, 1975). It is assumed that the tangential dipole represents the activity in the depth of the Sylvian fissure (planum temporale, Heschl's gyrus) and the radial dipole, the activity in the secondary auditory areas at lateral regions of the upper temporal lobe (Picton et al., 1999).

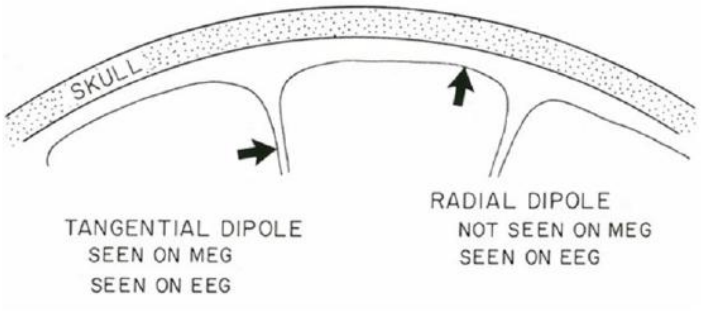

Figure 1: *The rotation of the dipoles. Two sources in the cortex; one is oriented tangentially to the skull, and the other is oriented radially. Each arrow is represented as a current dipole. Because sources in the cortex are generally oriented perpendicularly to the cortical surface, tangential sources are located in the sulci while radial sources are in the gyri (an exception is where the cortex turns away from the skulls, so that giri and sulci are reversed) (Cohen & Halgren, 2003).*

Specifically, the T-complex is primarily generated in the secondary auditory areas located in the posterior and lateral portions of the superior temporal gyrus (Brodmann areas 42 and 22), reflecting neural processing within the belt and para belt regions of the auditory cortex (Howard et al., 2000; Scherg & Von Cramon, 1986; Wolpaw & Penry, 1975). In contrast, frontocentral responses primarily originate from the primary auditory cortex located in Heschl's gyrus (Brodmann area 41) on the temporal plane (Wolpaw & Penry, 1975). Maturational studies have identified the robust presence of the Na, Ta, and Tb components as early as 5–8 years of age, with their morphology remaining relatively stable across development (Pang & Taylor, 2000; V. L. Shafer et al., 2015; Tonnquist-Uhlen et al., 2003). This pattern contrasts with the maturation of the P1–N1–P2 and N2 components, which typically emerge and stabilize later, around 8–11 years of age (Ponton et al., 2002; Ponton et al., 2000; Tonnquist-Uhlen et al., 2003). The earlier maturation of T-complex than frontocentral responses could emphasize the different underlying neural generators for these auditory responses (Mahajan & McArthur, 2013; Ponton et al., 2002; Ponton et al., 2000; Tonnquist-

Uhlen et al., 2003). The developmental pattern observed in subcomponents Ta and Tb implies a probable underlying neuronal localization within the deep layers of auditory cortex; layer IV of temporal cortex, as neurons in layer IV tend to mature earlier than those in layers II and III (Moore et al., 1997). The frontocentral responses like N1, in contrast, indexes neural activity in Layers II and III of superior temporal cortex and these layers generally mature later than Layer IV (Moore et al., 1997).

Numerous studies (Bruneau et al., 2015; Bruneau et al., 2003; Cacace et al., 1988; Carrillo-de-la-Peña, 1999; Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018; Connolly, 1993; Jarmo A. Hämäläinen et al., 2011) employing diverse study designs and stimulus have examined the characteristics of the T-complex. Similar to frontocentral responses, T-complex can be elicited with verbal and nonverbal stimuli (D. V. Bishop et al., 2012; Margriet Anna Groen et al., 2008; Hine & Debener, 2007) and modulated with changes in stimuli parameters like intensity, longer ISI and/or SOA (Clunies-Ross et al., 2018; Jarmo A. Hämäläinen et al., 2011). T-complex can represent the spectral-temporal attributes of speech (Silva et al., 2020; Wagner et al., 2016). Additionally, some studies have reported hemispheric differences, with distinct modulation patterns observed at the left (T7) and right (T8) temporal sites. These findings suggest that the T-complex reflects differential processing between the two hemispheres of the secondary auditory cortex (Bruneau et al., 2015; Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018).

Although various stimulus parameters influencing the T-complex have been extensively investigated, certain features, such as the representation of long versus short voice onset times (VOTs), have been scarcely examined in either adult or pediatric populations. VOT signifies the time interval between consonant release and the onset of low-frequency periodicity in the speech sound generated by rhythmic glottal pulsations, serving as a crucial temporal cue for discriminating stop consonants (Sharma & Dorman, 1999; Steinschneider et al., 1999). In American English, voiced stop consonants (ba, da, and ga) in syllable-initial positions exhibit short VOTs (0-20ms), while unvoiced stop consonants (pa, ta, and ka) have prolonged VOTs (30-100ms) (K. A. King et al., 2008; Sharma & Dorman, 1999; Steinschneider et al., 1999).

Only one study (Hoonhorst et al., 2009) has investigated the representation of long and short VOT on the T-complex in adult. In this study, the Na was recorded in response to syllables with onset /t/ and /d/ in quiet listening condition (Hoonhorst et al., 2009). Results showed that the Na, was double-peaked for long VOT values, while it was one solely peak for short VOTs (Hoonhorst et al., 2009). Little is still known about how long and short VOT could change the

appearance of the Ta and Tb components. A significant effect of VOT on the T-complex components is theoretically plausible, given evidence reporting the neural generators underlying the T-complex play a critical role in encoding rapid temporal dynamics at syllable onset (Belin et al., 2002; Belin et al., 2000; Rupp et al., 2022). Neuroimaging studies indicates that the superior temporal gyrus (STG), considered a primary neural generator of the T-complex, demonstrates greater activation in response to voiced relative to voiceless speech sounds (Belin et al., 2002; Belin et al., 2000; Rupp et al., 2022). Given the involvement of the T-complex generators in encoding voice onset time (VOT), examining T-complex responses to syllables with different VOTs may provide valuable insights into how the superior temporal gyrus processes the temporal features of speech.

Another parameter that appears to have been understudied in relation to the T-complex is its modulation in the presence of background noise. Consequently, the effect of noise on auditory processing within the secondary auditory cortex, as indexed by the T-complex, remains unclear. Considering the different neural generators of the T-complex and P1-N1-P2-N2 sequence, an examination of the T-complex under noisy conditions in response to various VOTs and comparing it to the auditory frontal responses may provide more insight into the neurophysiological underpinnings of auditory processing within different areas of the auditory cortex (i.e., primary and secondary auditory cortex). Moreover, such findings may have clinical relevance, as they improve identification and characterization of speech perception difficulties in young children who struggle to understand speech in noisy environments like APD, at the level of secondary auditory cortex.

1.4.1.4. T-complex, results in clinical populations and research gap

As part of the preliminary work for this thesis, a scoping review was conducted to determine whether any studies had examined the T-complex in children with APD. The results were published in 2024 (Ahmadi et al., 2024; see Appendix for the full review). A systematic search of the literature identified no published AEP studies reporting T-complex measures in an APD population.

The review also summarized evidence on T-complex patterns in neurodevelopmental conditions that may co-occur with APD. Across these clinical populations, atypical T-complex responses have been reported in children with language impairment (LI) (D. V. Bishop et al., 2012; Rinker et al., 2021; Shafer et al., 2011; Tonnquist-Uhlén, 1996b), Attention-deficit/hyperactivity disorder (ADHD) (Gomes et al., 2012), and dyslexia (Jarmo A. Hämäläinen et al., 2011; Taylor et al., 2006). Importantly, ADHD, LI and dyslexia have been

reported to show comorbidity with APD (de Wit et al., 2018; Gomez & Condon, 1999; Iliadou et al., 2009; Sharma et al., 2009).

Although these findings suggest that the T-complex can reflect atypical auditory cortical processing in several clinical groups, the scoping review indicated that T-complex responses have not yet been investigated in children with APD, particularly under noisy listening conditions (Ahmadi et al., 2024). This gap limits our understanding of whether the T-complex may serve as a neural indicator of the auditory processing difficulties that characterize APD and whether it has potential value as a clinically meaningful electrophysiological marker.

Furthermore, certain studies identified the discrepancies between T-complex and frontocentral responses in clinical populations (Bruneau et al., 1999; Gomes et al., 2012). In fact, there were atypical results of T-complex in Dyslexia and ADHD populations (Gomes et al., 2012; Hämäläinen et al., 2011;Taylor et al., 2003), despite of normal pattern of frontocentral responses. The different patterns observed between these frontocentral, and temporal responses could be the manifestation of the origins of the auditory deficits (Gomes et al., 2012; Hämäläinen et al., 2011;Taylor et al., 2003). Accordingly, examining both frontocentral responses and T-complex waveforms, using different background noise and using both speech and non-speech stimuli, in individuals with APD might provide information that is complementary to the more commonly reported frontocentral AEP components. Such research could bring important insights into the related auditory processing impairments and the origin of the deficit and, specifically, whether it arises from the superior temporal plane or the lateral superior temporal gyrus (Wagner et al., 2017).

Therefore, the present thesis investigates the effect of noise on frontocentral and temporal cortical auditory responses using different stimulus types in children with APD. The specific purpose and objectives of current thesis are elaborated in more details in the following sections.

1.5. Purpose and objectives

The primary objective of this thesis was to investigate the frontocentral and T-complex responses, recorded under both quiet and noisy conditions, in children with auditory processing disorder. However, interpreting AEP findings in APD requires a well-defined reference for typical responses. At the outset of this work, the literature showed important gaps regarding (1) how noise type and stimulus type modulate frontocentral responses and the T-complex in

typical populations, and (2) how the T-complex represents speech stimuli with different VOTs in typical listeners. Therefore, a series of experiments was first conducted in normal-hearing adults and children to characterize typical cortical auditory patterns under the same experimental manipulations. These data were then used to support interpretation of group differences in children with APD.

Accordingly, the thesis was organized into two phases: a typical phase and an APD phase. In the typical phase, two experimental lines were conducted in typical populations (adults and children): a Noise study and a Stimuli/VOT study. Building on these findings, the APD phase examined whether children with APD show atypical frontocentral and/or T-complex responses in quiet and noise, and whether VOT-related neural representations differ from those of typically developing peers. The objectives for each study are listed below.

1.5.1. Noise study (typical populations)

1. To investigate the effect of noise on T-complex in normal-hearing adults.
2. To investigate the effect of noise on T-complex in normal-hearing children.

1.5.2. Stimuli study long vs. short VOT (typical populations)

1. To investigate the representation of long and short VOTs in the T-complex in normal-hearing adults, and to assess corresponding behavioral measures of VOT perception.
2. To investigate the representation of long and short VOTs in the T-complex in normal-hearing children, and to assess corresponding behavioral measures of VOT perception.

1.5.3. APD study

1. To examine differences between TD and APD groups in frontocentral and T-complex responses under quiet and noisy conditions.

2. To examine the representation of long and short VOTs in the T-complex in children with APD, compare it with TD children, and assess corresponding behavioral measures of VOT perception.

1.5.4. Hypotheses

The objectives of this thesis have been based on the following hypotheses:

1.5.4.1. Effect of Noise

H1. Background noise will modulate both T-complex and frontocentral AEPs responses. Because these components have different neural generators, noise-related changes are expected to differ between temporal and frontocentral responses.

H2. The magnitude and direction of noise-related amplitude changes depend on stimulus type and noise type.

H3. The pattern of noise-induced changes of AEPs responses will differ between normal hearing adults and TD children, reflecting ongoing maturation of the auditory system in children.

1.5.4.2. Effect of stimuli (long and short VOTs)

H4. The T-complex will be modulated by VOT, such that cortical temporal responses will differ for short versus long VOT stimuli.

H5. VOT-related neural encoding will differ between children and adults consistent with developmental maturation of auditory cortical processing.

1.5.4.3. Effect of APD:

H6. Children with APD will show atypical auditory response morphology—particularly under noisy conditions—compared with TD peers.

H7. Children with APD will exhibit an atypical pattern of T-complex responses that may differ from patterns observed in frontocentral components.

H8. VOT representation (neural and/or behavioral) will differ between children with APD and TD children.

1.6. Summary of thesis project organization and manuscripts

To address the objectives of the thesis, a series of studies was conducted in normal hearing adults and TD children, followed by a study in children with APD. Electrophysiological recording of cortical auditory evoked potentials was obtained using auditory stimuli presented in quiet and noise conditions. The behavioral measurements were also collected during the electrophysiological recording sessions.

Findings addressing the objectives of Noise studies are reported in manuscript 1 and 2, whereas results from the stimuli (VOT) in manuscripts 3 and 4. All the manuscripts including manuscript 1to 4 (Ahmadi et al., 2026a published, 2026b acceptedd, 2026c submitted and 2026d submitted) have been submitted to journals for publication. Subsequently, data from the

APD population are analyzed and compared with TD children in manuscript 5 (see Figure 2). The thesis concludes with a general discussion and overall conclusion.

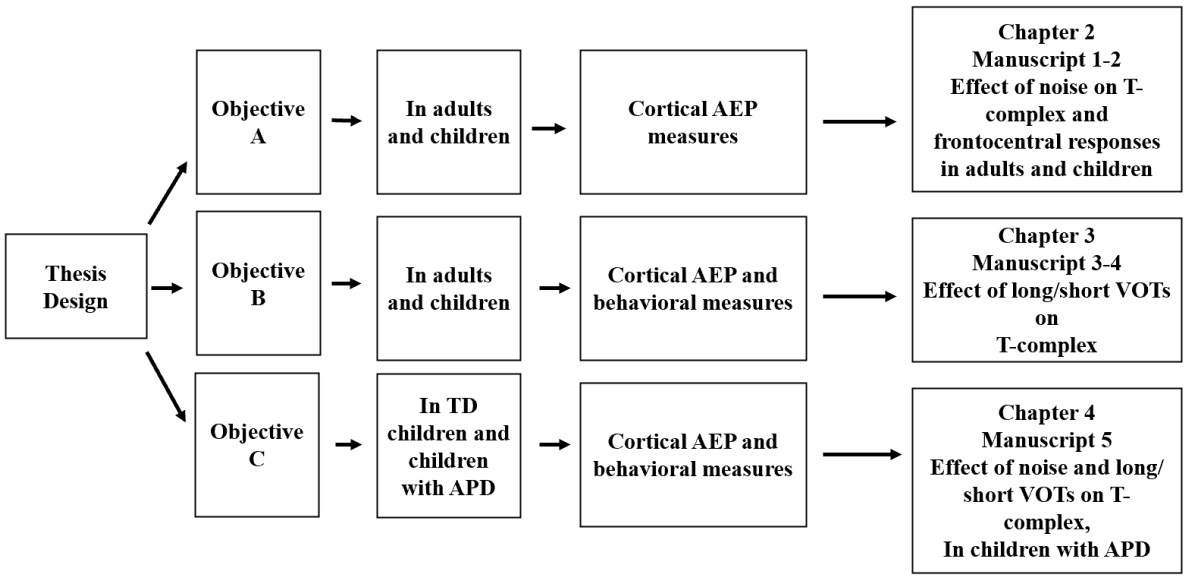

Figure 2: *Organization of the thesis project and associated manuscripts.*

Chapter 2. Manuscript 1

Effect of noise on T-complex responses: an evoked-related potential study

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Abstract

Cortical auditory evoked potentials (CAEPs), including the frontocentral (P1–N1–P2) and temporal (T-complex) components, have distinct neural generators. The T-complex appears to mature earlier and remains relatively stable with age. This stability may suggest that the T-complex may better reflect auditory processing in real-life listening conditions compared to frontocentral responses. One critical aspect of real-world listening is the presence of background noise; however, no previous study has examined its impact on the T-complex. The present study investigated the effects of background noise on both T-complex and frontocentral responses in adults during a passive paradigm, using /da/ and a 1-kHz tone pip presented in white and babble noise. In noise compared to quiet, the latency of all components was generally prolonged, and CAEP amplitudes were reduced. However, N1 and Ta amplitudes were enhanced in non-speech noise, and Tb amplitude was enhanced in babble noise. This enhancement pattern was observed only with speech stimuli. Larger CAEP amplitudes in noise may reflect top-down adaptive mechanisms that support speech understanding in adverse conditions. Notably, the larger Tb response to speech in babble noise suggests that the T-complex may index greater adaptation to real-life listening environments compared to frontocentral responses. The differences in CAEPs evoked by noise masked stimuli may highlight the various contributions of different auditory cortical structures to listening in noise. The T-complex also showed rightward lateralization in noise, consistent with findings from the primary auditory cortex.

Keywords

Cortical auditory responses, T-complex, noise-induced enhancement, lateralization.

Highlights

- The type of stimuli and noise can affect the pattern of noise-induced changes in auditory evoked potentials.
- Frontocentral and temporal responses showed distinct patterns of noise-induced changes.
- Speech stimuli may result in larger amplitude of CAEPs in the presence of background noise. This finding may show the adaptation of auditory processing to maintain the representation of speech stimuli in noise.

2.1. Introduction

Background noise interrupts everyday communication, requiring our auditory system to focus on relevant information while suppressing irrelevant sounds. Successful perception of speech in noise is dependent on hearing status and sound processing at peripheral, subcortical and cortical levels (Anderson, Skoe, et al., 2010; Bureš et al., 2025). Multiple behavioral studies have shown the detrimental effect of noise on speech perception (Dirks et al., 1982; Le Prell & Clavier, 2017; Lee et al., 2015; Meyer et al., 2013; Pittman & Wiley, 2001), though these approaches have limitations. Behavioral measures are influenced by non-auditory factors such as attention and language (Jerger & Musiek, 2000) and cannot directly assess the underlying neurophysiological mechanisms of speech processing in noise. Auditory evoked potentials (AEPs) provide an effective tool to evaluate auditory processing in the presence of background noise (Anderson, Skoe, et al., 2010). Prior research consistently shows that noise degrades auditory subcortical responses, resulting in delayed neural timing and reduced response amplitudes (Cunningham et al., 2002; Hecox et al., 1989; Parbery-Clark et al., 2011; Russo et al., 2005; Song et al., 2011).

At the cortical level, auditory evoked potentials can be categorized into two responses including frontocentral responses (i.e., P1-N1-P2-N2) and temporal responses (i.e., the T-complex). The P1–N1–P2–N2 sequence reflects auditory processes occurring primarily in the primary auditory cortex, particularly Heschl's gyrus, where the neural generators are tangentially oriented (Lindenberger & Baltes, 1994; Picton et al., 1999; Ponton et al., 2002; Wolpaw & Penry, 1975). The P1 component indexes pre-perceptual encoding of acoustic features such as frequency and timing and is thought to arise from the primary auditory cortex (Čeponienė et al., 2005; Liégeois-Chauvel et al., 1994). The N1 is an onset response generated by multiple sources, including the temporal and frontal lobes, thalamus, and midbrain reticular formation (Näätänen & Picton, 1987). It has both exogenous and endogenous properties, reflecting the presence of an audible sound (Näätänen & Picton, 1987) while becoming more pronounced under attention (Hillyard et al., 1973). The P2 appears to originate mainly from the anterolateral portions of Heschl's gyrus (Ross & Tremblay, 2009) and the insular cortex (Ponton et al., 2000) and is associated with the acoustic and temporal characteristics of stimuli (Näätänen & Picton, 1987). Finally, the N2 is thought to originate from activity in the supratemporal plane of the auditory cortex (Ceponiene et al., 2002). It reflects the physical discrimination of acoustic features and is linked to sensory auditory processing, being

modulated by attention, perception, discrimination, and recognition of sounds (Näätänen et al., 1982).

While subcortical responses exhibit consistent changes in the presence of background noise, cortical auditory evoked potentials (CAEP; P1-N1-P2-N2), show less consistent effect. Most studies report that noise degrades CAEPs, resulting in smaller amplitude and prolonged latency (Androulidakis & Jones, 2006; Billings et al., 2013; Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006; Russo et al., 2009; Katherine A. Whiting et al., 1998a). However, a few studies report that N1 amplitude is increased in noise (Alain et al., 2009; Duquette-Laplante et al., 2024; Papesh et al., 2015; Parbery-Clark et al., 2011), a pattern not observed for P1 and P2. It may suggest that the structures of the central auditory system that generate these waveforms may contribute differently to the processing of sound in noise.

The methodological differences likely account for the discrepancies in N1 findings. The enhanced N1 was recorded in studies employing binaural stimuli presentations at relatively fast rates (Alain et al., 2009; Parbery-Clark et al., 2011), whereas smaller N1 amplitude in noise was reported in studies using monaural presentations at slower rates (Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006). However, this distinction is not fully supported, as Papesh et al. (2015) reported CAEP in both monaural and binaural conditions (Papesh et al., 2015). Another possible explanation for the noise-induced enhancement is the modulation of auditory efferent system in the presence of background noise (Guinan, 2006; Huffman & Henson, 1990). Efferent pathways may facilitate the intelligibility of speech in noise through inhibition (Chintanpalli et al., 2012; Huffman & Henson, 1990; Scharf et al., 1997). This top-down inhibition may reduce the baseline level of spontaneous and/or excitatory activities that prolong the neural response to transient auditory events in noise (Alain et al., 2009; Haider et al., 2013). Animal studies have shown that the efferent stimulation can enhance auditory neurons responses leading to increase the signal-to-noise ratio for evoked potentials when stimuli are presented in noise (Kawase & Liberman, 1993; Tomchik & Lu, 2006). Hypothetically, it may be speculated that efferent system may be more sensitive to speech stimuli, allowing more efficient adaptation in noise and yielding stronger auditory responses to speech compared to non-speech stimuli.

More research on CAEPs in noise using speech and non-speech stimuli in various noises is needed to clarify whether stimulus type influences noise-induced enhancement. To our knowledge, only one study has examined the effect of both stimuli type (speech vs., non-speech) and noise type (non-speech noise vs. 8-talker babble noise) (Duquette-Laplante et al.,

2024). This study observed an N1 enhancement only when speech stimuli was delivered in the presence of non-speech noise. The author suggested that the speech stimuli were more salient than tonal stimuli in non-speech noise (Duquette-Laplante et al., 2024). However, because most of the CAEPs waves were not identifiable in 8-talker babble noise, comparison between non-speech stimuli and speech noise was not possible (Duquette-Laplante et al., 2024). Babble noise with fewer talkers, for example 4-talker appears to be more efficient in CAEP research, as studies have reported identifiable waveforms under this condition (Benítez-Barrera et al., 2021; Billings et al., 2011; B. R. Zendel et al., 2015). Although direct comparisons between non-speech noise and 4 talker babble have not been made, it is plausible that the N1 would be larger in the presence of non-speech noise and there would be no similar effect in babble noise. Therefore, further studies on CAEPs are needed to examine the effect of noise type using speech and non-speech stimuli. Moreover, existing research on CAEPs in noise has not addressed how background noise impacts temporal auditory evoked responses change. The T-complex, or temporal response, including Na-Ta-Tb sequence correspond to processes in secondary auditory areas, specifically the lateral superior temporal gyrus, where dipole sources are radially oriented (Lindenberger & Baltes, 1994; Uhlén et al., 1996; Wolpaw & Penry, 1975). The Na-Ta-Tb responses occur between 50-200ms, are typically observed at electrodes located at T7 and T8, and exhibit negative, positive and negative polarity, respectively. In addition to the different neural generators between the T-complex and frontocentral responses, these two responses mature at distinct ages (Albrecht et al., 2000; Billings et al., 2011; Pang & Taylor, 2000). Notably, developmental research has shown that the T-complex matures earlier than frontocentral responses and remains relatively stable with age (Bishop et al., 2011; Ponton et al., 2002; Tonnquist-Uhlen et al., 2003). The T-complex can be modulated by changes in stimulus intensity, interstimulus interval (ISI) and Stimulus Onset Asynchrony (SOA) (Clunies-Ross et al., 2018; Jarmo A. Hämäläinen et al., 2011). Verbal stimuli elicit a larger amplitude and/or shorter latency of the T-complex compared to non-verbal stimuli (D. V. Bishop et al., 2012; Margriet Anna Groen et al., 2008; Hine & Debener, 2007), and it also represents the spectral-temporal attributes of speech stimuli (Silva et al., 2020; Wagner et al., 2016). Additionally, some studies have reported hemispheric differences, with distinct modulation patterns observed at left (T7) versus right (T8) temporal sites, suggesting that the T-complex reflects differential processing in the two hemispheres of the secondary auditory cortex (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018).

Like frontocentral responses, the T-complex can be affected by stimulus features, however, to the best of our knowledge, no study evaluated noise-induced changes in the T-complex or

compared these patterns to noise-induced changes in frontocentral responses. Consequently, the effect of noise on auditory processes in secondary auditory as indexed by the T-complex areas remains unclear.

The aim of this study was to evaluate the effect of noise on the T-complex and to compare these effects with those observed in the frontocentral responses. In addition, this study assessed whether CAEPs are differentially modulated by stimulus type (speech vs. non-speech) and noise type (non-speech noise vs. 4-talker babble).

Considering the different neural generators of the T-complex and P1-N1-P2-N2 sequence, an examination of the T-complex under noisy conditions and comparing it to the auditory frontal responses may provide more insight into the neurophysiological underpinnings of auditory processing within different areas of the auditory cortex (i.e., primary and secondary auditory cortex). Moreover, such findings may have clinical relevance, as they could inform our understanding of how noise affects processing in secondary auditory areas in populations with communication disorders, such as people with auditory processing disorder, whose main complaint is difficulty understanding speech in noisy environments.

2.1.1. Hypotheses

Given the greater stability of T-complex responses throughout life, it is hypothesized that the T-complex may better represent the auditory processing in real-life noisy listening conditions than frontocentral responses. This suggests that the T-complex may better represent auditory information in the presence of noise. Moreover, if the larger auditory responses in noise reflect the top-down adaptive mechanisms acting on speech processing, then the noise-induced enhancement in CAEPs is expected to occur primarily in response to speech stimuli rather than non-speech stimuli. Additionally, given the distinct stimulus-induced modulation patterns of the T-complex at electrodes T7 and T8, differential effects of noise on the T-complex are anticipated between these two sites.

2.2. Method

2.2.1. Ethics approval

The University of Ottawa Research Ethics Board approved this study, Ethic file number=H-07-24-10451. All participants received and signed a written consent form.

2.2.2. Participants

Twenty students aged 18 to 30 years (M = 28, SD = 4.6; 9 males) participated in this study.

2.2.3. Inclusion and exclusion criteria

All participants had normal audiometric thresholds (≤15 dB HL) at octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996) protocol. Tympanograms were normal, showing an admittance curve with a single peak between +50 and –100 daPa using a 226-Hz probe tone. Participants were right-handed and fluent in English for both speaking and listening, as determined by the Language Experience and Proficiency Questionnaire (LEAP-Q) (Marian et al., 2007). No history of otological or neurological disorders was reported by any of the adult participants. In addition, participants with musical training were excluded, as previous studies have shown that music practice can enhance speech-in-noise perception and increase the amplitude of CAEPs in the presence of background noise compared to quiet (Anderson et al., 2013; Benjamin Rich Zendel et al., 2015).

2.2.4. Procedure

During the experiment, after familiarization with the testing equipment and procedures, participants were seated in a sound-attenuated room to complete the tasks. They were instructed to ignore the auditory stimuli and focus their attention on a silent, subtitled movie

2.2.5. Stimuli

Participants completed two experiments as part of this study; here we report the results of studies that explored the effect of noise on CAEP using non-speech stimuli, and the effect of noise on CAEP using a speech stimulus. The second study explored the manipulation of voice onset time of a speech stimulus, and the results will be reported elsewhere. Two experiments were conducted in one day, starting with the first experiment followed by the second experiment.

The non-speech stimulus was a pure tone 1kHz with 250ms duration and 10ms rise and fall time, created in Audacity software (Audacity Team, 1999). The speech stimulus was a natural /da/, it was originally recorded by a male voice and used in the study of Dubno & Schaefer (1992). The speech stimuli had the same duration (250ms) and rise-fall time (10ms) as non-speech stimuli. Stimuli were presented using insert earphones (EARTone 3A) in three listening conditions including quiet, in the presence of babble noise and in the presence of non-speech noise in experiment 1, there were three blocks of 1kHz in three listening conditions including

quiet, non-speech noise and babble noise. In the experiment 2, considering the goal of the study, each block included /da/ and /ta/, and three blocks in total were delivered in three listening conditions. The order of listening conditions was random between subjects. Non-speech noise (similar to white noise but was band-pass limited to have equal energy from 250-6000 Hz was created and extracted from Audacity software (Audacity Team, 1999). English four-talker babble noise was the same as Zendel et al, 2015 (Benjamin Rich Zendel et al., 2015). Since hearing in noise is typically a binaural process, it is important to implement test measures that increase the ecological validity, or the extent to which the results can generalize to real-life listening situations. The stimuli were delivered in a passive non-oddball, homogenous paradigm, because Oddball paradigms introduce cognitive variables such as attention and stimulus context effects, which complicate the interpretation of earlier CAEP components (Billings et al., 2011). Stimuli were equalized for root mean square (rms) amplitude and then played at 80 dB SPL. For noisy conditions, signal-to-noise ratio was kept as +5dB, by reducing the intensity of noise to 75dB SPL. The SNR= +5dB, because it seems that CAEPs are still recognizable in this level of noise, as previous studies could detect reliable responses at SNR= +5dB (Bidelman & Howell, 2016; C. J. Billings et al., 2017; Curtis J. Billings et al., 2009; Cunningham et al., 2002; Duquette-Laplante et al., 2024). Interstimulus interval (ISI) was randomly between 1100-1250ms.The number of presentations of each stimulus was 300 in each block, for each listening condition, which were delivered by E-prime software. The whole project including the two paradigms took around two hours, 30 minutes for non-speech experiment and 60 minutes for speech stimuli experiment with few short breaks between the blocks and experiments to decrease fatigue.

2.2.6. Electrophysiological recording

The electrical responses were recorded from 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). The electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp, using 10-20 standard system. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. The tip of the nose served as an online reference for all channels, including the electro-oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. The EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for later analyses.

2.2.7. Electrophysiological data analysis

The data were analysed using Brain Products' Analyzer2 software. The signals were digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts were corrected in the EEG using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1996). To achieve this, vertical and horizontal EOG activity was computed. Vertical EOG was derived by subtracting the inferior orbital channel from FP2, while horizontal eye movements were obtained by subtracting FT9 from FT10 (Duda-Milloy et al., 2019). The ICA model assumes that the observed electrophysiological signals represent a linear mixture of neural sources and artifacts that are mutually statistically independent. Using blind source separation, the algorithm was trained to identify each participant's ocular artifact signature, which was then removed from the EEG traces. Epochs were excluded if they had amplitudes exceeding ±100μV at any site. Less than 10% of trials were excluded. Moreover, the EEG was visually inspected to ensure adequate artifact rejection. Epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset. Segmented waveforms were baseline corrected around the 100ms pre-stimulus interval and averaged based on condition. Although both Cz and Fz were selected for the detection of frontocentral responses (P1-N1-P2), only the responses at Cz are reported as there were more robust responses at Cz compared to Fz (Näätänen & Picton, 1987). Additionally, the amplitude of the N1 at Cz has been considered the strongest predictor of individual speech-in-noise perception thresholds (Billings et al., 2013). The T-complex was extracted from the temporal regions, T7 and T8 (Wolpaw & Penry, 1975). Tonnquist-Uhlen and colleagues (2003) also suggest that T7 and T8 are optimal for measuring the T-complex since they reflect little activity from the sources underlying the P1, N1 and P2. Regarding T-complex components, as Na was not detected clearly in most of the participants, only Ta and Tb are reported in the results.

A peak detection procedure was performed for each participant to measure peak latency for each component. Regarding amplitude measurements a mean amplitude method was applied. Mean amplitude rather than peak amplitude is less affected by noise, leading to more efficient responses (Clayson et al., 2013; Luck, 2012). The mean amplitudes were calculated based on an interval between two points (25ms) on either side of the peak (i.e., a 50ms window) for each peak. This time window was based on the grand averaged ERPs at sites Cz, T7 and T8 (Anderson, Chandrasekaran, et al., 2010; J. A. Hämäläinen et al., 2011). The latency intervals over which each of the mean amplitudes were calculated are reported in Table 1.

Table 1. *Latency intervals over which each of the mean amplitudes were calculated.*

Peak-site	The latency interval based on Listening condition- Stimuli					
	Q-1kHz	**Q-da**	**W-1kHz**	**W-da**	**B-1kHz**	**B-da**
P1	43-93ms	43-93ms	47-97ms	67-117ms	47-97ms	77-137ms
N1	89-139ms	91-141ms	93-143ms	123-173ms	97-147ms	195-245ms
P2	149-199ms	163-213ms	167-217ms	195-245ms	161-211ms	319-369ms
Ta-T7	89-139ms	93-143ms	95-145ms	127-177ms	99-149ms	197-247ms
Tb-T7	143-193ms	155-205ms	153-203ms	185-235ms	155-205ms	329-379ms
Ta-T8	91-141ms	87-137ms	99-149ms	125-175ms	95-145ms	195-245ms
Tb-T8	143-193ms	161-211ms	157-207ms	191-241ms	159-209ms	317-367ms

Q: Quiet, W: Non-speech noise, B: Babble noise.

2.2.8. Statistical analysis

Statistical analyses were conducted using JASP (Jeffreys's Amazing Statistics Program). Repeated-measures analyses of variance (ANOVAs) were performed to compare the amplitude and latency of the CAEPs. Prior to analysis, data were screened to ensure that the assumptions of ANOVA were met. The normality of residuals was assessed using Q–Q plots generated in JASP, which indicated that the residuals were approximately normally distributed, as the points closely followed the diagonal line without substantial deviations. When Mauchly's test of sphericity was violated, the Greenhouse–Geisser correction was automatically applied to adjust the degrees of freedom

A 2 (Stimuli: tone, speech) by 3 (Listening condition: no noise (quiet), non-speech noise, babble noise) repeated measures ANOVA was used to calculate separately latency and amplitude of P1, N1 and P2. A 2 (Electrode location: T7, T8) by 2 (Stimuli: tone, speech) by 3 (Listening condition: no noise (quiet), non-speech noise (marked as W), babble noise) repeated measures ANOVA was used to calculate separately latency and amplitude of Ta and Tb. The level of significance was set at 5% and statistically significant values were addressed as ($p<0.05$).

In addition, an exploratory analysis was conducted to examine whether individual factors such as age or sex moderated noise-related effects. For this purpose, age was included as a continuous covariate and sex as a between-subjects factor, along with their interactions with listening condition and stimulus type. These analyses were considered exploratory, as the study was not designed or powered to assess individual-difference effects, and the age range was restricted to adults only.

2.3. Results

The following sections describe the primary effects of listening conditions, stimulus type and electrode location on auditory evoked potentials. Exploratory analyses examining whether age or sex moderated the primary effects did not show reliable interaction effects for P1, N1, P2, or T-complex measures (all p-values > .05).

2.3.1. Amplitude

2.3.1.1. Temporal responses, Ta peak

Repeated-measures ANOVA (Table 2) revealed no significant main effect of listening condition, $F(2, 38) = 1.896$, $p = .164$, $\eta^2 = .009$. However, significant main effects were found for stimuli, $F(1, 19) = 7.559$, $p = .013$, $\eta^2 = .032$, and electrode location, $F(1, 19) = 9.849$, $p = .005$, $\eta^2 = .070$. These effects were qualified by a significant three-way interaction among listening condition, stimuli, and electrode location, $F(2, 38) = 13.704$, $p < .001$, $\eta^2 = .042$. All two-way interactions were also significant: listening condition × stimuli, $F(1, 38) = 61.811$, $p < .001$, $\eta^2 = .218$; listening condition × electrode location, $F(1, 38) = 13.704$, $p < .001$, $\eta^2 = .042$; and stimuli × electrode location, $F(1, 19) = 25.569$, $p < .001$, $\eta^2 = .073$. Given the significant three-way interaction, follow-up two-way ANOVAs were conducted separately for each stimulus, electrode, and listening condition.

Table 2. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on amplitude of Ta.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	1.311[a]	2[a]	0.656[a]	1.896[a]	0.164[a]	0.009
Residuals	13.145	38	0.346			
Stimuli	4.923	1	4.923	7.559	0.013	0.032
Residuals	12.373	19	0.651			
Location	10.668	1	10.668	9.849	0.005	0.070
Residuals	20.580	19	1.083			
Listening condition ✻ Stimuli	33.084	2	16.542	61.811	< .001	0.218
Residuals	10.170	38	0.268			
Listening condition ✻ Location	6.372	2	3.186	13.704	< .001	0.042
Residuals	8.834	38	0.232			
Stimuli ✻ Location	11.061	1	11.061	25.569	< .001	0.073
Residuals	8.220	19	0.433			

Table 2. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on amplitude of Ta.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition ✻ Stimuli ✻ Location	4.387[a]	2[a]	2.194[a]	12.452[a]	< .001[a]	0.029
Residuals	6.695	38	0.176			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

A two-way ANOVA was done to evaluate the interaction between Listening condition and Electrode for each Stimulus, separately (Table 3 and 4).

Table 3. *The two-way ANOVA to assess the interaction between Listening condition and Electrode, with 1kHz.*

Cases	Sum of Squares	df	Mean Square	F	p
Listening condition	19.482	2	9.741	35.803	< .001
Residuals	10.339	38	0.272		
Electrode	21.728	1	21.728	26.549	< .001
Residuals	15.549	19	0.818		
Listening condition ✻ Electrode	10.265	2	5.133	20.470	< .001
Residuals	9.528	38	0.251		

Note. Type III Sum of Squares

Table 4. *The two-way ANOVA to assess the interaction between Listening condition and Electrode, with da.*

Cases	Sum of Squares	df	Mean Square	F	p
Listening condition	14.913	2	7.456	21.837	< .001
Residuals	12.976	38	0.341		
Electrode	0.002	1	0.002	0.003	0.960
Residuals	13.250	19	0.697		
Listening condition ✻ Electrode	0.494	2	0.247	1.565	0.222

Table 4. *The two-way ANOVA to assess the interaction between Listening condition and Electrode, with da.*

Cases	Sum of Squares	df	Mean Square	F	p
Residuals	6.001	38	0.158		

Note. Type III Sum of Squares

The results showed that the Electrode × Listening condition interaction depended on stimulus type and was significant only for the 1-kHz tone (Figure 1). Follow-up *t*-tests with Bonferroni correction confirmed that Ta was significantly larger at T8 than at T7 in non-speech noise ($p < .001$) and babble noise ($p < .001$). However, no such difference was observed in quiet ($p = 1.000$).

A two-way ANOVA was done to evaluate the interaction between Listening condition and Stimuli for each Electrode, separately (Table 5 and 6).

Table 5*. The two-way ANOVA to assess the interaction between Listening condition and Electrode, at T7.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	4.896	2	2.448	7.308	0.002	0.060
Residuals	12.730	38	0.335			
Stimuli	15.371	1	15.371	43.391	< .001	0.188
Residuals	6.731	19	0.354			
Listening condition ✻ Stimuli	29.954[a]	2[a]	14.977[a]	47.896[a]	< .001[a]	0.367
Residuals	11.883	38	0.313			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

Table 6. *The two-way ANOVA to assess the interaction between Listening condition and Electrode, at T8.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	2.787[a]	2[a]	1.394[a]	5.725[a]	0.007[a]	0.071
Residuals	9.249	38	0.243			
Stimuli	0.613	1	0.613	0.840	0.371	0.016
Residuals	13.862	19	0.730			
Listening condition ✻ Stimuli	7.517	2	3.759	28.669	< .001	0.193
Residuals	4.982	38	0.131			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

The Listening condition × Stimuli interaction was significant at both T7 and T8 (Figure 1). Follow-up *t*-tests with Bonferroni correction showed that at T7, for the 1-kHz tone, Ta amplitude was smaller in non-speech noise ($p < .001$) and babble noise ($p < .001$). At the same electrode, for /da/, Ta amplitude was larger in non-speech noise ($p < .001$), with no significant effect of babble noise ($p = .24$). At T8, for the 1-kHz tone, there was no significant effect of either non-speech noise ($p = .352$) or babble noise ($p = .163$). For /da/ at T8, Ta amplitude was larger in non-speech noise ($p < .001$) and marginally larger in babble noise ($p = .054$).

A two-way ANOVA was done to evaluate the interaction between Electrode and Stimuli for each Listening condition, separately (Table 7, 8 and 9).

Table 7*. The two-way ANOVA to assess the interaction between Electrode and Stimuli, Q.*

Cases	Sum of Squares	df	Mean Square	F	p
Stimuli	9.919	1	9.919	23.322	< .001
Residuals	8.081	19	0.425		
Electrode	0.005	1	0.005	0.007	0.936

Table 7*. The two-way ANOVA to assess the interaction between Electrode and Stimuli, Q.*

Cases	Sum of Squares	df	Mean Square	F	p
Residuals	14.717	19	0.775		
Stimuli ✻ Electrode	0.390	1	0.390	1.858	0.189
Residuals	3.991	19	0.210		

Note. Type III Sum of Squares

Table 8. *The two-way ANOVA to assess the interaction between Electrode and Stimuli, W.*

Cases	Sum of Squares	df	Mean Square	F	p
Stimuli	23.478	1	23.478	41.784	< .(
Residuals	10.676	19	0.562		
Electrode	11.723	1	11.723	21.699	< .(
Residuals	10.265	19	0.540		
Stimuli ✻ Electrode	12.495	1	12.495	36.734	< .(
Residuals	6.463	19	0.340		

Note. Type III Sum of Squares

Table 9*. The two-way ANOVA to assess the interaction between electrode and stimuli, B.*

Cases	Sum of Squares	df	Mean Square	F	p
Stimuli	4.610	1	4.610	23.134	< .001
Residuals	3.786	19	0.199		
Electrode	5.312	1	5.312	22.769	< .001
Residuals	4.432	19	0.233		
Stimuli ✻ Electrode	2.563	1	2.563	10.919	0.004
Residuals	4.460	19	0.235		

Note. Type III Sum of Squares

The Electrode × Stimuli interaction was significant only under noisy conditions (Figure 1). Follow-up *t*-tests showed that for the 1-kHz tone, Ta amplitude was significantly larger at T8

than at T7 in both non-speech noise and babble noise ($p < .001$), whereas no significant electrode effect was observed for /da/ ($p > .05$).

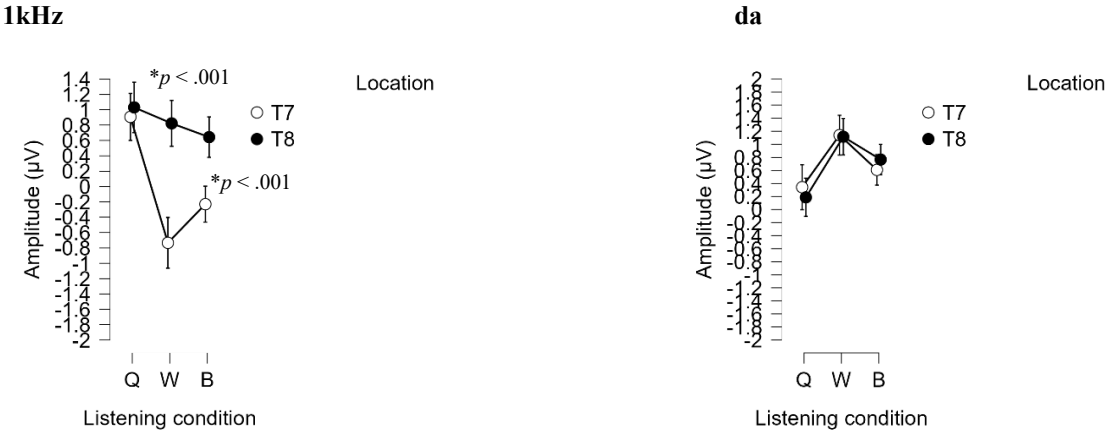

Figure 1 *The mean amplitude of Ta, to da and 1 kHz, in quiet (Q), non-speech noise (W) and babble noise (B), at T7 and T8. The most significant conditions have been marked with star symbol : *.*

2.3.1.2. Temporal responses, Tb peak

Repeated-measures ANOVA (Table 10) revealed significant main effects of listening condition, $F(2, 38) = 24.749$, $p < .001$, $\eta^2 = .213$; stimuli, $F(1, 19) = 20.752$, $p < .001$, $\eta^2 = .076$; and location, $F(1, 19) = 10.437$, $p = .004$, $\eta^2 = .063$. Significant interactions were also observed between listening condition and stimuli, $F(2, 38) = 7.752$, $p = .020$, $\eta^2 = .024$, and between stimuli and location, $F(2, 38) = 5.587$, $p = .029$, $\eta^2 = .024$. Follow-up *t*-tests with Bonferroni correction showed that for the 1-kHz tone, there was no significant difference in Tb amplitude between non-speech noise and quiet ($p = 1.000$). However, Tb amplitude was significantly smaller in babble noise compared to quiet ($p < .030$; Figure 2). For /da/, Tb amplitude was significantly different between non-speech noise and quiet ($p = .025$), babble noise and quiet ($p < .001$), and babble noise and non-speech noise ($p < .001$; Figure 2)

Follow-up *t*-tests showed that the effect of location was significant only for /da/ ($p < .001$; Figure 2). With speech stimuli, Tb amplitude was larger at T8 than at T7 across all listening conditions. Notably, in quiet, Tb amplitude was larger at T7 than at T8, whereas in both noise conditions it was larger at T8 than at T7. The mean differences were 0.59, 0.40, and 0.70 μV in quiet, non-speech noise, and babble noise, respectively. In addition, the effect of stimulus type depended on location, being significant only at T8 ($p < .001$) and not at T7 ($p = 1.000$).

Table 2. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on amplitude of Tb.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	25.300	2	12.650	24.749	< .001	0.213
Residuals	19.423	38	0.511			
Stimuli	9.065	1	9.065	20.752	< .001	0.076
Residuals	8.299	19	0.437			
Location	7.456	1	7.456	10.437	0.004	0.063
Residuals	13.573	19	0.714			
Listening condition ✻ Stimuli	2.827	2	1.414	7.752	0.002	0.024
Residuals	6.930	38	0.182			
Listening condition ✻ Location	0.588	2	0.294	1.778	0.183	0.005
Residuals	6.280	38	0.165			
Stimuli ✻ Location	2.800	1	2.800	5.587	0.029	0.024
Residuals	9.524	19	0.501			
Listening condition ✻ Stimuli ✻ Location	0.317	2	0.159	0.946	0.397	0.003
Residuals	6.372	38	0.168			

Note. Type III Sum of Squares
Effect sizes were reported as η^2 and interpreted using conventional benchmarks (.01 = small, .06 = medium, .14 = large).

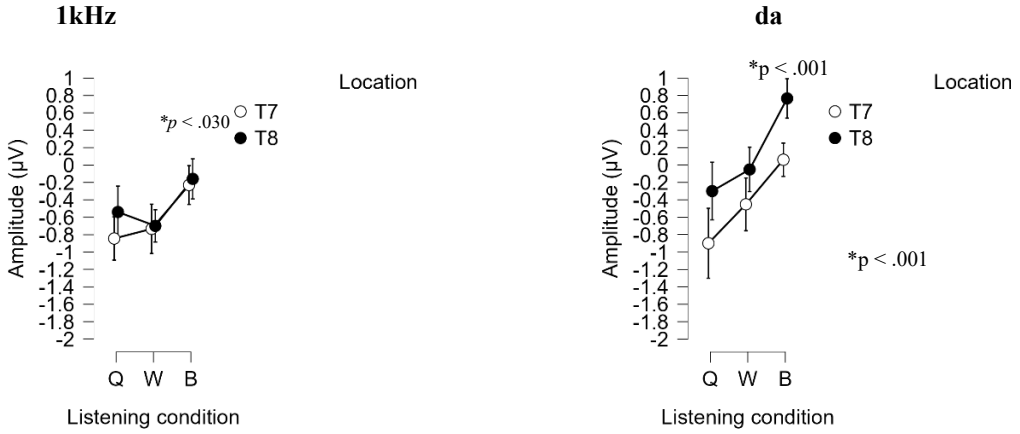

Figure 2 *The mean amplitude of Tb, to da and 1 kHz, in Quiet, Non-speech noise, and Babble noise, at T7 and T8. The most significant conditions have been marked with star symbol : *.*

2.3.1.3. Frontocentral responses, P1 peak

Repeated-measures ANOVA (Table 11) revealed a significant main effect of listening condition, $F(2, 38) = 14.221$, $p < .001$, $\eta^2 = .229$. No significant main effect of stimuli was observed, $F(1, 19) = 0.459$, $p = .506$, $\eta^2 = .005$, and the interaction between listening condition and stimuli was not significant, $F(2, 38) = 1.180$, $p = .318$, $\eta^2 = .016$. Follow-up *t*-tests with Bonferroni correction showed that P1 amplitude was significantly smaller in non-speech noise than in quiet ($p < .001$) and in non-speech noise than in babble noise ($p < .001$). No significant effect of babble noise was observed (Figure 3).

Table 3. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on amplitude of P1.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	3.336	2	1.668	14.221	< .001	0.229
Residuals	4.457	38	0.117			
Stimuli	0.069	1	0.069	0.459	0.506	0.005
Residuals	2.835	19	0.149			
Listening condition ⁎ Stimuli	0.226	2	0.113	1.180	0.318	0.016
Residuals	3.639	38	0.096			

Note. Type III Sum of Squares

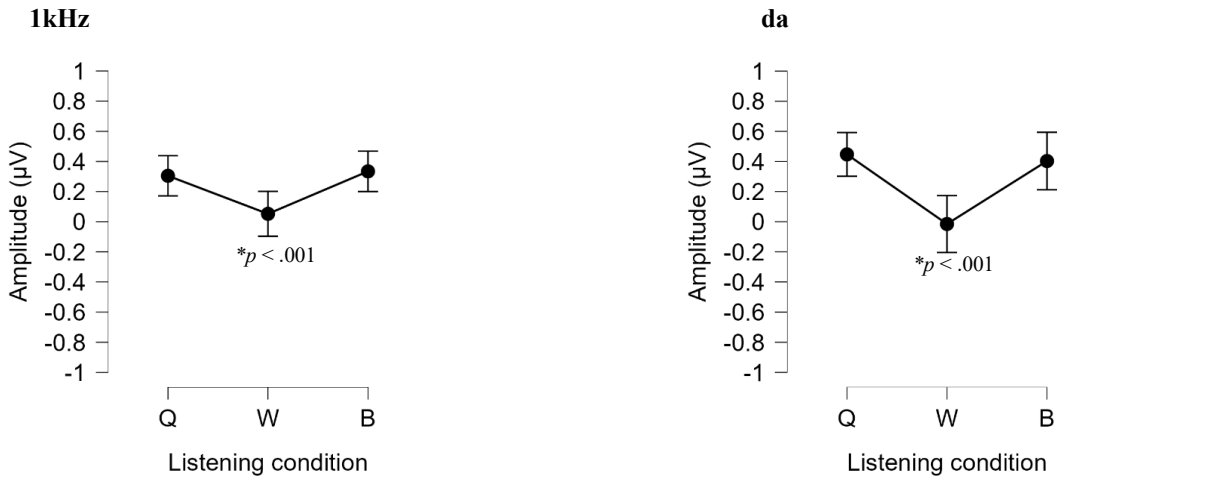

Figure 3. *The mean amplitude of P1, to da and 1 kHz, in quiet (Q), non-speech noise (W) and babble noise (B). The most significant conditions have been marked with star symbol : *.*

2.3.1.4. Frontocentral responses, N1 peak

Repeated-measures ANOVA (Table 12) revealed a significant main effect of listening condition, $F(2, 38) = 13.196$, $p < .001$, $\eta^2 = .180$. No significant main effect of stimuli was observed, $F(1, 19) = 0.540$, $p = .471$, $\eta^2 = .006$. However, the interaction between stimuli and listening condition was significant, $F(2, 38) = 31.763$, $p < .001$, $\eta^2 = .223$. Follow-up *t*-tests with Bonferroni correction showed that for the 1-kHz tone, N1 amplitude was significantly smaller in non-speech noise than in quiet ($p = .008$), in babble noise than in quiet ($p < .001$), and in babble noise than in non-speech noise ($p = .003$; Figure 6). For /da/, N1 amplitude was significantly larger in non-speech noise than in quiet ($p < .001$) and in babble noise ($p = .002$). However, no significant difference was found between babble noise and quiet ($p = 1.000$; Figure 4).

Table 4. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on amplitude of N1.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	7.012	2	3.506	13.196	< .001	0.180
Residuals	10.097	38	0.266			
Stimuli	0.219	1	0.219	0.540	0.471	0.006
Residuals	7.692	19	0.405			
Listening condition ⁎ Stimuli	8.680	2	4.340	31.763	< .001	0.223
Residuals	5.192	38	0.137			

Note. Type III Sum of Squares

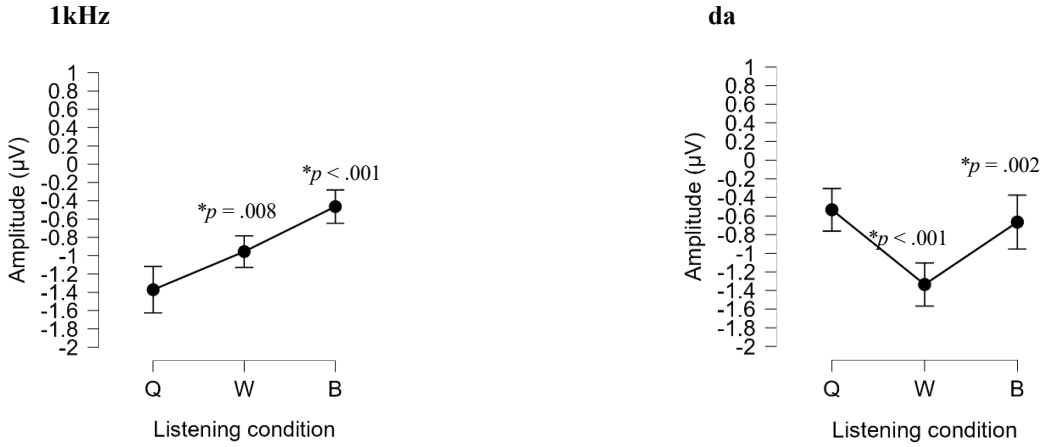

Figure 4. *The mean amplitude of N1, to da and 1 kHz, in quiet (Q), non-speech noise (W) and babble noise (B). The most significant conditions have been marked with star symbol : *.*

2.3.1.5. Frontocentral responses, P2 peak

Repeated-measures ANOVA (Table 13) revealed a significant main effect of listening condition, $F(2, 38) = 34.414$, $p < .001$, $\eta^2 = .530$. No significant main effect of stimuli was observed, $F(1, 19) = 0.040$, $p = .844$, $\eta^2 = .0001$, and the interaction between factors was not significant, $F(2, 38) = 2.458$, $p = .099$, $\eta^2 = .012$. Follow-up *t*-tests with Bonferroni correction showed that P2 amplitude was significantly smaller in non-speech noise than in quiet ($p = .003$), in babble noise than in quiet ($p < .001$), and in babble noise than in non-speech noise ($p < .001$; Figure 5).

Table 5. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on amplitude of P2.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	40.564[a]	2[a]	20.282[a]	34.414[a]	< .001[a]	0.536
Residuals	22.396	38	0.589			
Stimuli	0.010	1	0.010	0.040	0.844	1.371×10^{-4}
Residuals	4.981	19	0.262			
Listening condition ✻ Stimuli	0.887	2	0.444	2.458	0.099	0.012
Residuals	6.857	38	0.180			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

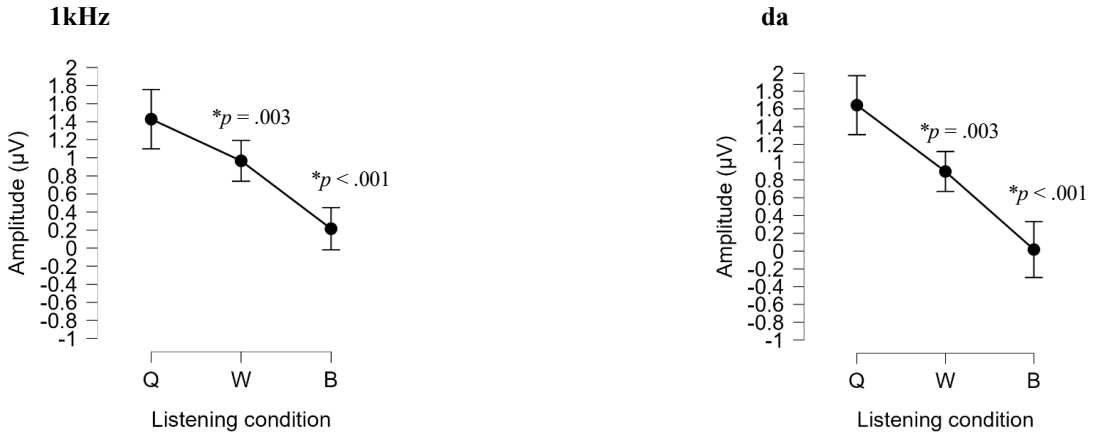

Figure 5 *The mean amplitude of P2, to da and 1 kHz, in quiet (Q), non-speech noise (W) and babble noise (B). The most significant conditions have been marked with star symbol : *.*

2.4. Latency

2.4.1. Temporal responses, Ta peak

Repeated-measures ANOVA (Table 14) revealed significant main effects of listening condition, $F(2, 38) = 146.499$, $p < .001$, $\eta^2 = .328$, and stimuli, $F(1, 19) = 417.083$, $p < .001$, $\eta^2 = .277$. No significant effect of location (T7 vs. T8) was observed, $F(1, 19) = 0.010$, $p = .900$, $\eta^2 = .001$. Significant interactions were found between listening condition and stimuli, $F(2, 38) = 83.397$, $p = .043$, $\eta^2 = .203$, and between listening condition and location, $F(2, 38) = 3.435$, $p < .001$, $\eta^2 = .003$. Follow-up *t*-tests with Bonferroni correction confirmed the effect of listening condition, showing longer latency in non-speech noise versus quiet, babble noise versus quiet, and babble noise versus non-speech noise for both stimuli ($p < .01$; Figure 8). However, no significant differences were observed between T7 and T8 in any listening condition ($p > .05$; Figure 6).

Table 6. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on latency of Ta.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	122533.104[a]	2[a]	61266.552[a]	146.499[a]	< .001[a]	0.328
Residuals	15891.751	38	418.204			
Stimuli	103369.448	1	103369.448	417.083	< .001	0.277
Residuals	4708.941	19	247.839			
Locations	6.393	1	6.393	0.014	0.906	1.712×10^{-5}
Residuals	8438.050	19	444.108			
Listening condition ✻ Stimuli	75866.829[a]	2[a]	37933.415[a]	83.397[a]	< .001[a]	0.203
Residuals	17284.339	38	454.851			
Listening condition✻ Locations	1279.774	2	639.887	3.435	0.043	0.003
Residuals	7079.021	38	186.290			
Stimuli ✻ Locations	171.417	1	171.417	0.389	0.540	4.590×10^{-4}
Residuals	8365.412	19	440.285			
Listening condition ✻ Stimuli ✻ Locations	322.063	2	161.032	0.751	0.479	8.624×10^{-4}
Residuals	8147.965	38	214.420			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

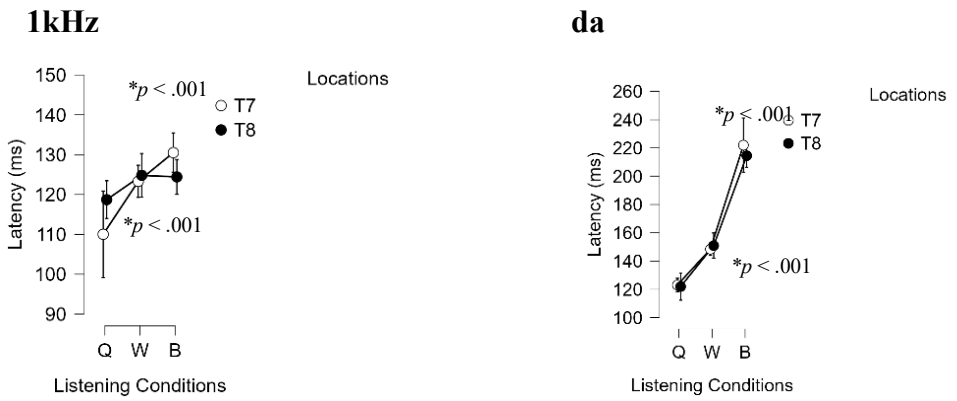

Figure 6. *The average of Ta latency to 1kHz and da, in quiet (Q), non-speech noise (W) and babble noise (B), at T7 and T8. The most significant conditions have been marked with star symbol : *.*

2.4.2. Temporal responses, Tb peak

Repeated-measures ANOVA (Table 15) revealed significant main effects of listening condition (quiet, non-speech noise, and babble noise), $F(2, 38) = 334.004$, $p < .001$, $\eta^2 = .356$, and stimuli, $F(1, 19) = 454.589$, $p < .001$, $\eta^2 = .293$. No significant main effect of location (T7 vs. T8) was observed, $F(1, 19) = 2.392$, $p = .138$, $\eta^2 = .001$. The only significant interaction was between listening condition and stimuli, $F(2, 38) = 371.992$, $p < .001$, $\eta^2 = .295$. Follow-up *t*-tests with Bonferroni correction confirmed the effect of listening condition for both stimuli ($p < .01$), showing longer latency in non-speech noise versus quiet, babble noise versus quiet, and babble noise versus non-speech noise (Figure 7).

Table 7. *The results of Repeated Measures ANOVA evaluating the effect Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on latency of Tb.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening Condition	259640.558[a]	2[a]	129820.279[a]	334.004[a]	< .001[a]	0.356
Residuals	14769.775	38	388.678			
Stimuli	213785.704	1	213785.704	454.589	< .001	0.293
Residuals	8935.379	19	470.283			
Location	885.504	1	885.504	2.392	0.138	0.001
Residuals	7033.579	19	370.188			
Listening Condition ✻ Stimuli	193382.408	2	96691.204	371.992	< .001	0.265
Residuals	9877.258	38	259.928			
Listening Condition ✻ Location	660.058	2	330.029	1.589	0.217	9.040×10^{-4}
Residuals	7891.608	38	207.674			
Stimuli ✻ Location	456.504	1	456.504	1.537	0.230	6.252×10^{-4}
Residuals	5642.246	19	296.960			

Table 7. *The results of Repeated Measures ANOVA evaluating the effect Listening conditions (Q, W, B), Stimuli (1kHz and /da/) and Electrode (T7 and T8) on latency of Tb.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening Condition ✻ Stimuli ✻ Locations	386.808[a]	2[a]	193.404[a]	1.082[a]	0.349[a]	5.298×10^{-4}
Residuals	6792.192	38	178.742			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated ($p < .05$).

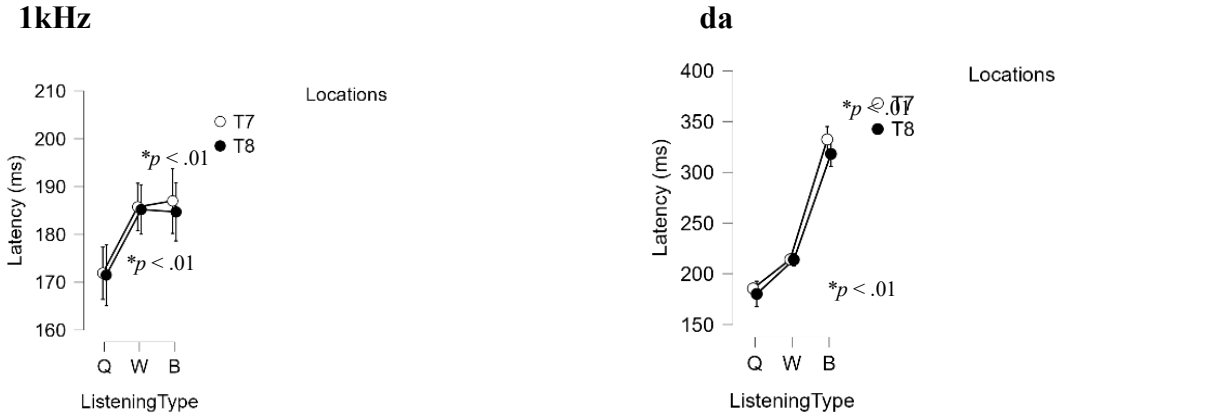

Figure 7 *The average of Tb latency to 1kHz and da, in quiet (Q), non-speech noise (W) and babble noise (B), at T7 and T8. The most significant conditions have been marked with star symbol : *.*

2.4.3. Frontocentral responses, P1 peak

Repeated-measures ANOVA (Table 16) revealed significant main effects of listening condition, $F(2, 38) = 83.045$, $p < .001$, $\eta^2 = .327$, and stimuli, $F(1, 19) = 281.426$, $p < .001$, $\eta^2 = .301$, as well as a significant interaction between stimuli and listening condition, $F(2, 38) = 27.299$, $p < .001$, $\eta^2 = .163$. Follow-up *t*-tests with Bonferroni correction showed significantly longer latency in non-speech noise versus quiet, babble noise versus quiet, and babble noise versus non-speech noise for /da/ ($p < .001$). For the 1-kHz tone, P1 latency was marginally longer in non-speech noise than in quiet ($p = .070$; Figure 8).

Table 8. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on latency of P1.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	11908.717	2	5954.358	83.045	< .001	0.327
Residuals	2724.617	38	71.700			
Stimuli	10982.533	1	10982.533	281.426	< .001	0.301

Table 8. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on latency of P1.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Residuals	741.467	19	39.025			
Listening condition ✻ Stimuli	5941.017	2	2970.508	27.299	< .001	0.163
Residuals	4134.983	38	108.815			

Note. Type III Sum of Squares

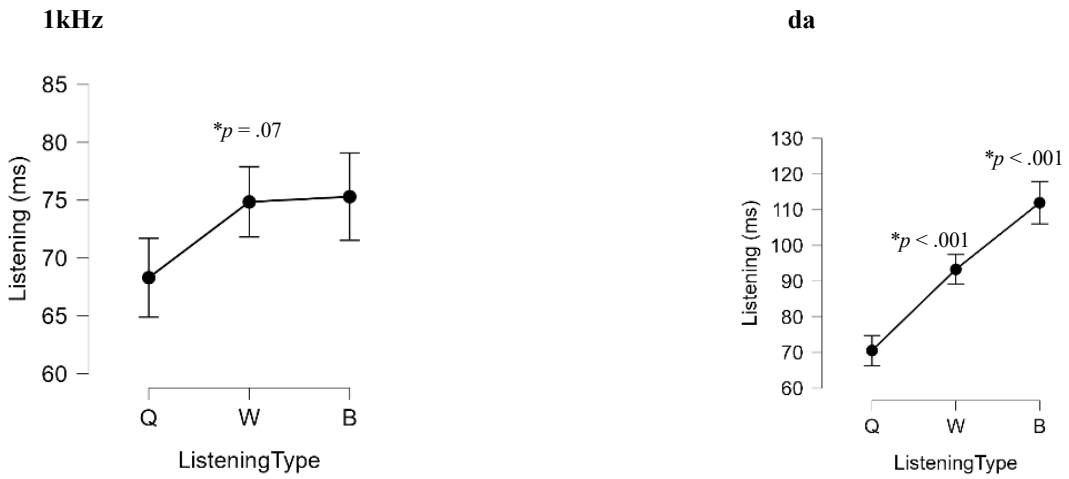

Figure 8 *The average of P1 latency to 1kHz and da, in quiet (Q), non-speech noise (W) and babble noise (B). The most significant conditions have been marked with star symbol : *.*

2.4.4. Frontocentral responses, N1 peak

Repeated-measures ANOVA (Table 17) revealed significant main effects of listening condition, $F(2, 38) = 199.449$, $p < .001$, $\eta^2 = .390$, and stimuli , $F(1, 19) = 286.171$, $p < .001$, $\eta^2 = .287$, as well as a significant interaction between stimuli and listening condition, $F(2, 38) = 142.724$, $p < .001$, $\eta^2 = .234$. Follow-up *t*-tests with Bonferroni correction confirmed the effect of listening condition, showing longer latency in non-speech noise versus quiet, babble noise versus quiet, and babble noise versus non-speech noise for both stimuli ($p < .01$; Figure 9).

Table 9. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on latency of N1.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	61255.017[a]	2[a]	30627.508[a]	199.449[a]	< .001[a]	0.390
Residuals	5835.317	38	153.561			
Stimuli	44969.408	1	44969.408	268.171	< .001	0.287

Table 9. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on latency of N1.*

Cases	Sum of Squares	df	Mean Square	F	p	η²
Residuals	3186.092	19	167.689			
Listening condition ⁎ Stimuli	36791.217[a]	2[a]	18395.608[a]	142.724[a]	< .001[a]	0.234
Residuals	4897.783	38	128.889			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

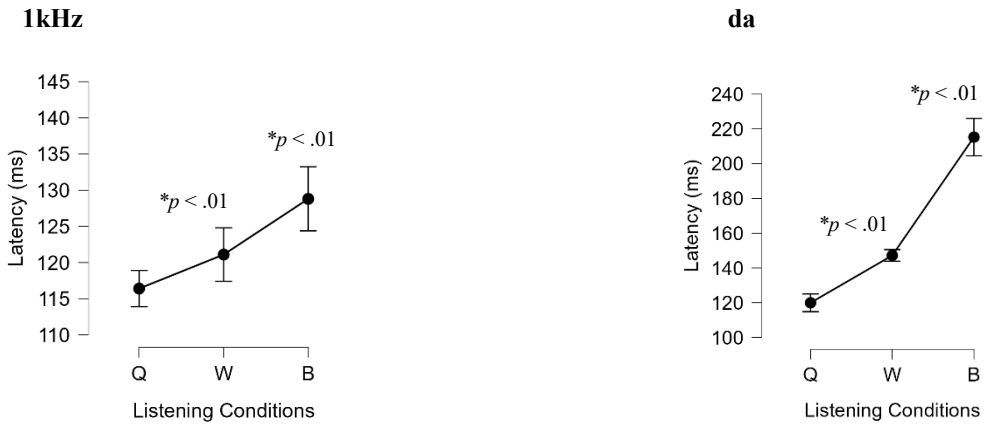

Figure 9. *The average of N1 latency to 1kHz and da, in quiet (Q), non-speech noise (W) and babble noise (B). The most significant conditions have been marked with star symbol : *.*

2.4.5. Frontocentral responses, P2 peak

Repeated-measures ANOVA (Table 18) revealed significant main effects of listening condition, $F(2, 38) = 177.444$, $p < .001$, $\eta^2 = .358$, and stimulus type, $F(1, 19) = 157.026$, $p < .001$, $\eta^2 = .277$, as well as a significant interaction between stimulus type and listening condition, $F(2, 38) = 153.546$, $p < .001$, $\eta^2 = .261$. Follow-up *t*-tests with Bonferroni correction confirmed the effect of listening condition. For /da/, P2 latency was significantly longer in non-speech noise versus quiet, babble noise versus quiet, and babble noise versus non-speech noise ($p < .001$; Figure 12). For the 1-kHz tone, latency was significantly longer in non-speech noise versus quiet and in babble noise versus quiet ($p < .001$), with no significant difference between non-speech noise and babble noise ($p = 1.000$; Figure 10).

Table 108. *The results of Repeated Measures ANOVA evaluating the effect of Listening conditions (Q, W, B), Stimuli (1kHz and /da/) on latency of P2.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	122802.450[a]	2[a]	61401.225[a]	177.444[a]	< .001[a]	0.358
Residuals	13149.217	38	346.032			
Stimuli	95034.408	1	95034.408	157.026	< .001	0.277
Residuals	11499.092	19	605.215			
Listening condition ✻ Stimuli	89694.117[a]	2[a]	44847.058[a]	153.546[a]	< .001[a]	0.261
Residuals	11098.883	38	292.076			

Note. Type III Sum of Squares

[a] Mauchly's test of sphericity indicates that the assumption of sphericity is violated (p < .05).

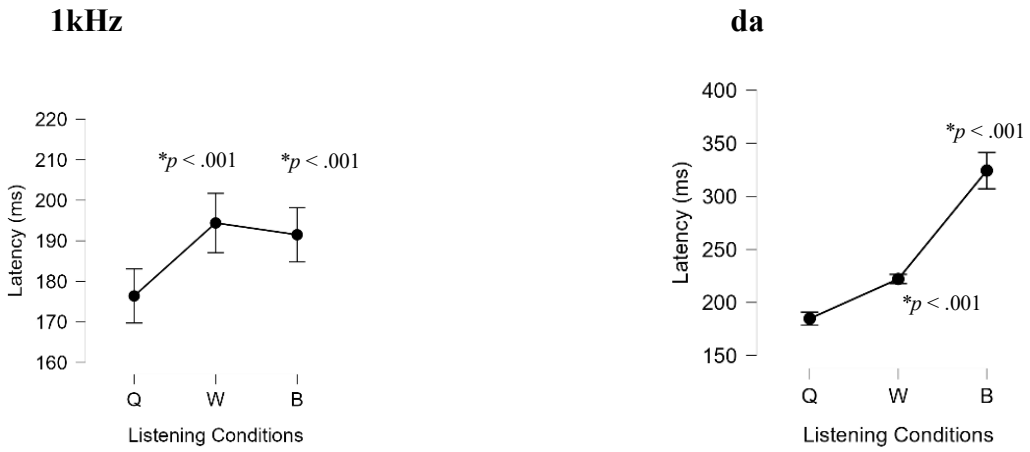

Figure 3. *The average of P2 latency to 1kHz and da, quiet (Q), non-speech noise (W) and babble noise (B). . The most significant conditions have been marked with star symbol : *.*

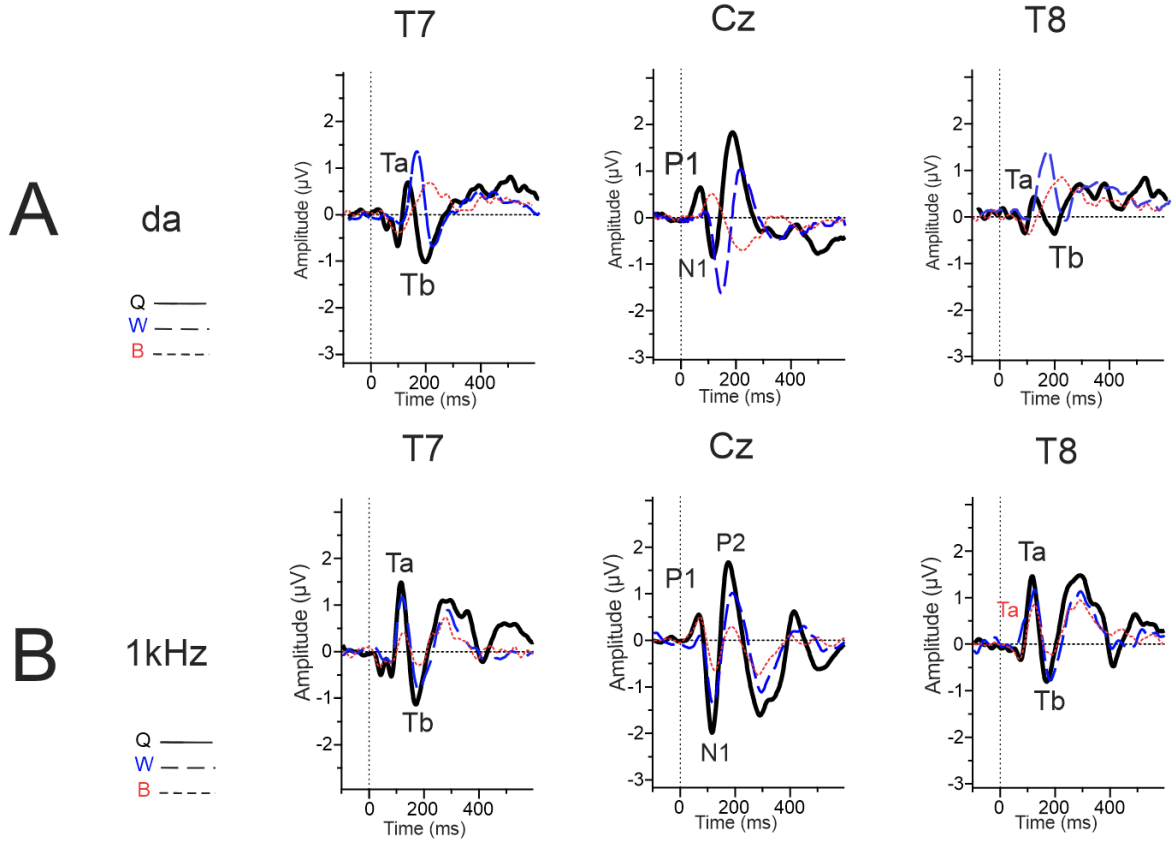

Figure 4. *Grand average of Auditory Cortical Responses to* ***da*** *(A) and* ***1kHz*** *(B), recorded at electrode Cz, T7 and T8, in quiet (black curve), non-speech noise (blue curve) and babble noise (red curve).* ***A****, with /da/, mostly the latency of all CAEPs was prolonged in noise. The amplitude of P1 and P2 were reduced. However, the amplitude of N1, Ta and Tb were reduced and increased in noise. Also, the Tb's amplitude was larger at T8, than T7.* ***B****, with 1kHz, the latency was prolonged, and the amplitude was reduced for most of the AEPs in noise. Also, the Ta's amplitude was larger at electrode T8, than responses at electrode T7, in noisy conditions.*

Table 19. *Summary of findings.*

Component	Amplitude modulation in noise	Latency modulation in noise
P1	There was a reduction pattern in amplitude of P1 in noises, significantly only with speech stimulus in non-speech noise.	Longer latency in noises, it was significant only with speech stimuli.
N1	There were a reduction and an enhancement pattern in noises. The amplitude of N1 was decreased using 1kHz, but it was increased using speech, significantly in non-speech noise.	Longer latency in noises.
P2	There was a reduction pattern in amplitude of P2, significantly in babble noise using both stimuli. In non-speech noise, the P2 amplitude decreased significantly with speech.	Longer latency in noises, significantly with speech stimulus.

	At T7 and with 1kHz, there was a significant reduction pattern in noises, with /da/ there was a significant enhancement pattern in non-speech noise and no significant change in babble noise.	
Ta	At T8, there was no significant change on Ta with 1kHz, and significantly larger amplitude in noises with /da/. Also, with only 1kHz, there was significantly larger amplitude of Ta at electrode T8 than Ta recorded at electrode T7, in noises. No effect of location on Ta in quiet.	Longer latency in noises, significantly with speech stimulus.
Tb	In non-speech noise, there was reduction pattern in amplitude of Tb, non significantly using 1kHz and significant using speech. In babble noise, the amplitude of Tb increased with speech stimuli only at T8 and decreased with 1kHz at both electrodes T7 and T8.	Longer latency in noises, significantly with speech stimulus.

2.5. Discussion

The aim of this study was to evaluate the effect of noise on cortical auditory evoked potentials, particularly by comparing noise-induced changes between temporal and frontocentral components using various types of noise and stimuli. Given the different neural generators and developmental trajectories of these responses, we hypothesized that temporal and central responses would show distinct patterns. Consistent with our expectation, the effect of noise on temporal responses differed from that on frontocentral responses (Figure 11). Our second hypothesis predicted that noise-induced enhancement of N1 would be observed only with speech stimuli, not with non-speech stimuli, as speculated (Figurer 11A). The current study also showed larger Ta and Tb amplitudes in noise for speech stimuli. Supporting our third hypothesis, the results revealed different patterns of T-complex modulation between electrodes T7 and T8 under certain conditions (Figure 11A). Specifically, with non-speech stimuli, Ta amplitude was larger in noise at T8 than at T7 (Figure 11B), whereas with speech stimuli, Tb amplitude increased in noise only at T8 (Figure 11A).

2.5.1. Pattern of changes in the temporal and frontocentral responses

2.5.1.1. Changes of amplitude

The results showed that noise-induced modulations in CAEP amplitude included both suppression and enhancement patterns, depending on the type of noise and the stimulus. The amplitudes of Ta and Tb were affected by background noise. Ta was reduced when non-speech stimuli were presented in noise compared to quiet. In contrast, Ta was enhanced in noise when the target stimulus was /da/, reaching significance in non-speech noise but not in babble noise. Tb was reduced in non-speech noise (not significant) but was significantly enhanced in babble noise when the target stimulus was /da/. The different Tb amplitude patterns observed in babble versus white noise may reflect differences in auditory scene analysis (ASA) demands (Bregman, 1990). ASA refers to object formation, segregation of speech streams and extracting meaning during a noisy environment (Bregman, 1990; Cunningham et al., 2002; Schnupp et al., 2010; Shinn-Cunningham & Best, 2008). Informational masking, such as babble noise, reflects the competing speech sounds that not only physically interfere with the target speech but also impair the separating of the sound of the speaker from the background speech (Douglas S Brungart et al., 2001). It is assumed that informational masking is more detrimental to speech understanding than energetic masking like white noise (Douglas S Brungart et al., 2001), due to failures of subject's attention leading to lower ability to attend to and segregate out the target speech in the presence of other similar speech sounds (D. S. Brungart et al.,

2001; B. G. Shinn-Cunningham, 2008). So, it may be plausible to speculate that babble noise requires greater cortical processing to separate the target speech signal from the background that might be observed as a larger amplitude of Tb in babble noise than white noise in current study. To our knowledge, this is the first study to report the effects of noise on Ta and Tb components, with no previous results available for direct comparison. More research is needed to confirm these findings.

Among the central responses, the amplitudes of P1 and P2 showed a reduction pattern in noise. Suppression of frontocentral auditory evoked potential amplitudes has been reported in several previous studies (Androulidakis & Jones, 2006; C. J. Billings et al., 2017; Curtis J. Billings et al., 2009; McCullagh et al., 2012; Katherine A. Whiting et al., 1998a).It has been suggested that the addition of background noise suppresses neural discharge and reduces the synchronized firing of neural populations, leading to a decrease in the magnitude of auditory responses (Cunningham et al., 2002; Kaplan-Neeman et al., 2006; McCullagh et al., 2012). In the current study, changes in P1 were dependent on the type of noise. P1 amplitude was reduced in non-speech noise but not in babble noise. Reports on P1 amplitude in the literature are inconsistent, with some studies showing decreases in P1 under noise conditions (Bertoli et al., 2005; Papesh et al., 2015), although other studies have reported no change in P1 amplitude under noise conditions (Curtis J. Billings et al., 2009; Gott & Hughes, 1989). It has been suggested that variability in P1 amplitude modulation in noise may stem from individual differences as well as methodological differences in the acquisition and analysis of CAEP data across studies (Papesh et al., 2015). However, none of the previous studies have reported such effect of noise type on P1 amplitude. In the current study, a reduction was observed only in the non-speech noise condition, suggesting that noise type influences the strength of the P1 response. This may indicate that the neural generators of P1 are more resistant to, and better adapted to, speech noise than to non-speech noise, thereby maintaining the representation of auditory stimuli. Also, the non-significant changes in the results might be observed because of the small sample size (n=20) of the current study. So, further research using the same paradigm with larger sample size is needed to confirm this interpretation.

P2 amplitude was reduced in the presence of both babble and non-speech noise, for both speech and non-speech stimuli. This suppression of P2 amplitude is consistent with previous findings in the literature (Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006; McCullagh et al.,

2012; Papesh et al., 2015; Katherine A. Whiting et al., 1998a). The majority of studies have demonstrated that P2 amplitude is consistently reduced, regardless of methodological differences, stimulus type, or noise type (Curtis J. Billings et al., 2009; Kaplan-Neeman et al., 2006; Katherine A. Whiting et al., 1998a).

N1 was reduced for non-speech stimuli in noise but was enhanced for speech stimuli in the presence of non-speech noise. Consistent with expectations, the current findings replicate those of Duquette-Laplante et al. (2024), who reported decreased N1 amplitude for non-speech and increased amplitude for speech stimuli in noise. Additional parameters, such as binaural versus monaural presentation, fast versus slow stimulation rates (90ms vs. 2000ms), and the inclusion of low-frequency components (e.g., 1–30 Hz vs. 3–30 Hz high-pass filtering), may also contribute to the enhancement of N1 amplitude (Alain et al., 2009; Baltzell & Billings, 2014; Curtis J. Billings et al., 2009; Papesh et al., 2015), The current study demonstrated that stimulus type interacts with background noise to influence cortical responses. Specifically, background noise reduced N1 amplitude when the stimulus was a tone, whereas non-speech noise enhanced the N1 response when the stimulus was /da/. Moreover, N1 amplitude was not affected by babble noise when the stimulus was a 1-kHz tone, possibly because 1 kHz is a salient frequency within babble noise. This finding does not fully support previous explanations for the enhanced N1 observed with speech stimuli in non-speech noise but instead suggests that stimulus type; speech versus non-speech, plays a critical role in modulating changes in amplitude.

In addition to Duquette-Laplante et al. (2024), who reported enhanced N1 amplitude in noise with speech stimuli, two out of three other studies have also demonstrated this enhancement (Alain et al., 2009; Papesh et al., 2015; Parbery-Clark et al., 2011), used speech stimuli (Papesh et al., 2015; Parbery-Clark et al., 2011). Considering the consistency between the current study and previous research, speech stimuli appear to be an additional factor contributing to increased N1 amplitude in the presence of background noise. A possible explanation may lie in reports suggesting that modulation of the efferent auditory system in noise differs depending on stimulus type (Namasivayam et al., 2013; Namasivayam et al., 2015). It has been demonstrated that speech stimuli may exhibit greater release from suppression than tones (Namasivayam et al., 2015). As such, speech stimuli may trigger the efferent system in noise differently and/or more efficiently than non-speech stimuli (Bidelman & Howell, 2016). However, more research is needed to confirm this hypothesis.

2.5.1.2. Changes of latency

The results showed longer latencies for both frontocentral and temporal responses in noise compared to quiet. This finding replicates previous reports demonstrating delayed auditory evoked potentials in the presence of background noise (Billings et al., 2011; Curtis J. Billings et al., 2009; Benjamin Rich Zendel et al., 2015). Noise degrades the neural synchronization, leading to slower transmission and longer latency of auditory response (Anderson, Skoe, et al., 2010; Curtis J. Billings et al., 2009).

2.5.2. The comparison between temporal and frontocentral responses

This study, as one the first addressing the effect of noise on the T-complex components, has shown remarkable and novel finding of noise-induced changes on temporal responses, which were distinct from frontocentral responses. Most frontocentral responses were suppressed in noise, with N1 being the only component to show enhancement, which reached significance only in non-speech noise. However, both temporal components showed larger responses under certain noisy conditions, with Tb in particular exhibiting significant amplitude increases, most prominently in the presence of babble noise.

Babble noise is probably the closest noise to real-life listening environments. This result may support the idea of the T-complex indexing greater adaptation to real-life listening situations than the frontocentral responses. It may highlight the robust function of the T-complex in processing auditory information at the secondary auditory areas in noisy listening conditions compared to the frontal responses at the primary auditory cortex. As this is the first study reporting this result, more research is needed to confirm the distinctive processing of auditory information in noise occurring in primary and secondary auditory areas.

2.5.3. Effect of Location (T7 vs. T8)

One of the interesting and novel findings of this study was the difference in noise-induced modulations between the right and left hemispheres. Both Ta and Tb showed significantly larger responses at T8 compared to T7 in at least one noisy listening condition, indicating greater activity in the right hemisphere than in the left under noise. No such hemispheric difference was observed in quiet, suggesting that this auditory lateralization is induced by noise. Rightward lateralization in noise has also been reported in the literature and is thought to reflect greater right-hemisphere involvement in processing auditory signals under degraded

listening conditions (Shtyrov et al., 1998; Shtyrov et al., 1999; Wong et al., 2009). Greater activity in the right hemisphere is likely the result of additional compensatory processing that supports the understanding of impoverished speech (Bidelman & Dexter, 2015; Du et al., 2014). An additional explanatory framework for the observed lateralization involves hemispheric specialization in auditory speech processing (Poeppel, 2003; Zatorre & Belin, 2001). Classically, the specto-temporal asymetry model posits that the left hemisphere is specialized for rapid temporal features characteristic of speech, whereas the right hemisphere preferentially processes spectral information, maybe tonal processing and music perception (Belin et al., 1998; Liégeois-Chauvel et al., 1999; Zatorre & Belin, 2001). However, this classical dichotomy has been refined by the *Asymmetric Sampling in Time* (AST) hypothesis, which proposes that speech processing is bilaterally organized, with hemispheric differences emerging from distinct temporal integration windows rather than strict domain specialization (Poeppel, 2003). According to the AST framework, the left hemisphere is preferentially tuned to fast temporal modulations, whereas the right hemisphere is more sensitive to slower temporal fluctuations that extend over longer integration windows (Abrams et al., 2008; Arnal et al., 2015; Giraud & Poeppel, 2012; Poeppel, 2003). Within this conceptualization, the noise-induced rightward lateralization observed in the present study is theoretically meaningful rather than incidental. Degraded listening conditions alter the acoustic structure of speech by reducing access to rapidly changing cues and increasing reliance on slower, more robust temporal information (Russo et al., 2004). Indeed, cortical neurons exhibit adaptive synchronization in noise, shifting their tracking toward lower temporal modulation rates (Ding & Simon, 2013; Kachlicka et al., 2022). From this perspective, the enhanced right-hemisphere response in noise may reflect an adaptive reweighting of cortical processing strategies, whereby the auditory system preferentially recruits neural mechanisms optimized for slower temporal integration. Previous CAEP studies have demonstrated rightward lateralization in auditory responses such as the mismatch negativity (MMN) (Shtyrov et al., 1998), N1(Bidelman & Howell, 2016), or N400 (Benjamin Rich Zendel et al., 2015), This is the first study to report rightward lateralization in noise as indexed by the T-complex. Given that these responses are primarily generated in secondary auditory areas, this finding extends previous studies that demonstrated rightward lateralization in noise within the primary auditory cortex (Bidelman & Dexter, 2015; Bidelman & Howell, 2016).

Moreover, the current study showed that rightward lateralization of Ta was observed only with tones in noisy conditions, whereas rightward lateralization of Tb emerged with speech stimuli

in noise. This may indicate that stimulus type influences auditory processing in noise and that speech and non-speech stimuli are processed differently within secondary auditory areas. Further research is needed to confirm this finding

The novel results of this study showed the distinct noise-induced changes between temporal and frontocentral responses. This discrepancy may suggest the distinct roles of different auditory cortex structures in listening ability in noise. The stimuli and noise type interaction can affect the amplitude modulation of AEPs. Importantly, the Tb saw increased amplitude when noise was babble and stimuli was speech. It can show special adaptation of auditory processing for perception of speech in real-life noisy conditions. Furthermore, T-complex generators supported the rightward lateralization of cortical auditory processing in noise. This outcome can be utilized for clinical application purposes to evaluate the effect of noise at secondary auditory areas in clinical populations with communication disorders, such as people with auditory processing disorder.

2.6. Future Directions

Future studies in normal-hearing children can bring more information regarding the effect of noise on T-complex. Also, according to the distinct results between temporal and frontocentral responses, it may be promising to examine the pattern of changes of these two responses in noise, in population who have listening difficulties in noise, like individuals with auditory processing disorder. Regarding the methodology, more research using various SNR with speech and non-speech stimuli can demonstrate if the increasing change of amplitude is observed in more favorable or effortful listening conditions.

2.7. CRediT authorship contribution statement

Zohreh Ahmadi: Writing – review & editing, Writing – original draft, Visualization, Methodology, Project administration, Investigation, Formal analysis, Conceptualization. **Shanna Kousaie:** Writing – review & editing, Visualization. **Benjamin Rich Zendel:** Writing – review & editing, Methodology, Funding acquisition, Formal analysis. **Amineh Koravand:** Writing – review & editing, Supervision, Methodology, Resources, Project administration.

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2.9. Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

2.10. Data availability

Data will be made available on request.

2.11. Declaration of generative AI and AI-assisted technologies in the writing process

No AI tools or services were used during the preparation of this work.

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Chapter 2. Manuscript 2

The effect of noise on frontocentral and temporal auditory cortical responses in typically developing children using speech and non-speech stimuli

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Abstract

Cortical auditory evoked potentials (AEPs) consist of frontocentral components (P1–N1–P2) and temporal components known as the T-complex (Ta, Tb), which originate from distinct cortical generators. The T-complex seems to emerge with adult-like appearance early and remains relatively stable across age, suggesting it may show different contribution to auditory cortical processing in ecologically valid listening situations. Despite the importance of background noise in real-world communication, its effects on the T-complex, particularly in children have not been previously examined. Background noise significantly reduced amplitude of Ta, similar to frontocentral components. Latencies were generally longer in noise, particularly for speech and in babble noise. Taken together, results may add insights into what variables are driving cortical signal-in-noise encoding and how auditory cortical components would react to them distinctively. Also, the second component of Tb and the N1 frontocentral response did not show significant changes in amplitude in the presence of noise, compared to quiet. The non-significant results may show the immature efferent and presumably afferent auditory pathway in generators of N1 in children leading to non-suppression of the response and non-meaningful changes in the amplitude of Tb.

Keywords

Cortical auditory responses, T-complex, enhancement response in noise, babble noise.

3.1. Introduction

Classroom instruction takes place in noisy, reverberant environments (Eranovic et al., 2022; Neuman et al., 2010; O'Hara & Mealings, 2018; Prodi & Visentin, 2022), accordingly, the ability to listen and understand verbal information in adverse listening conditions are critical for communication and learning processes (Hunsaker, 1983). Listening skills are even more crucial for young students as over 60% of elementary classroom time is spent "learning by listening" (Hunsaker, 1983). Diminished understanding speech in noise is a hallmark consequence of Auditory Processing Disorder (APD) in children (Bamiou et al., 2001; Chermak, 2002; Musiek & Geurkink, 1980; Vanniasegaram et al., 2004). Misdiagnosis and late detection of speech in noise difficulties can result in negative effects on language, learning and educational achievements (Canale et al., 2014; Cestnick & Jerger, 2000; Del Zoppo et al., 2015).

Evaluating the effect of noise on auditory skills can lead to better understanding of auditory impairments in populations with APD. Multiple behavioral studies have shown the detrimental effect of noise on speech perception (Dirks et al., 1982; Le Prell & Clavier, 2017; Lee et al., 2015; Meyer et al., 2013; Pittman & Wiley, 2001), although they face some important limitations. For example, behavioral measures can be affected by non-auditory factors like attention and language (Jerger & Musiek, 2000), and behavior does not evaluate the underlying neurophysiologic mechanisms governing speech processing in noise.

Auditory evoked potentials (AEPs) can be measured using electroencephalography (EEG) and used to effectively evaluate sound processing ability along the auditory system (Anderson, Skoe, et al., 2010). In young children, typically before the age of 9, auditory late responses (ALRs) are primarily characterized by two waveforms/components, P1 and N2, which emerge early in development (Ponton et al., 2000). As children mature, the N1–P2 complex becomes more prominent, accompanied by a general reduction in the latencies and amplitudes of the initial P1 and N2 waves (Čeponienė et al., 2005; Pang & Taylor, 2000; Ponton et al., 2000). Literature consistently shows that noise degrades the auditory subcortical responses resulting in delayed timing and reduced amplitude of the response in normal-hearing children (J. Cunningham et al., 2001; de Boer et al., 2020; Song et al., 2012; Strait et al., 2012). P1, N1, P2 and N2 are the frontocentral category of AEPs and have been extensively studied to evaluate the effect of noise on auditory processing at the cortical levels. These components are long latency AEPs, which represent synchronous neural activation of structures mainly in the primary auditory cortex and associated areas of the temporal lobe (Eggermont & Ponton,

2002; Näätänen & Picton, 1987; Picton et al., 1999). The P1, N1 and P2 encode acoustical features such as frequency and timing (Čeponienė et al., 2005; Liégeois-Chauvel et al., 1994; Näätänen & Picton, 1987). The N2 is associated with sensory auditory processing, and can also be modulated by attention, perception, discrimination, and recognition of sounds (Näätänen et al., 1982).

The latency of frontocentral responses are prolonged in noise and latency seems to be more sensitive to noise than amplitude (Anderson, Chandrasekaran, et al., 2010; J. Cunningham et al., 2001; Duquette-Laplante et al., 2024; Gustafson et al., 2019; Morlet et al., 2019; Ubiali et al., 2025). The pattern of changes in amplitude of frontocentral responses seems to depend on the type of stimuli (Billings et al., 2011; Billings & Grush, 2016). In normal-hearing children, Ubiali et al. (2025) recorded AEPs using speech (/da/ and /ba/) and non speech (tone burst 2 and 1kHz) stimuli in the presence of white noise. Results showed no effect of noise on the amplitude of N1 when stimuli were non speech. Contrarily, in response to speech stimuli, the amplitude of N1 was reduced in noise. It has been shown that AEPs elicited by non periodic stimuli like speech are more degraded by noise than the AEPs elicited by simple periodic stimuli like pure tones (Billings et al., 2011).

Importantly, Ubiali et al., (2025) used only non-speech noise (white noise), and it is not clear if the noise-induced changes in the amplitude of AEPs is dependent on the interaction between stimuli and noise type. Duquette-Laplante et al. (2024) evaluated the effect of noise on normal-hearing children using speech (/da/) and non-speech stimuli (pure tone 1kHz) in the presence of white and babble noise (8-talker). AEPs were identifiable only in white noise and the N1 was enhanced in noise with speech stimuli. This pattern was not observed with non speech stimuli. Duquette-Laplante et al. argued that the enhancement in the N1 was observed because speech stimuli were detected as a salient event when the background is white noise, leading to an increased amplitude of N1. However, because most of the AEPs waves were not identifiable in 8-talker babble noise, a similar comparison between non-speech stimuli and speech noise was not possible.

Babble noise with fewer talkers, for example 4-talkers may be more appropriate in AEP studies as multiple studies have reported identifiable AEP waves with 4-talker babble noise (Benítez-Barrera et al., 2021; Billings et al., 2011; Zendel et al., 2015). Supportively, the study of Gustafson et al. (2019) showed a larger N1 in response to speech stimuli in the presence of 4-talker babble noise than quiet in normal-hearing children. The authors suggested that the

immature auditory system led to a larger N1 in noise. This interpretation is not consistent with that of Duquette-Laplante et al. (2024) for the observed increased amplitude of N1 in noise versus quiet, who suggested that the larger N1 was due to the salience of the speech stimuli. Considering inconsistent results, more research using speech and non-speech stimuli in the presence of white noise and 4-talker babble-noise will confirm the results.

While there is some research on the impact of background noise on tangentially oriented auditory evoked responses (i.e., P1-N1-P2-N2), little is known about the impact of background noise on radially oriented sources that produce an auditory evoked response at temporal sites. Temporal responses or the T-complex is another auditory evoked potential (Wolpaw & Penry, 1975). It includes the Na-Ta-Tb sequence corresponding to processes mainly in secondary auditory areas, specifically the lateral superior temporal gyrus, where dipole sources are radially oriented (Lindenberger & Baltes, 1994; Uhlén et al., 1996; Wolpaw & Penry, 1975). In children, studies observed the T-complex as negative-positive-negative deflections between 50-250ms (Pang & Taylor, 2000; Tonnquist-Uhlen et al., 2003). T-complex and frontocentral responses arise from different generators and mature at distinct ages (Albrecht et al., 2000; Billings et al., 2011; Pang & Taylor, 2000). Developmental studies have shown that the T-complex seems to be adult-like earlier than the frontocentral responses and remains relatively stable with age (Ponton et al., 2002; Tonnquist-Uhlen et al., 2003).

Similar to frontocentral responses, the T-complex can be modulated by changes in stimulus intensity, interstimulus interval (ISI) and Stimulus Onset Asynchrony (SOA) (Clunies-Ross et al., 2018; Jarmo A. Hämäläinen et al., 2011), and both speech and non-speech stimuli can elicit the T-complex (D. V. Bishop et al., 2012; Margriet Anna Groen et al., 2008; Hine & Debener, 2007). Additionally, some studies have reported different modulation patterns of the T-complex recorded at the right and the left temporal sites (T7 versus T8) in response to stimuli parameters, showing that the T-complex represents different processes in the right and left secondary auditory areas (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). According to the literature, Tb, the second component of T-complex shows larger responses at right hemisphere to stimuli with slow temporal changes such as paired-tone with 200ms intervals, while in response to stimuli with faster temporal changes like paired-tone with 50ms intervals Tb seems to be larger at the left hemisphere (Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). Such findings are consistent with Asymmetric Sampling in Time theory, which argues that the right hemisphere is specialized for processing slow temporal changes and left hemisphere is sensitive to fast temporal changes.

Regarding the effect of background noise, to the best of our knowledge there is still no study evaluating noise-induced changes in the T-complex. Accordingly, the effect of noise on auditory processes in secondary auditory areas remains unclear. Given that the T-complex matures earlier and is more stable than AEPs at frontocentral sites, and that the T-complex is susceptible to acoustic manipulations, it is possible that the T-complex may be related to separating speech from background noise. Understanding the functioning of the central neural auditory system at primary and secondary auditory areas, in the presence of noise, may help in the development of future assessment tools for early detection and individualized intervention planning for children with listening difficulties like APD.

Therefore, the aim of this study is to evaluate the effect of noise on the T-complex and to compare the pattern of effects with those observed in the frontocentral responses in children with normal hearing. Additionally, this study will determine if there is a differential impact of speech or non-speech stimuli in the presence of white noise or 4-talker babble noise. Considering different generators between frontocentral and T-complex, in general, it is hypothesized that there will be distinct noise-induced changes between these two responses.

3.2. Method

3.2.1. Ethics approval

The University of Ottawa Research Ethics Board approved this study, Ethic file number=H-07-24-10451. All participants, and their parent/guardian provided written informed consent.

3.2.2. Participants

Fifteen students between 8-12 years old (Mean=10.438, SD=1.315, Male:7) participated in this study. The sample size was comparable to previous studies which could identify the significant effect of noise on cortical auditory evoked potentials (Anderson, Chandrasekaran, et al., 2010; Han et al., 2020; Hassaan, 2015; Hussain et al., 2022).

3.2.3. Inclusion and exclusion criteria

All participants had normal hearing detection thresholds at 15 dB HL or less in octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996)'s protocol. Tympanograms were normal (admittance curve with a single peak between +50 to -100daPa using a 226-Hz probe tone). Participants were right-handed, fluent in speaking and listening skills in English language according to the questionnaire of Languages known in order of dominance (LEAP-Q), as the babble noise used in this study was in English (Marian et al., 2007).

No history of otological or neurological disorders were reported for any of children participating in the study. The Auditory processing domain questionnaire (APDQ) was also used to evaluate auditory processing, attention and language skills. APDQ is a screening tool, developed in 2018 and it has demonstrated 89% sensitivity and 82% specificity in distinguishing children with auditory processing disorder (APD) from typically developing peers (O'Hara & Mealings, 2018). It has also shown significant ability to separate clinical groups including language disorder and attention disorder/hyper activity (ADHD) and APD from each other, with an average inter-group sensitivity and specificity of 89% (O'Hara & Mealings, 2018). The BKB-SIN (Bamford-Kowal-Bench Speech-in-Noise) was also conducted to make sure participants do not have listening difficulties in noise. All included participants had normal performance in BKB (signal-to-noise ratio loss was 0-3dB) and the score of the APDQ were in normal range (>70%). Also, all had normal cognitive abilities. To assess cognitive abilities, the auditory Continuous Performance Test (ACPT) was conducted. ACPTs are cognitive tasks designed to measure a person's sustained attention and vigilance, particularly in an auditory context (Keith, 1994; Riccio et al., 1996). The results of ACPT test were in normal limit; the total of omission and commission errors were less than 10. Finally, this study excluded participants with musical training as previous studies show that musical training can enhance the understanding of speech in noise and result in improved AEPs in the presence of background noise (Anderson et al., 2013; B. R. Zendel et al., 2015). Results of the behavioral tests have been inserted in table 1.

Table 11. *The results of APDQ, BKB and ACPT tests.*

	Age	AP	LA	ATT	BKB	ACPT-Commission errors 2	ACPT-Omission errors
Valid	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0
Mean	10.881	86.813	90.438	83.000	1.919	3.063	1.688
Std. Deviation	1.270	11.600	10.844	10.545	0.732	1.436	0.704
Minimum	8.700	57.000	71.000	53.000	0.800	1.000	1.000
Maximum	12.900	99.000	100.000	97.000	3.000	5.000	3.000

AP=Auditory processing, ATT=Attention, ACPT=Auditory Continuous Performance Test, BKB=Bamford-Kowal-Bench Speech-in-Noise, LA=Language.

3.2.4. Procedure

During the experiment, after familiarization with the testing equipment and procedure, the participants were seated in a sound-attenuated testing room, where they completed the

experiment. The participants were instructed to ignore the auditory stimuli and to focus their attention on the movie, while the EEG was recorded. Prior to testing, the purpose of the study was explained to each participant and informed consent was obtained from the children and their parents.

3.2.5. Stimuli

Participants completed two experiments as part of this study; here we report the results of studies that explored the effect of noise on AEPs using speech and non-speech stimuli. The second study explored the manipulation of voice onset time (VOT) of a speech stimulus, and the VOT results will be reported elsewhere. The data from one of the speech tokens from this second study is reported here as the speech stimuli. The two experiments were conducted in one day, starting with the first experiment including non-speech stimuli followed by the second experiment including speech stimuli. The non-speech stimulus was a pure tone 1kHz that was 250ms in duration and had a 10ms rise and fall time, created in Audacity software (Audacity Team, 1999). The speech stimulus was a natural /da/, it was originally recorded by a male voice and used in the study of Dubno & Schaefer in 1992. The speech stimuli had the same duration (250ms) and rise-fall time (10ms) as the non-speech stimuli. Stimuli were presented in three listening conditions including quiet, in the presence of babble noise and in the presence of white noise. In experiment 1, there were three blocks of 1kHz tones in the three listening conditions including quiet, white noise, and babble noise. In experiment 2, considering the goal of the study, each block included /da/ and /ta/, and three blocks in total were delivered in the three listening conditions. The order of the listening conditions was randomized between subjects. There were 300 presentations of each stimulus in each listening conditions. White noise (with equal energy distributed to all frequencies from 250 to 6000 Hz) was created and extracted from Audacity software (Audacity Team, 1999) and babble noise (English-four-talker) was developed using the same method as the French babble noise generated by Zendel et al, 2015, but in English (Zendel et al., 2015).

Since hearing in noise is typically a binaural process, it is important to implement test measures that increase the ecological validity, or the extent to which the results can generalize to real-life listening situations. To this end, the stimuli were delivered in a passive non-oddball, homogenous paradigm, because oddball paradigms introduce cognitive variables such as attention and stimulus context effects, which complicate the interpretation of earlier CAEP components (Billings et al., 2011). Stimuli were equalized for root mean square (rms) amplitude and then played at 80 dB SPL; this level of intensity is consistent with previous

studies which showed significant effects (Anderson, Chandrasekaran, et al., 2010; Jenna Cunningham et al., 2001; Hassaan, 2015; Hussain et al., 2022). For noisy conditions, signal-to-noise ratio was kept as +5dB, by reducing the intensity of noise to 75dB SPL. Interstimulus interval (ISI) as calculated from the offset of the preceding stimulus to the onset of the next stimulus, was jittered randomly between 1100-1250ms, as it has been shown that a slower stimulation rate results in more robust AEP waveforms in the immature auditory nervous systems (Phillip M. Gilley et al., 2005). Each stimulus was presented 300 times in each block, for each listening condition for total of 900 presentations, which were delivered by E-prime software. The whole study including the two experimental paradigms took around two hours; 30 minutes for the non-speech experiment and 60 minutes for the speech stimuli experiment with a few short breaks between the blocks and experiments to decrease the risk of fatigue.

3.2.6. Electrophysiological recording

The electrical responses were recorded from 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). The electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp, using the international 10-20 system of electrode placement. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. The tip of the nose served as an online reference for all channels, including the electro-oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. The EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for later analyses.

3.2.7. Electrophysiological data analysis

The data were analysed using Brain Products' Analyzer2 software. The signals were digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts were corrected in the EEG using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1995). To do so required a computation of vertical and horizontal EOG activity. A vertical EOG was computed by subtracting the inferior orbital and the FP2 channels and horizontal eye movements were computed by subtracting the FT9 and FT10 channels (Duda-Milloy et al., 2019). The ICA model assumes that the observed electrophysiological signals represent a linear mixture of neural sources and artefacts that are unknown but are mutually statistically independent. The algorithm relies on blind source separation which was trained to recognize each participant's ocular artefact signature which

was then partitioned out of the EEG traces. Epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset. Segmented waveforms were baseline corrected around the 100ms pre-stimulus interval and averaged based on condition. Epochs were excluded if they had amplitudes exceeding ±100μV at any site. Less than 10% of trials were excluded. Moreover, the EEG was visually inspected to ensure adequate artifact rejection. Averaged waveforms were computed for each subject and then across subjects to obtain grand averaged waveforms for each condition. Although both Cz and Fz were selected for the detection of frontocentral responses (P1-N1-P2-N2), only the responses at Cz are reported as there were more robust responses at Cz compared to Fz (Näätänen & Picton, 1987). This method is consistent with previous studies that have reported the P1-N1-P2-N2 responses at Cz, because this electrode showed the largest amplitude values, and hence were more susceptible to reveal statistical findings in children (Duquette-Laplante et al., 2024; Hussain et al., 2022; T. S. Mattsson et al., 2019; Morlet et al., 2019). Additionally, the amplitude of the N1 at Cz has been considered the strongest predictor of individual speech-in-noise perception thresholds (Billings et al., 2013). The T-complex was extracted from the temporal regions, T7 and T8 (Wolpaw & Penry, 1975). Tonnquist-Uhlen and colleagues (2003) also suggest that T7 and T8 are optimal for measuring the T-complex since they reflect little activity from the sources underlying the P1, N1 and P2. Regarding T-complex components, as Na was not detected clearly in most of the participants; accordingly, only Ta and Tb are reported in the results. Also, Tb was not identifiable in Babble noise with /da/, so this condition was removed from the analysis of the amplitude and latency of Tb. The more information of distribution of data has been provided in supplementary documents.

A peak detection procedure was performed for each participant to measure peak latency for each component. Regarding amplitude measurements a mean amplitude method was applied. Mean amplitude rather than peak amplitude is less affected by noise, leading to more efficient responses (Clayson et al., 2013; Luck, 2012). The mean amplitudes were calculated based on an interval between two points (25ms) on either side of the peak (i.e., a 50ms window) for each peak. This time window was based on the grand averaged ERPs at sites Cz, T7 and T8 (Anderson, Chandrasekaran, et al., 2010; J. A. Hämäläinen et al., 2011). The latency intervals over which each of the mean amplitudes were calculated are reported in Table 2.

Table 12. *Latency intervals over which each of the mean amplitudes were calculated.*

Peak-site	The latency interval based on Listening condition- Stimuli type					
	Quiet-1kHz	Quiet-da	White noise-1kHz	White noise-da	Babble noise-1kHz	Babble noise-da

P1	61-111ms	55-105ms	61-111ms	75-125ms	47-97ms	89-139ms
N1	105-155ms	105-155ms	103-153ms	125-175ms	105-155ms	223-273ms
P2	153-203ms	161-211ms	155-205ms	189-239ms	145-195ms	351-401ms
N2	251-301ms	259-309ms	265-315ms	293-343ms	247-297ms	421-471ms
Ta-T7	103-153ms	123-173ms	115-165ms	135-185ms	103-153ms	231-281ms
Tb-T7	147-197ms	169-219ms	151-201ms	195-245ms	145-195ms	Not identifiable
Ta-T8	103-153ms	123-173ms	115-165ms	141-191ms	109-159ms	227-277ms
Tb-T8	147-197ms	171-221ms	161-211ms	189-239ms	151-201ms	Not identifiable

3.2.8. Statistical analysis

The statistical analysis was conducted using JASP (Jeffreys's Amazing Statistics Program). Repeated measures analyses of variance (ANOVA) were performed to compare the amplitude and latency of the CAEPs. All data underwent preliminary screening to ensure that the assumptions of ANOVA were met. Normality was assessed using both visual inspection of Q-Q plots generated from the present dataset and the Shapiro–Wilk test, which is appropriate for small sample sizes, the results of both analyses have been included in an appendix. The results of the Shapiro–Wilk tests indicated that the majority of the variables were normally distributed. Therefore, the assumptions for the parametric analyses were adequately met. The Q-Q plots also showed that the residuals were approximately normally distributed, as the points closely followed the diagonal line, with no substantial deviations. The Greenhouse-Geisser correction was automatically applied to adjust the degrees of freedom in case Mauchly's test of sphericity was violated.

A 2 (Stimulus: 1kHz pure tone, syllable /da/) x 2 (Electrode Location: T7, T8) x 3 (Listening condition (quiet, white noise, babble noise) repeated measures ANOVA was calculated separately for the amplitude and latency of the Ta and Tb responses. A 2 (Stimulus: 1kHz pure tone, syllable /da/) x 3 (Listening condition (quiet, white noise, babble noise) repeated measures ANOVA was calculated separately for the amplitude and latency of P1, N1, P2 and N2.

3.3. Results

3.3.1. Amplitude

3.3.1.1. Ta

The repeated measures ANOVA revealed that the Ta response was larger when evoked by a speech sound compared to a pure-tone (*F (1,14) = 18.629, p<0.001, η²=0.146*) and was larger over the right hemisphere compared to the left (*F (1,14) = 7.095, p=0.019, η²=0.07*). There was also a marginal main effect of Listening Condition (*F (2, 28) = 3.045, p=0.064, η²= 0.032*). Follow-up *t*-tests, using the Bonferroni correction revealed a significantly smaller amplitude of Ta in white noise vs. quiet (*t*(df) = 3.071, *p=0.025*, *d* = 0.428) (Figure1, 2 and 3), and no difference between babble noise and quiet (*t*(df) = 0.994, *p=1.000*, *d* = 0.186) *p=1.000*) or. white noise (*t*(df) = -1.271, *p=0.673*, *d* = -0.241). None of the interactions were significant (*p >0 .05*).

3.3.1.2. Tb

The repeated measures ANOVA revealed the significant effect of stimuli on amplitude of Tb (*F (1,14) = 11.488, p=0.004, η²=0.153*). The effect of listening condition (*F (2, 28) = 2.124, p=0.167 η²= 0.022*) and location was not significant (*F (1,14) = 0.530, p=0.479, η²=0.006*) (Figure1, 2 and 3). Follow up t-test showed a larger response of Tb when evoked by a pure-tone sound compared to a speech (*t*(df) = -3.389, *p=0.004*, *d* = -0.685). The interaction was not significant (*p>0.05*) (Figure 1 and 2).

3.3.1.3. P1

A repeated measures ANOVA showed not significant effect of Stimuli (*F (1, 14) = 4.014, p<0.065, η²= 0.041*). There was a significant effect of Listening condition (*F (2, 28) = 4.798, p<0.016, η²= 0.151*). Follow up t-test results with Bonferroni correction revealed a smaller amplitude of P1 in white noise vs. quiet (*t*(df) = 3.126, *p=0.022*, *d* = 0.791), and no difference in P1 amplitude in babble noise vs. quiet (*t*(df) = 2.217, *p=0.131*, *d* = 0.772) and in white noise vs. babble noise ((*t*(df) = 3.126, *p=1.000*, *d* = -0.019) (Figure1, 2 and 3). None of the interactions was significant (*p > .05*).

3.3.1.4. N1

A repeated measures ANOVA showed a significant effect of stimuli (*F (1, 14) = 13.377, p<0.003, η²= 0.064*) and Listening condition (*F (2, 28) = 6.340, p<0.005, η²= 0.159*), and a significant interaction between stimuli and listening condition (*F (2, 28) = 11.707, p<0.001, η²= 0.163*). Follow up T-test with Bonferroni correction was used to investigate the effect of

interaction and results showed that the significant effect of listening condition only for speech stimuli. There was a larger amplitude of N1 in babble noise, compared to white noise (t(df) = 3.220, $p=0.048$, d = 1.000). There was no difference in amplitude of N1 between quiet and white noise (t(df) = 0.397, $p=1.000$, d = 0.108), nor between quiet and babble noise (t(df) = 3.220, $p=0.093$, d = 1.000). Regarding the effect of stimuli, it was significant only in babble noise, there was larger amplitude of N1 in response to /da/ than 1kHz (t(df) = 4.727, $p=0.005$, d = 1.425) (Figure 1 and 2).

3.3.1.5. P2

A repeated measures ANOVA showed no significant effect of stimuli ($F (1, 14) = 0.125, p=0.729, \eta^2= 0.001$), but significant effect of Listening condition ($F (2, 28) = 9.578, p<0.001, \eta^2= 0.191$). Follow up t-test results with Bonferroni correction showed smaller amplitude of P2 in babble noise vs. quiet (t(df) = 4.655, $p<0.001$, d = 0.963) and vs. white noise (t(df) = 3.628, $p<0.008$, d = 0.881), and no difference between white noise and quiet (t(df) = 0.299, $p=1.000$, d = 0.083) (Figure1, 2 and 3). The interaction between stimuli and listening condition was not significant ($p> 0.05$).

3.3.1.6. N2

A repeated measures ANOVA showed significant effects of stimuli ($F (1, 14) = 4.920, p=0.044, \eta^2= 0.037$) and Listening condition ($F (2, 28) = 19.983, p < 0.001, \eta^2= 0.369$), and interaction between stimuli and listening condition ($F (2, 28) = 4.600, p=0.019, \eta^2= 0.054$). Follow up t-test with Bonferroni correction was used to investigate the effect of interaction and results showed that the effect of listening conditions was dependent on stimuli. Using 1kHz stimuli, there was a significantly smaller amplitude of N2 in white noise vs. quiet (t(df) = -4.159, $p=0.014$, d = -1.011) and babble noise vs. quiet (t(df) = -5.174, $p=0.002$, d = -1.237) (Figure1, 2 and 3), and no difference between white noise and babble noise (t(df) = -0.993, $p=1.000$, d = -0.226). With /da/, there was smaller amplitude of N2 in babble noise vs. quiet (t(df) = -3.795, $p=0.030$, d = -1.654) and vs. white noise (t(df) = -3.999, $p=0.02$, d = -1.322) (Figure1 and 3), and no difference between white noise and quiet (t(df) = -1.323, $p=1.000$, d = -0.332). Also, according to the follow up test, there was no significant difference in amplitude of N2 between 1kHz and /da/ in any listening conditions (quiet; t(df) = -1.984, $p=1.000$, d = -0.460, white noise; t(df) = 1.302, $p=1.000$, d = 0.219, babble noise; t(df) = -2.380, $p=0.481$, d = -0.877.

3.3.2. Latency

3.3.2.1. Ta

A repeated measures ANOVA showed significant effect of Listening condition (quiet, white noise and babble noise) (*F (2, 32) = 169.552, p<0.001, η²= 0.315*) and stimuli *F (1, 14) = 179.527, p<.001, η²= 0.295*, but no significant effects of location (T7 vs. T8) (*F (2, 32) = 0.793, p<.388, η²=* 5.822×10^{-4}). There was also a significant interaction between Listening condition and Stimuli (*F (2, 32) = 134.000, p<.001, η²=* 0.262). Follow up t-test with Bonferroni correction was used to investigate the interaction between listening conditions and stimuli. Results showed that the effect of listening conditions was different between stimuli. Using 1kHz, there was significantly longer latency of Ta only in white noise vs. quiet (*t*(df) = -3.458, *p=0.058*, *d* = 0.058), no difference between babble noise and quiet (*t*(df) = -2.347, *p=0.363*, *d* = 0.512), and no difference between white noise and babble noise (*t*(df) = 0.118, *p=1.000*, *d* = 0.020). Using /da/ there was longer latency of Ta in babble noise vs. quiet (*t*(df) = -16.014, *p<0.001*, *d = 6.093*) and white noise (*t*(df) = -11.604, *p<0.001*, *d* = -6.093), and in white noise vs. quiet, (*t*(df) = -8.152, *p<0.001*, *d* = -1.411) (Figure 2). Also, the effect of stimuli was significant only in noisy conditions, there was longer latency to /da/ than 1kHz in white noise (*t*(df) = -7.719, *p<0.001*, *d = -1.409*), and in babble noise (*t*(df) = -13.456, *p<0.001*, *d = -6.110*).

3.3.2.2. Tb

A repeated measures ANOVA (Table 9) showed significant effects of Listening condition (*F (1, 14) = 50.510, p<0.001, η²= 0.208)*, and stimuli (*F (1, 14) = 42.258, p<.001, η²= 0.394*), but no significant effect of location (*F (1, 14) = 0.0496, p=0.493, η²=0.002*). There was a significant interaction between Listening condition and stimuli (*F (1, 14) = 10.828, p=.005, η²=0.017*). Follow up test with Bonferroni correction was used to investigate the effect of interaction. The effect of listening condition was significant for both stimuli. With /da/, there was longer latency of Tb in white noise vs. quiet (*t*(df) = -6.798, *p<0.001*, *d = -1.603*) (Figure 2 and 3). Using 1kHz, the t-test showed a significantly longer latency of Tb in white noise vs. quiet (*t*(df) = -5.188, *p<0.001*, *d = -0.890*). Also, regarding the effect of stimuli, there was a longer latency to /da/ than 1kHz in quiet (*t*(df) = -4.751, *p<0.001*, *d = -1.359*), and in white noise *t*(df) = -7.283, *p<0.001*, *d = -2.073*).

3.3.2.3. P1

A repeated measures ANOVA showed significant effects of Listening condition (*F (2, 28) = 40.100, p<0.001, η²= 0.319*) and stimuli (*F (1, 14) = 35.052, p<0.001, η²= 0.203*), and a significant interaction between listening conditions and stimuli (*F (2, 28) = 12.637, p<0.001, η²= 0.135*). Follow up *t*-tests with Bonferroni correction was done for evaluating the effect of interaction. Results showed the effect of listening condition was different between stimuli. Using /da/, there was a significant longer latency of P1 in white noise vs. quiet (*t*(df) = -8.447, *p<0.001*, *d = -1.781*), and in babble noise vs. quiet *t*(df) = -8.427, *p<0.001*, *d = -3.201*), and no difference between white noise and babble noise *t*(df) = -3.097, *p=0.118*, *d = -1.420*). With 1kHz, there was no significant effect of white noise *t*(df) = -2.714, *p=0.252*, *d = -0.749*) and babble noise on latency of P1 *t*(df) = -2.440, *p=0.429*, *d = -0.661*) (Figure 2 and 3). Also, the effect of stimuli was significant in babble noise and P1 had a longer latency for /da/ vs. 1kHz (*t*(df) = -5.775, *p<0.001*, *d = -2.628*), stimuli did not show significant effect on latency of P1 in white noise (*t*(df) = -6.522, *p=0.189*, *d = -2.539)*, P1 had a longer latency for /da/ vs. 1kHz.

3.3.2.4. N1

A repeated measures ANOVA showed significant effects of Listening condition (*F (2, 32) = 107.556, p<0.001, η²= 0.363*) and stimuli (*F (1, 14) = 72.186, p<0.001, η²= 0.215*), and a significant interaction between listening condition and stimuli (*F (2, 28) = 79.039, p<0.001, η²= 0.268*). Follow up t-tests with Bonferroni correction was done for evaluating the effect of interaction. Results showed the effect of listening condition was different between stimuli. Using /da/, there was longer latency in babble noise vs. quiet (*t*(df) = -21.090, *p<0.001*, *d = -5.513)*, in white noise vs. quiet (*t*(df) = -3.733, *p=0.003*, *d = -1.674),* and in babble noise versus white noise (*t*(df) = -3.257, *p=0.086*, *d = -1.023)* (Figure 2 and 3). Using 1kHz, there was no significant effect of babble noise (*t*(df) = -2.390, *p=0.472*, *d = -0.591)*, nor white noise (*t*(df) = -3.045, *p=0.131*, *d = -0.698)* on latency of the N1 (Figure1, 2 and 3). Also, the effect of stimuli was significant in babble noise (*t*(df) = -21.531, *p<0.001*, *d = -4.862)*, N1 had a longer latency for /da/ vs. 1kHz, and it was near-to-significant in white noise (*t*(df) = -3.469, *p=0.056*, *d = -0.917)*.

3.3.2.5. P2

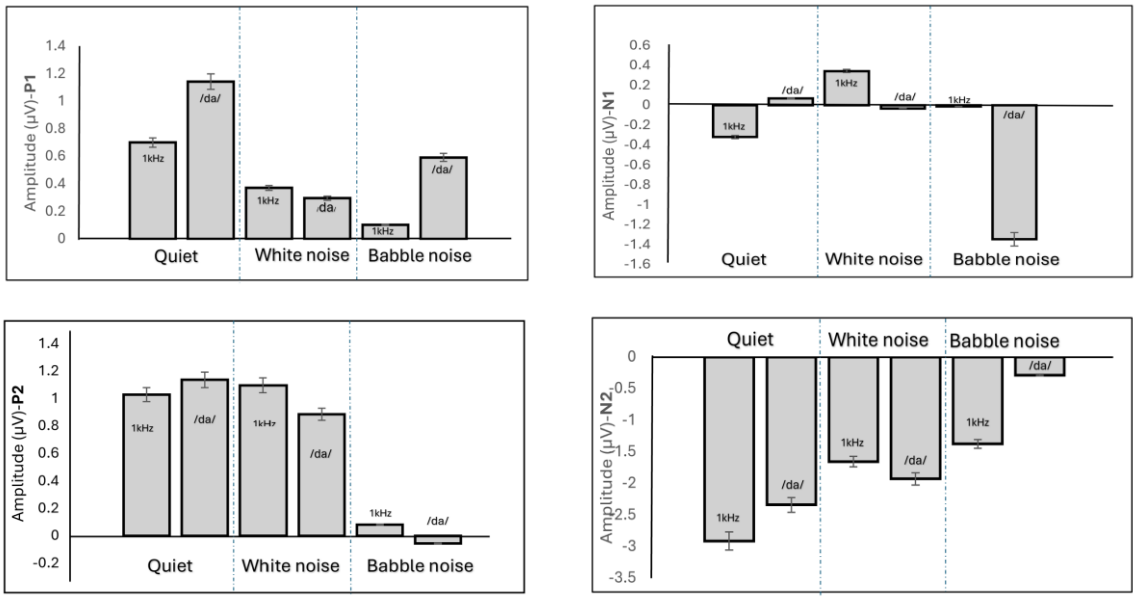
A repeated measures ANOVA showed significant effects of Listening condition (*F (2, 32) = 317.778, p<0.001, η²= 0.369*) and stimuli (*F (1, 14) = 192.763, p<0.001, η²= 0.265*),

and a significant interaction between listening conditions and stimuli (*F (2, 28) = 183.737, p<0.001, η²= 0.306*). Follow up with Bonferroni correction tests was done for evaluating the effect of interaction. Results showed the effect of listening condition was different between stimuli. Using /da/, there was longer latency in babble noise vs. quiet (*t*(df) = -18.455, *p<0.001*, *d = -8.099)*, in white noise vs. quiet (*t*(df) = -4.661, *p=0.006*, *d = -1.229)* and in babble noise vs. white noise (*t*(df) = -23.919, *p<0.001*, *d = -6.972)*. With 1kHz, there was a near-to-significant longer latency of P2 in white noise vs. quiet (*t*(df) = -3.332, *p=0.074*, *d = -0.766)*, but not in babble noise vs. quiet (*t*(df) = -1.855, *p=1.000*, *d* = 1.000*)* (Figure 2 and 3). Also, the effect of stimuli was significant only in noisy conditions, P2 had a longer latency for /da/ vs. 1kHz in babble noise (*t*(df) = -27.031, *p<0.001*, *d = -7.979)* and in white noise (*t*(df) = -4.349, *p=0.01*, *d = -1.007)*.

3.3.2.6. N2

A repeated measures ANOVA showed significant effects of Listening condition (*F (2, 32) = 109.974, p<0.001, η²= 0.312*), and stimuli (*F (1, 14) = 132.970, p<.001, η²= 0.303*) and a significant interaction between Listening condition and stimuli (*F (2, 32) = 119.920, p<.001, η²= 0.294*). Follow up t-tests with Bonferroni correction was done for evaluating the effect of interaction. Results showed the effect of listening condition was different between stimuli. Using /da/ stimuli, there was a longer latency of N2 in babble noise vs. quiet (*t*(df) = -13.707, *p<0.001*, *d = -6.663)* and in white noise vs. quiet (*t*(df) = -5.223, *p=0.002*, *d = -1.374)*, and in babble noise vs. white noise (*t*(df) = -10.488, *p<0.001*, *d = -5.289)*. Using 1kHz, N2 had significantly longer latency of N2 in white noise vs. quiet (*t*(df) = -3.884, *p=0.024*, *d = -0.713)*. Using same stimuli, there was no significant effect of babble noise on latency of N2 (*t*(df) = -1.099, *p=1.000*, *d = -0.225)* (Figure 2 and 3). Regarding the effect of stimuli, it was significant only in noisy conditions, N2 had a longer latency for /da/ vs. 1kHz in babble noise (*t*(df) = -12.705, *p<0.001*, *d = -7.006)* and in white noise (*t*(df) = -5.612, *p<0.001*, *d = -1.228)*.

A)



B)

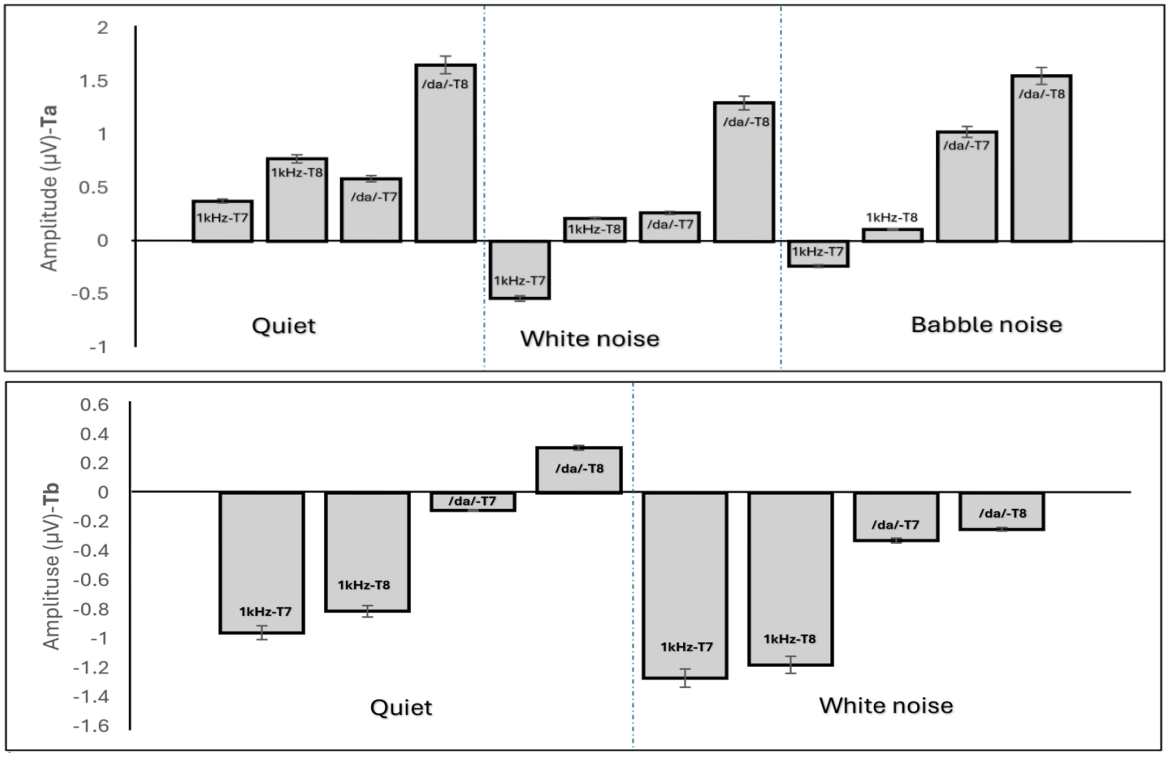
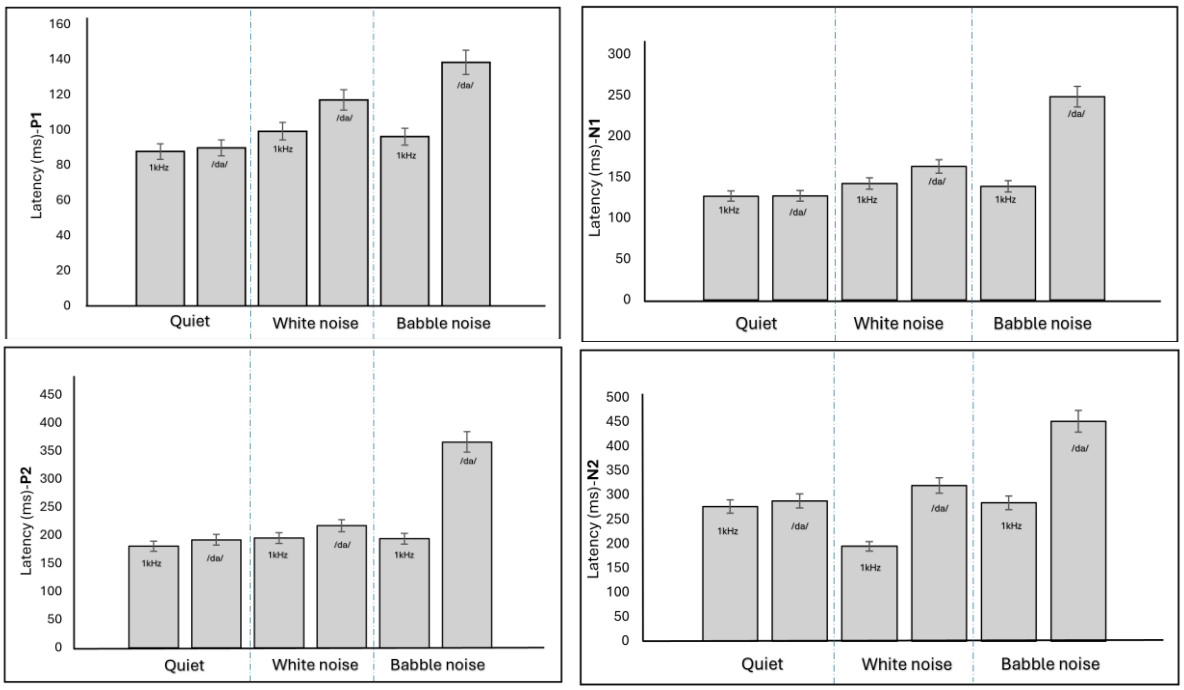


Figure 5. *A) The average of Amplitude of P1 and N1(A upper), P2 and N2 (A lower), recorded at electrode Cz, in three listening conditions, to /da/ and 1kHz. B) The average of Amplitude of Ta (B upper), recorded at electrodes T7 and T8 in three listening conditions including quiet, white noise and babble noise, to /da/ and 1kHz, and the average of Amplitude of Tb (B lower), at electrodes T7 and T8, in two listening conditions including quiet, white noise, to /da/ and 1kHz.*

A)



B)

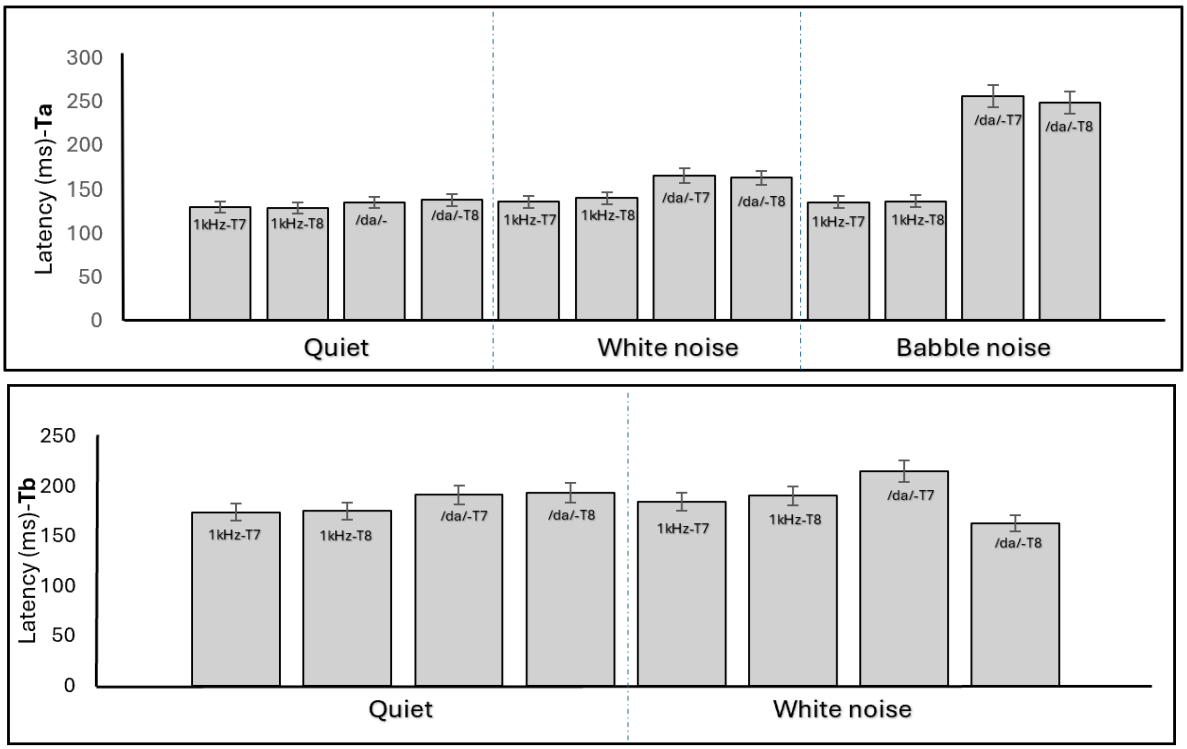


Figure 2. *A) The average of Latency of P1 and N1(A upper), P2 and N2 (A lower), recorded at electrode Cz, in three listening conditions, to /da/ and 1kHz. B) The average of Latency of Ta (B upper), recorded at electrodes T7 and T8 in three listening conditions including quiet, white noise and babble noise, to /da/ and 1kHz, and the average of Latency of Tb (B lower), at electrodes T7 and T8, in two listening conditions including quiet, white noise, to /da/ and 1kHz.*

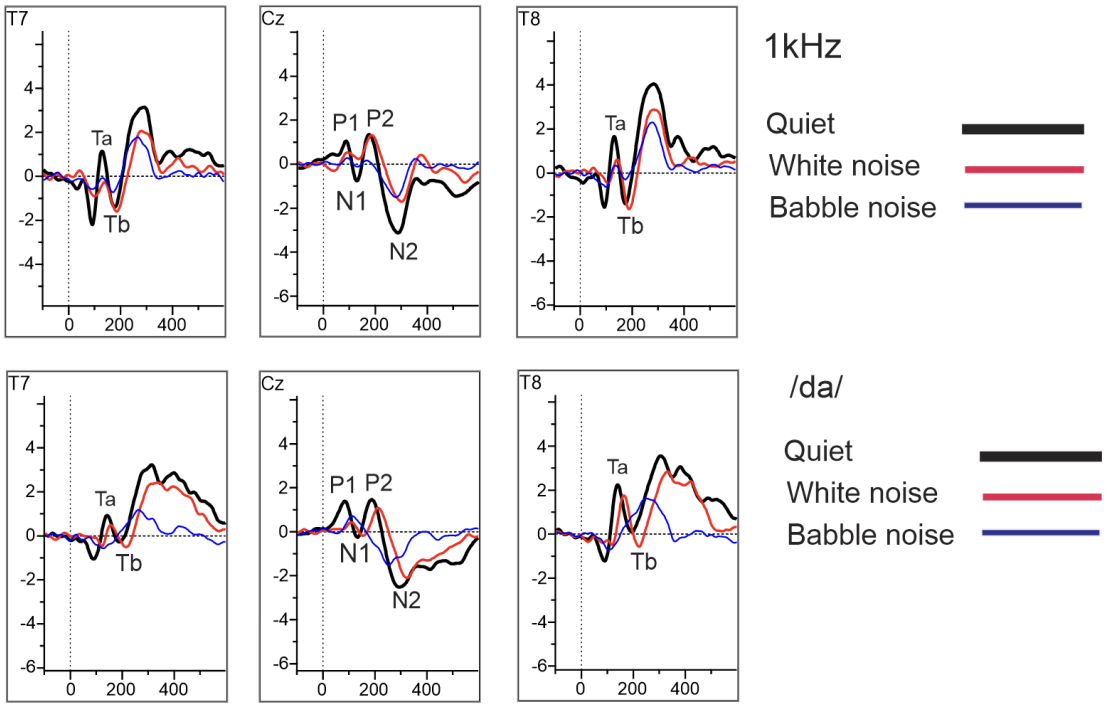


Figure 6. *The average of auditory evoked potentials waves, at electrodes Cz, T7 and T8, in response to 1kHz (upper) and da (lower), in Quiet, and in the presence of White noise and Babble noise. The amplitude of AEPs was reduced and latency of AEPs were prolonged in noisy conditions, compared to quiet.*

Table 13. *Summary of the results.*

Component	Amplitude modulation in noise	Latency modulation in noise
Ta	1kHz: Babble = White noise < Quiet = Babble /da/: Babble = White noise < Quiet = Babble	1kHz: White noise > Quiet = Babble = White noise. /da/: Babble > White noise > Quiet /da/ > 1kHz, in noises.
Tb	1kHz: Babble = White noise = Quiet /da/: Babble = White noise = Quiet	1kHz: White noise > Quiet /da/: White noise > Quiet /da/ > 1kHz.
P1	1kHz: Babble = White noise < Quiet = Babble /da/: Babble = White noise < Quiet = Babble	1kHz: Babble = White noise < Quiet /da/: Babble = White noise > Quiet < Babble /da/ > 1kHz, in noises.
N1	1kHz: Babble = White noise = Quiet /da/: Babble < White noise = Quiet = Babble	1kHz: Babble = White noise = Quiet /da/: Babble > White noise > Quiet. /da/ > 1kHz, in noises.
P2	1kHz: Babble < White noise = Quiet > Babble /da/: Babble < White noise = Quiet > Babble	1kHz: White noise > Quiet = Babble /da/: Babble > White noise > Quiet. /da/ > 1kHz, in noises.
N2	1kHz: Babble < Quiet > White noise /da/: Babble < White noise < Quiet	1kHz: White noise > Quiet = Babble /da/: Babble > White noise > Quiet. /da/ > 1kHz, in noises.

3.4. Discussion

This study aimed to evaluate the effect of noise on frontocentral and T-complex responses in children, using speech and non-speech stimuli in the presence of white noise and babble noise (Figure 3). Similar to frontocentral responses, the T-complex was expected to show noise-induced changes. There was significant interaction between listening condition and stimuli for amplitude changes of N1 and N2. Latency was consistent across all of the AEPs, with significantly longer latency in noise when stimulus was speech, while with non-speech stimuli the changes were smaller. Most of the AEPs showed an interaction between listening condition and stimuli, with longer latency to speech than non-speech, especially when noise was babble. The details of results have been elaborated in the following.

3.4.1. Amplitude changes

3.4.1.1. Temporal responses

The amplitude of both Ta and Tb were impacted by noise similarly for both types of stimuli; however, the impact on each was different and it was dependent to the noise type. Ta was smaller when the stimuli were embedded in white noise versus quiet for both speech and pure-tone stimuli, while the amplitude of Tb was not significantly impacted by white noise (Figure 3). Contrary to white noise, it seems that babble noise suppressed the Tb, while Ta did not change significantly in babble noise.

This is the first study evaluating the effect of noise on T-complex in children. Furthermore, there are no published reports in the adult population either, so it is impossible to compare the current results with previous studies. However, the suppression of frontocentral AEP amplitudes in noise have been reported before in children (Duquette-Laplante et al., 2024; Hussain et al., 2022; Ubiali et al., 2025). Bottom-up and top-down auditory pathways can be affected by noise leading to reduced amplitude of auditory cortical responses (Anderson, Chandrasekaran, et al., 2010; J. Cunningham et al., 2001; Katherine A. Whiting et al., 1998b; Wible et al., 2005). Noise would mask physically the onset of the stimuli and make it less audible leading to delayed and smaller amplitude of auditory evoked potentials (Billings et al., 2013; Kaplan-Neeman et al., 2006; Katherine A. Whiting et al., 1998b), also the reduced amplitude of AEPs in noise may reflect efficient inhibition control leading to the suppression of the unwanted background noise, manifested as smaller amplitude in noisy conditions (Anderson, Chandrasekaran, et al., 2010; Ceponiene et al., 2002; Guinan, 2006; Ryugo et al., 2011).

The novel finding of this study was the opposite pattern of changes in amplitude between Ta and Tb in noise. Tb disappeared in babble noise, compared to white noise for 1kHz. Regarding speech stimulus, the suppression of Tb was even stronger by babble noise, compared to white noise. In the babble noise condition, the Tb component in response to /da/ could be reliably identified in only 4 of the 15 participants. Because the waveform became flatter and broader in the remaining participants, preventing consistent peak identification, this condition was excluded from the statistical analyses. The greater suppression of Tb amplitude in 4-talker babble noise compared to white noise resembles previous studies showing more detrimental effects of babble noise than white noise on frontocentral responses (Billings et al., 2011; Duquette-Laplante et al., 2024). Babble noise introduces informational and energetic masking effects which affects the allocation of attention to target stimuli, while white noise, which is exclusively energetic masking, masks the acoustical features of the stimuli (Billings et al., 2011). As such, babble noise has been considered a stronger modulator of the auditory evoked responses compared to white noise (Billings et al., 2011; Billings & Grush, 2016). Considering this, it is not surprising to observe greater suppression of Tb in babble noise compared to white noise.

Interestingly, this pattern was contrary for Ta, which showed a non-significant effect of babble noise. The non-significant changes of Ta in babble noise versus white noise may presumably highlight greater adaptation of Ta's neural generators to babble noise compared to white noise due to the similarity between babble noise and real-world listening conditions. The distinct results between Ta and Tb may reflect the different neural generators and their contributions to speech perception in noise. Babble noise is an informational mask distracting attention to non-target speech, reducing the allocation of attention to target speech sounds. As Tb was strongly suppressed by babble noise, it may suggest that the neural generators of Tb contribute to or are influenced by top-down or efferent pathways leading Tb to be affected more by informational masking than energetic masking, compared to Ta which showed more resistance to adaptation by babble noise. Considering the neural generators, it has been proposed that the source of Tb is more localised to secondary auditory areas compared to Ta (Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). Presumably, the different sources may lead to different contributions to auditory processing and interaction with top-down processing. However, no previous studies have reported different effects of top-down processing on T-complex components, to our knowledge. Considering that the current results that have been observed using a passive paradigm, more research using active paradigms may bring more knowledge of

different interactions with top-down processing between Ta and Tb. Another possible reason for stronger suppression of Tb by babble noise while Ta was not changed, is that that multi-talker babble introduces both energetic and informational masking, which may reduce the temporal precision of the cortical response and make later components such as Tb more difficult to distinguish, compared to Ta. It is also important to consider that the relatively small sample size may have further limited the number of participants in whom a reliable Tb peak could be identified. This is the first study reporting the effect of noise on T-complex in children, so more research in the same population is warranted to confirm the results and bring more insights into how temporal responses in the developing auditory system change in noise.

3.4.1.2. Lateralization of T-complex, T7 vs. T8

Using both stimuli, there was a larger amplitude of Ta at electrode T8 compared to electrode T7. The larger amplitude of Ta in the right hemisphere observed in the present study aligns with earlier reports (Bruneau et al., 2015; Cacace et al., 1988; Wolpaw & Penry, 1975, 1977a) and may be rooted in well-documented anatomical asymmetries between the left and right temporal cortices. Prior studies examining brain structure have demonstrated asymmetry of the planum temporale surface, rightward asymmetry of the superior temporal sulcus (STS) and sulcal depth, as well as differences in the slope of the Sylvian fissure, with a steeper posterior angle typically found in the right hemisphere (Bonte et al., 2013; Geschwind & Levitsky, 1968; Hyde et al., 1973; LeMay & Culebras, 1972; Ochiai et al., 2004). Collectively, such anatomical variations could plausibly contribute to hemispheric differences in the orientation and location of the neural generators underlying the T-complex peaks, the right-hemispheric asymmetry for Ta observed in the current results. Another explanation for a larger response of Ta is the earlier maturation of auditory processing at right hemisphere than the left hemisphere leading to larger auditory responses at right sites than the left sites of the brain (Bishop et al., 2011; Valerie L. Shafer et al., 2015; Tonnquist-Uhlen et al., 2003). Compared to the left hemisphere that is sensitive to fast temporal processing, the right hemisphere contributes to processing of prosody and long duration modulations (Crystal, 1970; Mandel et al., 1994; Rose, 2002). Prosody is a suprasegmental linguistic cue that plays a crucial role in early language acquisition. The acoustic salience of prosody, even at the syllable level, such as in stressed syllables, helps capture infants' attention and guides them toward detecting speech structure (Kuhl, 2004; Mehler et al., 1988; Nazzi et al., 2000). Therefore, it might be plausible to observe earlier maturation of auditory processing at right hemisphere than the left

hemisphere as the right hemisphere is sensitive to representation of information that are critical for language development. More research is warranted to confirm this idea.

3.4.1.3. Frontocentral responses

The noise-induced changes in the amplitude of AEPs included primarily decreases in amplitude and were not always significant, especially for the N1 (Table 2 and Figure 3).

The most significant finding was observed for P2 and N2. The amplitude of P2 was suppressed in babble noise, similarly for both stimuli, but it was not affected by white noise. This finding again may highlight the more detrimental effect of babble noise compared to white noise on auditory processing (Billings et al., 2011; Billings & Grush, 2016; Douglas S Brungart et al., 2001). The N2 showed significantly smaller amplitude in both types of noise with non-speech stimuli. N2 also showed a significant interaction between listening conditions and stimuli, with smaller amplitude in babble noise compared to white noise, only when stimulus was speech. These results may show that the most difficult listening condition is the perception of speech in speech background noise (Billings et al., 2011). As no previous studies have reported the interaction of stimuli and noise type for N2 in children, more research is warranted to confirm this finding of more sensitivity of N2 to real-life listening conditions.

P1 had a smaller amplitude in white noise versus quiet for both types of stimuli, although it did not change significantly in babble noise. These results replicated recent findings reporting smaller amplitude of P1 in white noise versus quiet in children (Duquette-Laplante et al., 2024). However, generally, reports on P1 amplitude in the literature are inconsistent. Some studies have shown a decrease in P1 amplitude under noise conditions (Bertoli et al., 2005; Papesh et al., 2015), whereas others have reported no change (Billings et al., 2009; Gott & Hughes, 1989). Variability in P1 modulation by noise has been attributed to individual differences and methodological variations like using different signal-to-noise ratios in the acquisition and analysis of cortical AEP data across studies (Papesh et al., 2015). However, none of the previous studies have specifically examined the effect of noise type on P1 amplitude. In the present study, a reduction in P1 amplitude was observed only under the white noise condition, similar to Ta, it may suggest that noise type influences the strength of the P1 response. This finding may indicate that the neural generators of P1 are more resistant or better adapted to speech noise than to white noise, thereby preserving the representation of auditory stimuli. Further research using the same experimental paradigm is warranted to confirm this interpretation

N1 amplitude did not change significantly in noise conditions compared to quiet, but it showed a significantly larger amplitude in babble noise versus white noise for speech stimuli. The non-significant effects of noise on the N1 may be because of immature morphology (afferent auditory pathway) of this wave in the participants (Ubiali et al., 2016). A similar finding has been observed in the study of Ubiali et al., (2016), and it was mentioned that the absence of morphological maturity of the waveforms and the ongoing developing response of the N1 and P2 could probably mask any significant effect of noise (Ubiali et al., 2016).

The larger amplitude of N1 in babble noise, compared to white noise, for speech stimuli has not been observed in previous studies (Duquette-Laplante et al., 2024; Gustafson et al., 2019). However, the previous results have shown a larger amplitude of N1 in white noise (Duquette-Laplante et al., 2024) and in 4-talker babble noise (Gustafson et al., 2019) compared to quiet. Gustafson et al., 2019 suggested that the larger amplitude of N1 in babble noise is related t to incomplete maturation of auditory system in children. Duquette-Laplante et al., (2024) could not report the AEPs results in babble noise as they were not identifiable in 8-talker babble noise, which seems to be more detrimental than the 4-talker babble noise used in the current study. As the above studies did not report the noise type, it is hard to compare the currents result. The observed pattern; larger amplitude of N1 in babble noise compared to quiet is also contrary to the effect of type on AEPs in adult populations (Billings et al., 2011; Billings & Grush, 2016), which have shown a smaller amplitude N1 in babble noise versus white noise. However, the contradictory pattern of N1 amplitude in babble noise between adult studies and the current study with children may highlight the developmental changes and the greater vulnerability of the developing auditory system to speech in babble noise (Elliott, 1979; Leibold & Buss, 2013; Nittrouer & Boothroyd, 1990). Considering the nature of the babble noise which is influencing top-down processing and reducing the allocation of attention and inhibition processing to the target speech in noise, it may be speculated that babble noise would show a more detrimental effect on a population with less efficient inhibition processing like children, compared to adults. In other words, the current finding can be linked to immature top-down inhibitory processes that regulate auditory responses in noise. In particular, children are thought to have an underdeveloped corticofugal (efferent) system (Leibold, 2017; Rashid et al., 2016) leading to less suppression of irrelevant information, which in turn resulted in larger responses that are not finely tuned to the onset of speech stimuli (combination of competing speech and target speech information) (Anderson, Skoe, et al., 2010; Gustafson et al., 2019; Ryugo, 2010). However, the current results have been observed with a passive paradigm

reducing the engagement of top-down processing. Consequently, more research using active and passive paradigms will bring more knowledge on different affect of noise on children and adults. Another the possible explanation of larger amplitude of N1 in babble noise compared to white noise, for speech stimuli, is that it may reflect more engagement in processing the target in noise (Salisbury et al., 2002; van Dinteren et al., 2014), as the addition of babble noise results in an apparent increase in neural activity or effort, even when children are not attending to the stimulus (Anderson, Chandrasekaran, et al., 2010; Anderson, Skoe, et al., 2010). Given that the target speech in babble noise is close to real life communication settings and listening ability in noise is a key factor in learning among the children (Hunsaker, 1983), the accurate representation of speech in speech noise is highly crucial. Therefore, it may be speculated that more processing is happening in this condition to facilitate the encoding of speech in noise and is manifested by larger amplitude of N1 in babble noise with speech stimuli. However, more research using the same study design is warranted to confirm this hypothesis about how the developing auditory system responds in noise.

3.4.2. Latency changes

Latency of AEPs was generally longer in noise, than in quiet (Figure 3). The results on latency complement previous studies demonstrating prolonged auditory responses in noise at cortical levels (the slower neural conduction and processing time) (Anderson, Chandrasekaran, et al., 2010; K. A. King et al., 2008; Tremblay & Ross, 2007). The latency of all AEPs was generally longer in both types of noise, and it was longer in babble noise than white noise when the stimulus was speech compared to when it was non-speech. However, the latency of P1 and N1 did not change in noise when stimuli were non-speech, also the latency of P2, N2, Ta and Tb was prolonged using nonspeech stimulus only in white noise. The greater prolongation in noise with speech than non-speech combined with evidence of a non significant effect of noise on latency for non-speech stimuli may support the idea of more vulnerability to noise in speech AEPs than the non-speech evoked responses (Hannu Tiitinen et al., 1999). This finding replicated previous studies showing the difference in the effect of noise between responses to speech and non-speech stimuli in adults and children (Billings et al., 2011; Ubiali et al., 2025) . Billing et al. (2011) showed longer latency of N1 in noise, when stimuli was /ba/, compared to 1kHz in adults, and Ubiali et al., (2025) showed longer latency of P1-N1-P2-N2 in noise with /da/ than with 2kHz in children. The longer latency of AEPs to speech stimuli than non-speech stimuli have been explained by acoustical feature differences (Billings et al., 2011). Compared to tone, the speech stimuli contain more lower frequencies at the onset of the stimuli

than 1kHz, resulting in longer latency AEPs (Jacobson et al., 1992; H. Tiitinen et al., 1999). Also, it has been mentioned that as speech has greater acoustic complexity, the central auditory nervous system may need to employ more sophisticated top-down strategies to process it in noisy environments than it does for tones in noise (Billings et al., 2011; Ubiali et al., 2025).

However, according to the current results, the nonsignificant effect of noise for non-speech stimuli was observed only in white noise, not in babble noise. This is the first study showing this interaction between listening condition and stimuli on the latency of AEPs in children and there is no previous study to compare and interpret this finding. However, in normal-hearing adult populations, Billings et al., (2011) recorded AEPs with speech and non speech stimuli and three type of noises: interupted, continious and babble noise. There was an interaction between noise type and stimuli only for N1 latency. The longest latency of N1 was observed when the stimulus was speech and noise was speech. The author suggested that similarity between acoustic feature of the stimuli and noise may account for this pattern (Billings et al., 2011). Supportively, in the current study, there was stronger changes in the latency of AEPs when the stimuli and noise were the same, speech stimuli in speech noise and non-speech stimuli in non-speech noise.

3.5. Conclusion

This study showed the noise-induced changes in auditory frontocentral and temporal responses in children between 8 to 12 years old. Modulation included changes in amplitude and latency of AEPs in noise, using speech and non-speech stimuli. The noise-induced changes of some components seemed to interact with the type of the noise. While N2 showed smallest amplitude when stimuli and noise were speech, N1 did show a larger amplitude in babble noise compared to white noise with the same stimuli. However, the amplitude of P1, P2, Ta and Tb did not show effect of interaction of factors. This finding may demonstrate that the generators of AEP components may make different contributions to auditory processing in noise and may highlight the effect of top-down processing on auditory responses in noise. The latency was generally prolonged in noise, and it was longest when stimuli were speech. This finding may highlight that the representation of stimuli with various temporal-spectral features like speech in noise needs more time to processing compared to simple nonspeech stimuli like 1kHz tone. Taken together, these results may add insights into what variables are driving cortical signal-in-noise encoding and how auditory cortical components would react to them distinctively.

Also, the second component of Tb and the N1 frontocentral response did not show significant changes in amplitude in the presence of noise, compared to quiet. The non-significant results may show the immature afferent auditory pathway in generators of N1 and under-developed top-down processing or inhibition system in children leading to non-suppression of the response and non-meaningful changes in the amplitude of Tb.

This study as the first to show how noise modulates the auditory responses at different levels of auditory cortex including primary and secondary auditory areas recorded by frontocentral and temporal responses, respectively, in children. These results may further be effective to identify the origins of impairments of auditory processing in populations with poor listening abilities in noise, like children with APD.

3.6. Limitation and Future studies

While the current study provided valuable insights into frontocentral and temporal auditory late responses across different listening conditions, several limitations should be considered. First, the experimental paradigm included only one signal-to-noise ratio (SNR) level, which may have contributed to the nonsignificant noise-induced changes in the AEPs. Employing more challenging noise levels could potentially reveal stronger noise effects and lead to significant results. Second, only a passive listening paradigm was used. Given the possibility of top-down influences discussed earlier, conducting the same experiment but in an active task would provide additional information about the role of top-down processing in speech-in-noise perception. Third, the absence of significant noise effects may also reflect limited statistical power, as only 15 participants were tested. Studies with larger sample sizes and multiple noise levels would therefore be valuable. Fourth, considering the length of the experiments, there was a possibility for participants to get tired and lose their motivation. Therefore, fatigue could influence responses and reduce the clarity of auditory brain responses, however, break times between blocks were applied to reduce tiredness and increase motivation. An additional methodological consideration is the use of an offline common average reference. Although this approach is widely used in ERP research, its performance may be limited with relatively not very high-density EEG arrays (Luck, 2012), such as the 32-channel montage used in the present study. Consequently, the amplitudes of lateral temporal components, including the T-complex, should be interpreted with this consideration in mind. A final limitation of the present study is that behavioral speech perception measures were not obtained. Consequently, although the electrophysiological findings demonstrate the effects of background noise on

cortical auditory processing, they cannot be directly related to participants' speech perception performance. Future studies integrating behavioral and electrophysiological assessments within the same experimental paradigm would help clarify the functional significance of the observed cortical responses.

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Chapter 3. Manuscript 3

Lateralized representation of long and short voicing onset time with T-complex, in quiet and noise

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Abstract

Introduction: Voice onset time (VOT) is a temporal cue for differentiating voiced and voiceless consonants. Maintaining synchronized auditory neural responses to VOT is critical for successful speech perception in both quiet and noisy environments. Previous research has shown a left-hemisphere advantage for the processing of voiced versus unvoiced consonants. So far, no study has evaluated the representation of VOT using the T-complex, i.e., temporal auditory evoked potentials recorded at T7 and T8 (Ta and Tb components), particularly in noise. Examining the T-complex in response to syllables with varying VOTs may shed a light on the neural processing of temporal speech features within the superior temporal gyrus and in the secondary auditory areas. This study aimed to evaluate the modulation of the T-complex in response to short and long VOTs in quiet and in noise. It was hypothesized that T-complex components would reflect differential auditory processing of short versus long VOTs, with distinct patterns observed across the right and left hemispheres.

Method: Twenty students from the University of Ottawa (18-30 years old) participated in this study. During a passive listening paradigm, temporal auditory evoked potentials (AEPs; Ta and Tb components) in response to speech sounds /ta/ and /da/ syllables were recorded at T7 and T8 in both quiet and babble noise conditions (signal-to-noise ratio = 5dB SPL). In addition, behavioral measurements, including accuracy and reaction time during a task discriminating between /ta/ and /da/, were collected in both quiet and noise conditions.

Results: Ta had larger amplitudes in response to speech sound /da/ than speech sound /ta/ in both quiet and noise at electrodes T7 and T8. Tb amplitude was larger when evoked by the speech sound /ta/ compared to the speech sound /da/ in quiet, whereas the opposite pattern was observed in noise at electrode T8. Both components showed longer latencies in response to speech sound /ta/ than to speech sound /da/ in noise. In addition, lower accuracy and longer reaction times were observed for speech sound /ta/ compared to speech sound /da/ in noise.

Discussion: This study showed that long and short VOTs are represented by the T-complex. Tb, the last component of the T-complex, showed a larger amplitude in response to long VOTs compared to short VOTs in the right hemisphere, whereas this pattern was not same significant in the left hemisphere. These results indicate lateralization of temporal processing at the level of secondary auditory areas. This finding may be promising for characterizing impairments in temporal processing in populations with auditory processing difficulties at higher levels of the central auditory nervous system.

Keywords: T-complex, Voice onset time, Speech in noise, Lateralization.

4.1. Introduction

The ability to effectively perceive and comprehend speech in noisy conditions is essential for successful communication in educational, social, and professional settings (Anderson & Kraus, 2010). Speech comprehension in noisy environments depends on the accurate extraction and representation of the temporal characteristics of acoustic speech features (Cunningham et al., 2002; Schnupp et al., 2010; Shinn-Cunningham & Best, 2008). Difficulties in understanding speech, particularly in noise, can be partially attributed to impairments in the auditory system's ability to detect changes in speech features over time (i.e., temporal processing) (Anderson et al., 2011; Gordon-Salant et al., 2011; Tallal et al., 1993). Accordingly, detection and diagnosis of auditory temporal processing deficits may help early rehabilitation in populations with disrupted perception of speech in noise (Almeqbel & McMahon, 2015; Talcott et al., 2000; Warrier et al., 2004).

In natural speech, voice onset time (VOT) is a temporal cue for differentiating voiced and voiceless stop consonants and has been used for evaluation of temporal processing (Almeqbel & McMahon, 2015; Talcott et al., 2000; Warrier et al., 2004). VOT refers to the time interval between consonant release and the onset of low-frequency periodicity in the speech signal generated by rhythmic glottal pulsations (Sharma & Dorman, 1999). In American English, voiced stop consonants (/b/, /d/, and /g/) in syllable-initial positions exhibit short VOTs (0-20ms), whereas voiceless stop consonants (/p/, /t/, and /k/) are characterized by longer VOTs (30-100ms) (K. A. King et al., 2008; Sharma & Dorman, 1999; Steinschneider et al., 1999). Numerous studies have demonstrated asymmetric processing of VOT at the auditory cortex, with a left hemisphere advantage or enhanced processing for voiced compared to voiceless syllables (Sandmann et al., 2007; Steinschneider et al., 1999; Trébuchon-Da Fonseca et al., 2005).

VOT identification is highly dependent on synchronized neural responses evoked by the onset of voicing (Sinex & McDonald, 1989; Sinex et al., 1991). Maintaining neural responses that are time-locked to speech stimuli is critical for successful speech perception in both quiet and noise environments (Kraus and Nicol, 2003). One approach to observing subtle differences in synchronized responses to VOT is through auditory evoked potentials (AEPs). AEPs are neural responses generated by auditory stimulus and recorded from surface electrodes placed on the scalp (Luck, 2012). AEPs reflect neural activity associated with stimulus onset and have been widely used to investigate auditory neurophysiological correlates of voicing perception with high temporal precision (Hestvik et al., 2023; Hoonhorst et al., 2009; Martin & Boothroyd, 1999; Sharma & Dorman, 1999; Tremblay et al., 2003). Robust AEP responses are observed only when accurate neural synchronization to the auditory signal occurs (Luck, 2012). Therefore, using AEPs to examine the neurophysiological representation of the temporal speech features in quiet and in the presence of competing background noise may provide valuable insight into speech perception difficulties at multiple levels of the auditory cortex, including primary and secondary auditory cortex (Näätänen & Picton, 1987; Katherine A Whiting et al., 1998; Wolpaw & Penry, 1975).

The majority of AEPs studies have explored the impact of temporal speech features, such as the VOT of syllables, on frontocentral cortical evoked potentials (P1, N1, P2, N2), which are generated from Heschl's gyrus and other structures along the superior temporal plane (Sharma & Dorman, 1999; Sharma et al., 2000). In quiet listening conditions, syllables with long VOTs result in a double-peaked N1, with the first peak corresponding to consonant onset and the second time-locked to the onset of voicing. In contrast, syllables with short VOTs do not evoke a double-peaked N1, as the onset of voicing cannot be separated from the consonant release (Sharma & Dorman, 1999; Sharma et al., 2000). Additionally, longer VOTs are associated with increased latency (Frye et al., 2007; Tremblay et al., 2003) and decreased N1

amplitude (Toscano et al., 2010), compared to responses elicited by short VOT stimuli. Only a limited number of studies have investigated the representation of VOT under noisy listening conditions (Cushnie-Sparrow et al., 2016; J. H. Han et al., 2020; Kuruvilla-Mathew et al., 2015). For instance, Kuruvilla-Mathew (2015), demonstrated that in background noise, significant differences were observed in N1 latency and amplitude, as well as in P2 latency, when comparing responses to the voiced /di/ and the voiceless /ti/. Specifically, in noise, the N1 and P2 obtained from / ti/ were delayed, and the N1 amplitude was reduced relative to the N1/P2 responses evoked by /di/ (Kuruvilla-Mathew et al., 2015). In contrast, under quiet conditions, /ti/ and /di/ differed only in P2 latency. These findings indicate that background noise differentially affects neural encoding of onset characteristics and spectro-temporal features of speech stimuli, with longer syllable onsets being more susceptible to the adverse effects of noise (Kuruvilla-Mathew et al., 2015).

While the effects of background noise on VOT processing have been well documented using N1-P2, the effects of voicing time on temporal auditory responses have been relatively underexplored. Temporal cortical evoked potentials, referred to as T-complex, consist of the Na-Ta-Tb components and represent another class of auditory cortical responses. These responses originate from the superior temporal gyrus but are radially oriented (Pang & Taylor, 2000; Ponton et al., 2002; Ponton et al., 2000; Wolpaw & Penry, 1975). The Na-Ta-Tb components typically occur within a latency range of 50-200ms and are recorded at temporal electrodes located at T7 and T8. The Na, Ta and Tb components are characterized by negative, positive and negative polarities, respectively (Wolpaw & Penry, 1975).

To the best of our knowledge, only one study (Hoonhorst et al., 2009) has examined the representation of long and short VOTs in the T-complex, using stimuli presented in quiet conditions only. In that study, the Na component exhibited a double-peaked response for negative VOT values (Hoonhorst et al., 2009). However, no study has systematically

investigated all components of the T-complex, particularly the Ta and Tb components. Furthermore, the impact of background noise on the T-complex in response to long and short VOTs has not yet been examined.

Multiple studies have shown the capability of T-complex in representation of fast and slow temporal processing (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). So, it can be speculated that T-complex is sensitive to short and long duration of VOTs as well. Also, the superior temporal gyrus, the main generator of the T-complex, has been shown in neuroimaging studies to exhibit differential neural activity in response to voiced versus voiceless speech sounds (Belin et al., 2002; Belin et al., 2000; Rupp et al., 2022). Given the role of the T-complex generators in representing VOT, examining T-complex responses to syllables with varying VOTs may enhance our understanding of the neural processing of temporal speech features within the superior temporal gyrus. Such investigations, particularly in the presence of background noise, are clinically relevant as they may eventually contribute to better characterization of processing impairments in populations with poor speech-in-noise perception, such as individuals with auditory processing disorder (APD). A study by Hestvik et al. (2023) recorded auditory mismatch negativity (MMN) responses and reported impairments in VOT preprocessing in a group of individuals with APD. Considering that the generators of the MMN are thought to be mainly located in the primary auditory cortex and associated frontal lobes (Deouell et al., 1998), these findings may reflect abnormal encoding of VOTs at the level of primary auditory areas. Further research investigating the T-complex could provide additional insight into the representation of VOTs within secondary auditory areas.

The literature has also showed the sensitivity of the T-complex to atypical auditory processing, showing abnormal response patterns in children with listening difficulties compared to normal-hearing peers (M. A. Groen et al., 2008). These differences have been

observed in both latency (Bruneau et al., 1999; M. A. Groen et al., 2008; Rinker et al., 2021; Taylor et al., 2006; Tonnquist-Uhlén, 1996a) and amplitude measures (D. V. M. Bishop et al., 2012; Bruneau et al., 2003; Gomes et al., 2012; Rinker et al., 2021; Shafer et al., 2011; Taylor et al., 2003). Interestingly, several studies have reported discrepancies between T-complex and frontocentral responses (Bruneau et al., 1999; Gomes et al., 2012). Specifically, atypical T-complex patterns have been observed in populations with dyslexia and attention deficit/hyperactivity (ADHD) population (Gomes et al., 2012; Hämäläinen et al., 2011;Taylor et al., 2003), despite normal frontocentral responses. Accordingly, investigating T-complex responses to long and short VOTs in individuals with impaired auditory processing may provide additional insight into underlying neural deficits that are not captured by other AEPs. To evaluate deficits in VOT processing at higher levels of auditory system, as indexed by the T-complex, it is first necessary to characterize these responses in a normal-hearing population.

Therefore, this study aimed to assess T-complex responses to short and long VOTs in both quiet and noisy listening conditions. In the current investigation, both electrophysiological and behavioral responses to the syllables /da/ and /ta/ were evaluated in a group of normal-hearing adults under quiet and noise conditions. Comparing behavioral categorization performance with the auditory evoked potentials obtained by these syllables is expected to provide a more comprehensive understanding of VOT identification and perception.

Consistent with studies documenting differential modulation of frontocentral responses to short and long VOTs in quiet and noisy conditions (Cushnie-Sparrow et al., 2016; J. H. Han et al., 2020; Kuruvilla-Mathew et al., 2015), it is hypothesized that stimuli with varying VOTs and different listening conditions (quiet versus noise) will modulate T-complex responses. Specifically, T-complex components are expected to differentiate auditory processing of long and short VOTs in quiet and to show a more detrimental effect of background noise on temporal responses elicited by long VOTs. Moreover, previous studies have shown lateralization of

temporal processing in secondary auditory areas as indexed by the T-complex (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). According to Clunies-Ross et al. (2015), the Tb component could show different morphologies across the right and left hemispheres in response to tone-pair stimuli separated by short (25ms) and long (200ms) intervals. Accordingly, a significant effect of electrode location on T-complex responses is anticipated, with distinct patterns of temporal auditory responses between the right and left hemispheres for short and long VOTs.

4.2. Method

4.2.1. Ethics approval

The University of Ottawa Research Ethics Board approved this study, Ethic file number=H-07-24-10451. All participants received and signed a copy of the written consent form.

4.2.2. Participants

Twenty uOttawa students between 18-30 years old (Mean=28, SD=4.6; Male:9) participated in this study.

4.2.3. Inclusion and exclusion criteria

Participants had normal audiometric thresholds (i.e., 15 dB HL or less in octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996)'s protocol). Tympanograms were normal (admittance curve with a single peak between +50 to -100daPa using a 226-Hz probe tone). Participants were right-handed fluent in speaking and listening skills in English language according to the answers to the questionnaire of Languages known in order of dominance (LEAP-Q), as the babble noise was in English and it was ensured that all the participants were fluent in English language (Marian et al., 2007), with no history of otological or neurological disorders, and no history of musical training. All participants provided written informed consent.

4.2.4. Electroencephalography (EEG) Procedure

During the experiment, after familiarization with the testing equipment and procedure, the participants were seated in a sound-attenuated testing room, where they completed the experiment and EEG were recorded. The EEG study was passive, and participants were not required to make any responses to the stimuli. Participants were told to ignore the auditory stimuli and to focus their attention on the movie. Silent, subtitled films have been shown to capture attention without interfering with auditory processing (Pettigrew et al., 2004). This helps keep participants awake and alert during the study without attending to the auditory stimuli.

4.2.5. Stimuli

Stimuli were speech stimuli including syllables /da/ and /ta/ with a duration of 250ms (Dubno & Schaefer, 1992). These stimuli were natural; they were recorded a male voice and used by Dubno & Schaefer (1992). The /da/ and /ta/ have similar articulation place (Digeser et al., 2009) and contain similar formant frequencies (Figure 1). They differ mainly in consonant voicing which is cued by voice onset time (VOT), /da/ has a fast temporal dynamic at the onset of the syllables (short voicing time, around 20ms) and /ta/ has a slow temporal dynamic at the onset of the syllables (long voicing time, around 100ms) (Figure 1).

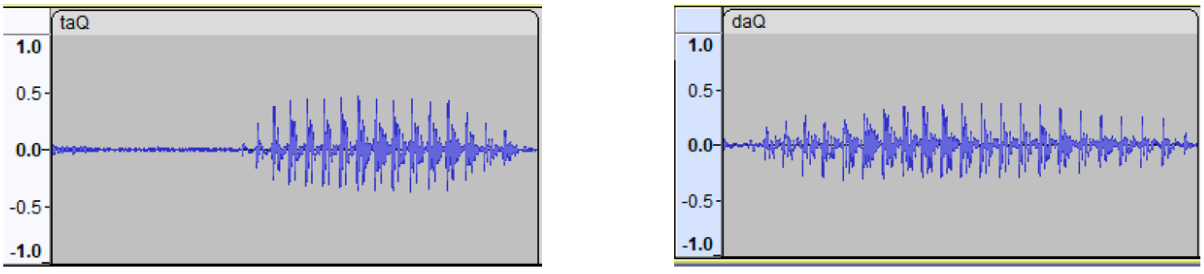

Figure 7. *The stimulus waveform and the spectrogram of syllables /da/ (left panel) and /ta/ (right panel). There is a longer duration at the onset of syllable ta versus /da/.*

Stimuli were presented using insert earphones (EARTone 3A) in two listening conditions: no babble noise (Quiet), continuous multi-talker babble noise (Babble noise), using

E-prime software. The babble noise (English-four-talker) was generated in the same method as the study of Zendel et al., (2015), but in English (Benjamin Rich Zendel et al., 2015). Stimuli and noise were equalized for root mean square (rms) amplitude and then played at 80 dB SPL. For the Babble noise condition, signal-to-noise ratio (SNR) was kept at +5dB, the intensity of noise was 75 dB SPL. Interstimulus interval (ISI), time from onset of one stimulus to the onset of next stimulus, was jittered between 1100-1250ms. There were 150 presentations of each speech stimulus (/da/ & /ta/) in each block, presented in each listening conditions, for a total of 600 trials in Quiet and 600 trials in Babble noise. In order to minimize evoked responses from the onset of babble noise, the vocal stimuli were presented in a random order within a block where the babble noise level was the same. The order of the blocks (Quiet, Babble noise) was random.

4.2.6. Behavioral (subjective) evaluation

In the behavioral experiment, the participant was instructed to discriminate between /ta/ and /da/. They needed to press the right button when they heard /ta/ and the left button when they are heard /da/. Accuracy was calculated according to the number of the number of hits (i.e., participant was presented /da/ and reported /da/) minus misses (participant was presented /da/ but reported /ta/), and Reaction time was calculated as the behavioral measure. The Accuracy and Reaction time for stimulus /da/ was compared to the results of stimulus /ta/.

4.2.7. Electrophysiological recording

The electrical responses were recorded from 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). The electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp, using 10-20 standard system. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. The tip of the nose served as an online reference for all channels, including the electro-

oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. The EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for later analyses.

4.2.8. Electrophysiological data analysis

The data were analysed using Brain Products' Analyzer2 software. The data were then digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as an off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts in the EEG were corrected using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1996). Then partitioned out of the EEG traces Epochs were excluded if they had amplitudes exceeding ±100μV at any site. Less than 10% of trials were excluded. Also, the EEG was visually inspected to ensure adequate artifact rejection. Epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset of stimulus. Averaged waveforms were computed for each subject and then across subjects to obtain grand averaged waveforms for each VOT value, which was time-locked to the onset of each syllable. Averaged waveforms also were baseline corrected using the 100ms pre-stimulus interval. The T-complex was extracted from the temporal sites (T7 and T8) (Wolpaw & Penry, 1975). Tonnquist-Uhlen and colleagues (2003) suggest that sites T7 and T8 are optimal for measuring the T-complex because they reflect little activity from the sources underlying the P1, N1 and P2. As Na was not clear in most of the participants, especially in the noisy condition, Ta and Tb were reported as the T-complex response. A peak detection procedure was performed for each participant to measure the latency of the peak. The first most positive deflection between 80-150ms as Ta, and Tb was the most negative deflection after Ta between 150-200ms. Mean amplitudes were calculated based on an interval two points (25ms) either side of the average peak (50ms window) for magnitude of each peak. This time window was based on the grand average ERP peaks at sites T7 and T8 (J. A. Hämäläinen et al., 2011). Mean amplitude

rather than peak amplitude was used because of concerns that peaks are more affected by babble noise in the signal (Luck, 2012).

4.2.9. Statistical analysis

The statistical analysis was conducted using JASP 0.19.1.0 software (Jeffreys's Amazing Statistics Program). A repeated measures ANOVA was used for comparing the AEPs measurements, the amplitude and latency. Firstly, all data underwent preliminary screening to ensure that the assumptions of ANOVA were met. The normality of residuals was assessed using Q-Q plots generated in JASP. The Q-Q plots showed that the residuals were approximately normally distributed, as the points closely followed the diagonal line, with no substantial deviations. The Greenhouse-Geisser correction was automatically applied to adjust the degrees of freedom in case that Mauchly's test of sphericity was violated.

Separate repeated-measures ANOVAs were performed for each response, analyzing amplitude and latency independently. Accordingly, A three-factor repeated-measures ANOVA was conducted separately for Ta and Tb. The within-subject factors were Listening Condition (Quiet, Babble noise), Stimuli (/da/, /ta/) and Electrode location (T7 and T8).

Regarding the behavioral responses, the paired sample T-Test was used for comparing the reaction time and accuracy for two groups including responses to speech sounds /da/ and /ta/.

4.3. Results

4.3.1. Behavioral results

In quiet, there was no significant difference in Accuracy ($t(19) = 0.047$, $p = 0.963$, Cohen's $d = 0.011$), between speech sounds /ta/ and /da/. However, the Reaction time was significantly longer for /ta/ compared to /da/ ($t(19) = -2.555$, $p = 0.019$, Cohen's $d = -0.571$). In babble noise, the speech sound /ta/ had significantly lower accuracy ($t(19) = 8.183$, $p =$

< .001, Cohen's *d* = 1.830), and longer Reaction time than speech sound /da/ (*t*(19) = -4.893, *p* = < .001, Cohen's *d* = -1.094) (Figure 2, Table 2).

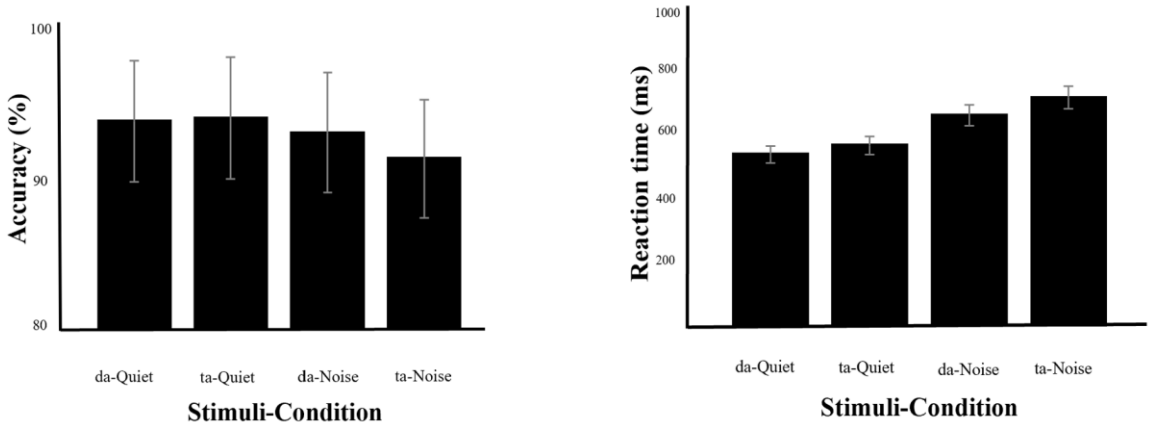

Figure 2. *The average of Accuracy (%) and Reaction time (ms) to /da/ and /ta/, in quiet and in babble noise. The accuracy of /da/ and /ta/ was similar in quiet, but accuracy was significantly lower to /ta/ versus /da /, in babble noise. The reaction time to /ta/ was significantly longer than /da/ in quiet and in noise.*

4.3.2. Electrophysiological results

4.3.2.1. Ta, Amplitude

A repeated measures ANOVA revealed no significant main effect of listening condition, *F* (1, 19) = 3.51, *p* = .076, η^2 = .056, and no significant effect of electrode, *F* (1, 19) = 0.01, *p* = .93, η^2 = .002. However, there was a significant main effect of stimulus, *F* (1, 19) = 20.56, *p* < .001, η^2 = .047. This effect was qualified by a significant interaction between listening condition and stimulus, *F* (1, 19) = 14.31, *p* = .001, η^2 = .020. Follow-up comparisons indicated that Ta amplitude was larger to speech sound /da/, compared to speech sound /ta/ in babble noise, *p* < .05, but not in quiet, *p* = .692 (Figure 3).

4.3.2.2. Tb, Amplitude

A repeated measures ANOVA showed a significant main effect of listening condition, *F* (1, 19) = 32.75, *p* < .001, η^2 = .341, a significant main effect of stimulus, *F*(1, 19) = 18.03, *p* < .001, η^2 = .040, and a significant main effect of electrode, *F*(1, 19) = 8.48, *p* = .009, η^2 = .068. There was smaller amplitude of Tb in babble noise, compared to quiet. The main effect of

stimulus was qualified by a significant interaction between stimulus and electrode, F (1, 19) = 15.11, $p < .001$, $\eta^2 = .016$. Follow-up comparisons revealed that Tb amplitude was significantly larger for speech sound /ta/ than for speech sound /da/ at electrode T8, when it was evoked in quiet, $p < .001$, whereas in babble noise showing there was a smaller mean amplitude of Tb for speech sound /ta/ than speech sound /da/. No significant difference was observed at electrode T7, $p = .483$ (Figure 3).

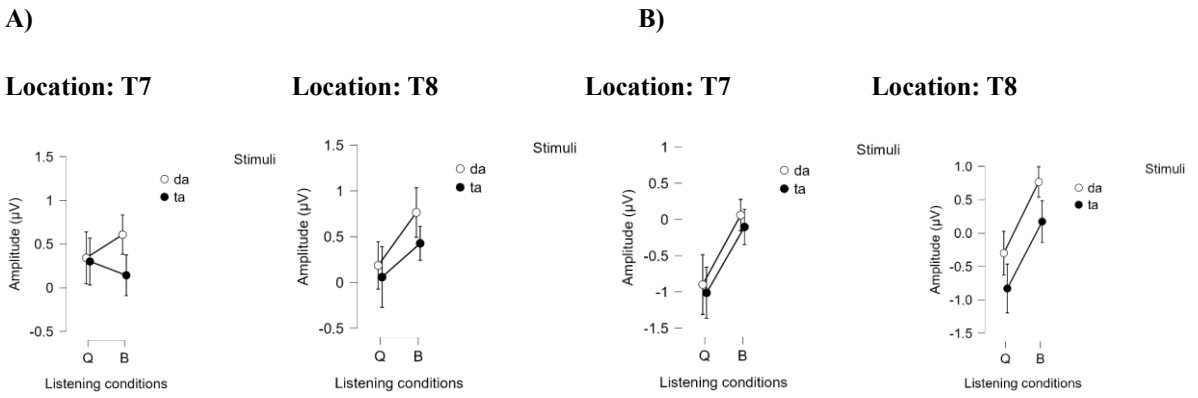

Figure 8 A. *The mean amplitude of Ta, to /da/ and /ta/, in quiet and noise, at T7 and T8. There was larger amplitude of Ta to /da/ than /ta/ in babble noise.* ***B.*** *The mean amplitude of Tb, to /da/ and /ta/, in quiet and noise, at T7 and T8. There was larger amplitude of Tb to /ta/ versus /da/ in quiet, and larger to /da/ than /ta/ in babble noise, at T8.*

4.3.2.3. Ta, latency

A repeated measures ANOVA revealed a significant main effect of listening condition, F (1, 19) = 570.80, $p < .001$, $\eta^2 = .836$, and a significant main effect of stimulus, F(1, 19) = 124.12, $p < .001$, $\eta^2 = .053$. There was no significant main effect of electrode, F(1, 19) = 0.01, $p = .914$, $\eta^2 < .001$. The interactions between listening condition and stimulus was significant, F(1, 19) = 68.72, $p < .001$, $\eta^2 = .042$ and between listening condition and electrode approached significance, F(1, 19) = 3.89, $p = .063$, $\eta^2 = .001$, while the stimulus and electrode interaction was not significant, F(1, 19) = 0.24, $p = .633$, $\eta^2 < .001$. Finally, the three-way interaction among listening condition, stimulus, and electrode was significant, F (1, 19) = 4.56, $p = .046$,

η^2 = .001. Analysis factors were followed up by conducting two-way ANOVAs separately for Electrode and Listening condition.

A two-way ANOVA was done to evaluate the interaction between Electrode and Stimuli for each Listening condition, separately. Results showed that the interaction between Electrode and Stimuli was not significant in quiet, F (1, 19) = 3.667, p = .071, η^2 = .043, and in babble noise F (1, 19) = 0.474, p =.499, η^2 = .001.

A two-way ANOVA was done to evaluate the interaction between Listening condition and Stimuli for each Electrode, separately. Results showed that the interaction between Listening condition and Stimuli was significant for both electrodes T7 F (1, 19) = 68.575, p <.001, η^2 = .051, and T8 F (1, 19) = 44.927, p < .001, η^2 = .034. The follow up tests showed there was significantly longer latency of Ta with speech sound /ta/, than speech sound /da/, only in babble noise for both electrodes T7 (at T7 (t(19) = -8.107, p = < .001, Cohen's d = -2.904) and T8 (t(19) = -10.600, p = < .001, Cohen's d = -2.812), not in quiet (at T7 (t(19) = .460, p = 1.000, Cohen's d = .042) and T8 (t (19) = -1.880, p = .453, Cohen's d = -0.400)) (Figure 4).

4.3.2.4. Tb, Latency

A repeated measures ANOVA revealed a significant main effect of listening condition, F (1, 19) = 1053.38, p < .001, η^2 = .666, a significant main effect of stimulus, F(1, 19) = 129.80, p < .001, η^2 = .067, and a significant main effect of electrode, F(1, 19) = 133.85, p < .001, η^2 = .035. A significant three-way interaction among listening condition, stimulus, and electrode was also observed, F(1, 19) = 44.20, p < .001, η^2 = .028. Given this interaction, follow-up two-way ANOVAs were conducted separately for electrode and listening condition.

A two-way ANOVA was done to evaluate the interaction between Electrode and Stimuli for each Listening condition, separately. Results showed that the interaction between Electrode

and Stimuli was not significant, in quiet F (1, 19) = 2.159, p =0.158, η^2 = .038, and in babble noise F (1, 19) = 7.519×10-4, p =0.978, η^2 = 1.066×10-6.

A two-way ANOVA was done to evaluate the interaction between Listening condition and Stimuli for each Electrode, separately. Results showed that the interaction between Listening condition and Stimuli was significant only at T8 F (1, 19) = 7.269, p =0.014, η^2 = .007 and not at electrode T7 F (1, 19) = 0.176, p =0.680, η^2 = 8.963×10-5. A follow up T-tests for electrode T8 showed a significantly longer latency of Tb with speech sound /ta/, than speech sound /da/, in babble noise (t (19) = -4.055, p = .004, Cohen's d = -1.305), not in quiet (t(19) = -0.957, p = 1.000, Cohen's d = -0.280), at T8 (Figure 4).

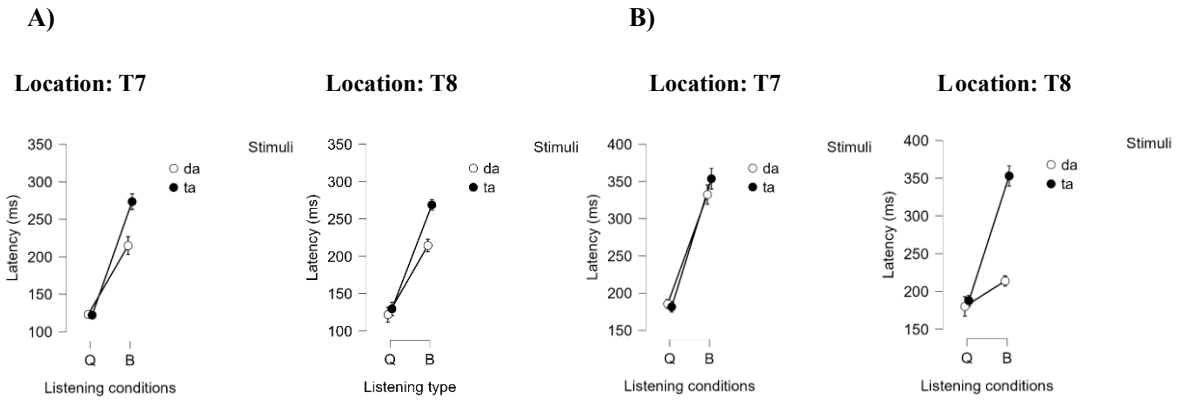

Figure 4. ***A)*** *The mean latency of Ta, to da and ta, in quiet and noise, at T7 and T8. There was longer latency of Ta in babble noise versus quiet, for both stimuli (/da/ and /ta/). Ta had longer latency to /ta/ versus /da/ in-babble noise.* ***B)*** *The mean latency of Tb, to /da/ and /ta/, in quiet and noise, at T7 and T8. There was longer of Tb in babble noise versus quiet, for both stimuli (/da/ and /ta/). Tb had significantly longer latency to /ta/ versus /da/ in-babble noise, significantly at T8.*

4.4. Discussion

This study aimed to evaluate the representation of long and short voice onset times using the T-complex, an auditory response originating from the superior temporal gyrus, in both quiet and babble noise across the right and left hemispheres. The findings demonstrated that the Tb component differentiated responses to speech sound /ta/ from speech sound /da/ in both quiet and babble noise conditions. In quiet, Tb amplitude was larger when evoked by /ta/ compared to /da/ at electrode T8. In contrast, in babble noise, Tb amplitude was larger for

speech sound /da/ than for /ta/ (Figure 5). The Ta component also showed larger amplitude in response to speech sound /da/ than to speech sound /ta/ in babble noise, with similar patterns observed across the right and left hemispheres. In quiet, both stimulus types drew out comparable Ta and Tb latencies (Figure 5). However, in the presence of babble noise, temporal response latencies were longer for speech sound /ta/ than for speech sound /da/ (Figure 5).

The following sections discuss each of these findings in more detail.

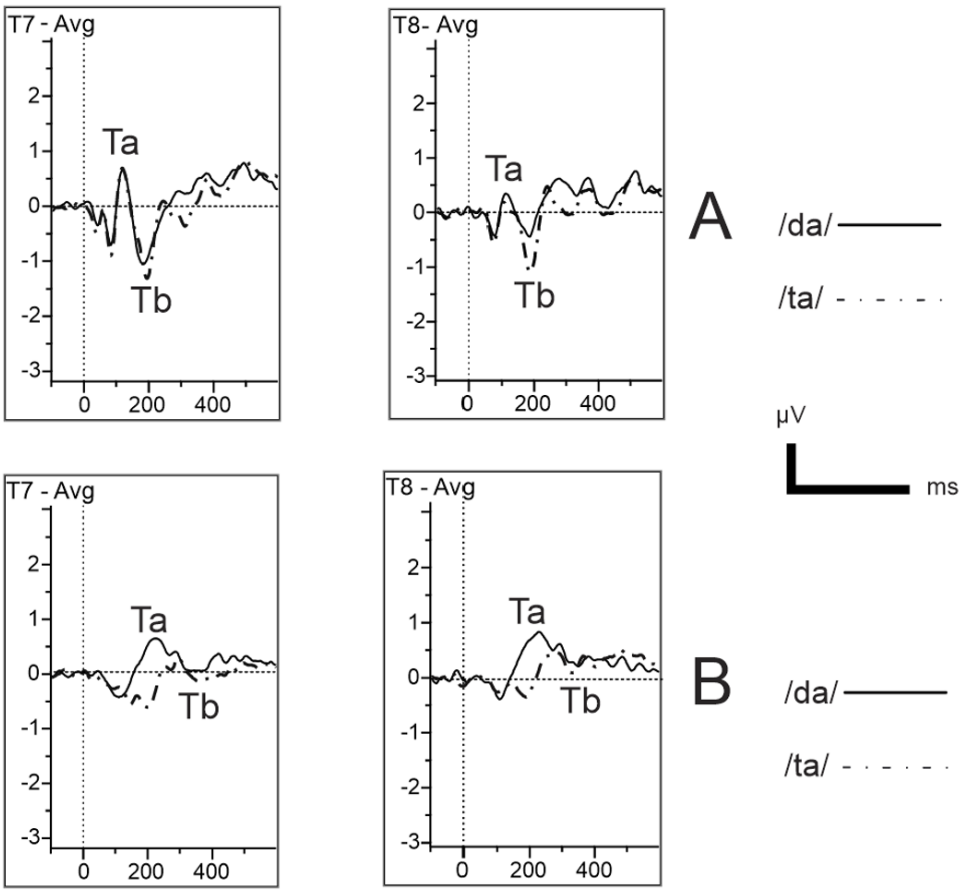

Figure 5. *The Ta and Tb responses to syllable /da/ and /ta/, in quiet (A) and in noise (B). Solid curve is response to /da/ and dashed curve is response to /ta/. In quiet, Tb showed larger response, at T8, to /ta/ than /da/. In noise, both Ta and Tb had longer latency and smaller amplitude to /ta/ than /da/.*

4.4.1. Difference between representation of /da/ and /ta/ in quiet

The results showed an effect of voice onset time on temporal auditory evoked potentials, with the Ta component exhibiting larger amplitudes in response to the short VOT syllable compared to the long VOT syllable across both the right and left hemispheres. This

finding is consistent with previous AEP studies demonstrating larger amplitudes for syllables with shorter temporal dynamics at onset compared to those with longer temporal changes (Kuruvilla-Mathew et al., 2015; Toscano et al., 2010).

In contrast to the Ta component, Tb amplitude was larger in response to the long VOT syllable compared to the short VOT syllable in the right hemisphere. In other words, a right-hemisphere advantage was observed for long VOTs, as responses to short VOTs were smaller at this site. Lateralization of speech feature processing has been reported in the literature (Jäncke et al., 2002; Shtyrov et al., 2000; Steinschneider et al., 1999; Tallal et al., 1993). It has been suggested that long and short voicing times are represented in the right and left hemispheres, respectively (Sandmann et al., 2007; Segalowitz & Cohen, 1989; Steinschneider et al., 1999). The results of the current study replicate a pattern of right-hemisphere advantage for syllables with long voice onset times.

The observed functional lateralization for long voice onset times may be explained by the Asymmetric Sampling in Time (AST) theory (Poeppel, 2003). This theory proposes that speech-sound lateralization is driven from hemispheric specialization in the time domain (Boemio et al., 2005; Poeppel, 2003). According to this model, auditory temporal processing is functionally asymmetric: neurons in the left hemisphere are for processing in short time scales and fast changing temporal information, whereas neurons in the right hemisphere are more sensitive to longer time scales and spectral aspects of speech, such as pitch and syllabic information, which are encoded over longer windows (150-300ms) (Luo & Poeppel, 2007; Zatorre & Belin, 2001; Zatorre et al., 1992). It has been suggested that microstructural anatomical differences between the right and left hemispheres may provide the neural substrates underlying these functional differences in spectral and temporal resolution (Klingberg et al., 2000).

Among studies reporting asymmetry in auditory processing of short and long VOTs (Giraud et al., 2008; Sandmann et al., 2007; Shtyrov et al., 2000; Trébuchon-Da Fonseca et al., 2005), none have examined the Ta and Tb components of the T-complex. The T-complex originates from the superior temporal gyrus, a brain region that plays a critical role in the neural coding of VOT (Bhaya-Grossman & Chang, 2022; Blumstein et al., 2005; Hutchison et al., 2008; Steinschneider et al., 1999). For instance, Steinschneider et al. (1999) recorded responses from Heschl's gyrus, the planum temporale, and the superior temporal gyrus and demonstrated that, among these auditory regions, the superior temporal gyrus showed distinct features in the neural representation of VOT.

To our knowledge, this study is the first to show auditory laterlazation in the neural coding of VOT as represented by the Tb component. Tb is the second component of T-complex originating from secondary auditory areas (Ponton et al., 2002; Wolpaw & Penry, 1975). Previous research has shown that Tb is sensitive to distinctions between short and long temporal intervals using paired-tone stimuli (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). In the study by Clunies-Ross et al. (2015), it was hyphothesized that electrophysiological responses to the second tone in a 50-ms tone pair would differ between the right and left hemispheres. It was predicted that a 50-ms tone pair would draw out two Tb components over the left hemisphere but only one Tb over the right hemisphere, reflecting integration of the second tone with the first in the right hemisphere. The results confirmed this hypothesis, showing a single Tb response over the right hemisphere and two Tb responses over the left hemisphere. Poeppel (2003) proposed that a larger proportion of neurons in the left hemisphere operate over short temporal integration windows, whereas a larger proportion of neurons in the right hemisphere operate over longer temporal integration windows. Accordingly, the observation of a single Tb responses to a 50-ms tone pair in the right hemisphere and two Tb responses in the left hemisphere aligns with the Asymmetric Sampling

in Time hypothesis and highlights the role of Tb generators in processing long and short temporal information (Poeppel, 2003). In the present study, the stronger Tb response in the right hemisphere to long VOTs compared to short VOTs may replicate hemispheric lateralization of temporal processing at the secondary auditory cortex and underscores the contribution of Tb neural generators in the representation of temporal dynamic in auditory stimuli.

It is worth mentioning that the hemispheric asymmetries in the current results were observed only for Tb, whereas Ta showed differentiation between speech sounds /da/ and /ta/ symmetrically in both the right and left hemispheres. This may highlight that generators of the T-complex components contribute to different levels of temporal processing of auditory stimuli, with Ta reflecting higher-level auditory processing (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). These findings support the results of Clunies-Ross et al. (2015), who reported that the neural orgins of Tb contribute to asymmetric temporal processing between the right and left hemispheres, while Ta showed symmetric responses to short and long temporal dynamics across hemispheres. Such results may shed a light on patterns of processing at higher levels of the auditory neural system and may be applied to understanding the underlying neurophysiological impairments in individuals with auditory processing difficulties. However, further research is warranted to confirm these findings.

4.4.2. Difference between representation of /da/ and /ta/ in noise

Noisy listening conditions could sigificantly affect the latency of both Ta and Tb and amplitude of Tb. For both stimuli, temporal responses showed longer latencies in noise than in quiet, which is consistent with previous findings from AEP studies conducted in noisy conditions (Anderson, Skoe, et al., 2010; Curtis J Billings et al., 2017; C. J. Billings et al., 2009).

Regaring amplitude, Tb responses showed smaller amplitude and/or less negativity for in noise than in quiet. According to the data, Tb amplitude in noise was largely suppressed, with the mean near 0 μV for /ta/ and around 0.7 μV (Figure 3). This suppression may not solely reflect a true reduction in neural activity, but may instead result from temporal overlap among ERP components (Luck, 2012; Luck, 2014). According to the ERP superposition principle, the observed waveform at any given time point represents the linear summation of multiple underlying neural processes that overlap in time (Luck, 2014). In the present data, Ta appears delayed and broader in noise, which would be expected to produce a similarly delayed and broadened Tb response. At the same time, a sustained positivity following Tb is present in both quiet and noise conditions, although with reduced amplitude in noise. If Tb is delayed into the time the window of this sustained positivity, the opposing polarities of these overlapping components may partially cancel each other in the averaged waveform. As a result, Tb may appear reduced, masked or even absent, despite the continued activity of its underlying neural generators. If this interpretation is correct, one would expect both a reduction in the late sustained positivity due to overlap with a delayed Tb and a reduction or apparent disappearance of Tb because it is subsumed within the positivity. Thus, the delayed Tb and the late sustained positivity may partially cancel each other out in the grand-average waveform. Given that this is the first study to examine the T-complex under noise conditions, further research using complementary approaches, such as source analysis and single-trial methods, will be necessary to determine whether the observed Tb suppression reflects true neural attenuation or component overlap effects.

In terms of stimulus effect, the current study showed increased amplitude and decreased latency of Ta and Tb in response to speech sound /da/ compared to speech sound /ta/ under noisy conditions. Similar findings have been reported for other cortical auditory evoked potentials, such as smaller N1 and P2 amplitudes obtained by syllables with a /t/ onset

compared to those with a /d/ onset in babble noise (Kuruvilla-Mathew et al., 2015). The observed differences in AEP measures in response to speech sounds /ta/ and /da/ may be attributed to acoustic features of these stimuli. Compared to /d/, /t/ contains more high-frequency at consonant onset and shows a slower rise time or reduced burst energy (Kuruvilla-Mathew et al., 2015). Voiced consonants typically have a short VOT and an onset burst with spectral energy similar to the fundamental frequencies (F0) of the following vowel (/a/), containing more periodic and low-frequency content. In contrast voiceless consonants have longer VOTs and minimal energy in the first formant (F1) frequency region prior to vowel onset (Kuruvilla-Mathew et al., 2015). The reduced onset energy of voiceless syllables likely makes them more vulnerable to babble noise, resulting in smaller amplitudes and longer latencies (Kuruvilla-Mathew et al., 2015). Additionally, greater resistance of periodic and lower frequency components of speech, compared to aperiodic segments, to babble noise has been reported previously (Curtis J Billings et al., 2017; Niemczak & Vander Werff, 2019; Ostroff et al., 1998). Auditory brainstem response studies using syllables in babble noise have also showed greater robustness of periodic F0 features in noise compared to formant transition components of syllables such as /da/ (Anderson, Skoe, et al., 2010; Cunningham et al., 2002; Russo et al., 2005). Similarly, cortical auditory evoked potential studies in babble noise have shown stronger responses to syllables beginning with the vowel /a/ compared to those beginning with consonants such as /s/ (Curtis J Billings et al., 2017; Ostroff et al., 1998). Together, these findings suggest that the more robust representation of vowels compared to consonants in noise may reflect adaptative neural encoding at the cortical level. In noisy environments, cortical neurons appear to adapt by synchronizing more strongly to low temporal modulation rates and periodic features of speech, such as vowels and voiced versus voiceless syllables (Ding & Simon, 2013).

The greater suppression of auditory responces evoked in response to speech sound /ta/ compared to speech sound /da/ in babble noise was also reflected in the behavioral results. Participants showed significantly longer reaction times and lower accuracy in detecting speech the sound /ta/ than the speech sound /da/ in babble noise.

In combination, this study showed the representation of long and short voicing onset time indexing objectively by T-complex in EEG recording and subjectively by accuracy and reaction time in behavioral evaluations. There was a rightward advantage to long VOT than short VOT, demonstrated by Tb, the late component of T-complex in quiet. These results revealed the lateralization of temporal processing at the level of secondary auditory areas. This finding may help to identify the impairments of auditory processing in populations with auditory processing difficulties.

While providing novel findings regarding the representation of short and long VOT by T-complex, this study has some limitations This study was conducted using a passive paradigm, adding an active experiment could bring more information regarding the temporal auditory processing in a real-life listening condition and when attention is involved. Also, this study used only one level of signal-to-noise ratio, it would be helpful to look at the modulation of representation of VOT in various levels of babble noise. This study investigated the T-complex only in adults with mature auditory systems and no history of neurological disorder. More research looking at various age population, and in the immature auditory system, will bring more information about the underlying processing of the temporal features of speech stimuli on populations with poor speech perception in babble noise, like APD.

4.5. Conflict of Interest Statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

4.6. Data accessibility statement

Data will be made available on request.

Reference

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Chapter 3. Manuscript 4

Representation of long and short voice onset time indexed by the T-complex in quiet and noise in normal-hearing children

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Highlights

- Short and long voice onset times (VOTs) modulate the morphology of the temporal auditory evoked potentials (T-complex: Ta and Tb) in children.
- There is a rightward lateralization in representation of the long VOT.
- The Tb component exhibited larger amplitudes in response to long VOTs than to short VOTs at electrode T8.
- Background noise suppressed auditory evoked potentials elicited by long-VOT stimuli to a greater extent than those elicited by short-VOT stimuli.

Abstract

Introduction: Voice onset time (VOT) is a temporal cue for differentiating voiced and voiceless consonants. Maintaining synchronized auditory neural responses to VOT is critical for successful speech perception. So far, no study has evaluated the representation of VOT using the T-complex, i.e., temporal auditory evoked potentials recorded at T7 and T8 (Ta and Tb components), particularly in noise. This study aimed to evaluate the modulation of the T-complex in children, in response to short and long VOTs in quiet and in noise.

Method: Fifteen typically developing children from the community of Ottawa (8-12 years old) participated in this study. Temporal auditory evoked potentials in response to speech sounds /ta/ and /da/ syllables were recorded at electrodes T7 and T8 in both quiet and babble noise (signal-to-noise ratio = 5dB). In addition, behavioral measurements, including accuracy and reaction time during a task discriminating between /ta/ and /da/, were collected in both quiet and noisy conditions.

Results: Reaction time was longer for /ta/ compared to /da/. In quiet, there was a larger amplitude of Ta for /da/ than /ta/, whereas Tb showed larger amplitude for /ta/ than for /da/ at electrode T8, in quiet. The latency of Ta was prolonged for /ta/ relative to /da/ in noise.

Discussion: These results indicate lateralization of temporal processing at the level of secondary auditory areas in children. This finding may be promising for characterizing impairments in temporal processing in populations with temporal processing difficulties at higher levels of the central auditory nervous system.

Keywords: T-complex, Temporal processing, Voice onset time, Speech in noise, Lateralization, Reaction time

5.1. Introduction

Effective listening skills and understanding of speech in noisy conditions are critical abilities for successful communication (Eranovic et al., 2022; Neuman et al., 2010; O'Hara & Mealings, 2018; Prodi & Visentin, 2022). These abilities are even more crucial for young students as over 60% of elementary classroom time is spent "learning by listening" (Hunsaker, 1983). Impairments in speech-in-noise understanding may hinder language development as well as social and academic functioning resulting in long-term consequences for learning and future life (Canale et al., 2014; Cestnick & Jerger, 2000; Del Zoppo et al., 2015). Understanding the effects of noise on speech features and perception may assist in identifying children who experience difficulties understanding speech in noise.

Background noise disrupts the representation of timing aspects of speech elements such as consonants and vowels (Anderson, Skoe, et al., 2010; Jenna Cunningham et al., 2001; Patro et al., 2025; Spoorthi et al., 2025). Vowels are primarily produced by vibrating vocal folds, resulting in high energy, and regular or repeating steady-state waveforms, while many consonants are generated by turbulent airflow or closures, creating low energy random or aperiodic acoustic patterns (Cullinan & Tekieli, 1979; Podesva & Callier, 2015). The most susceptible parts of the speech masked by background noise are those with low intensity or aperiodic features (e.g. consonants) (Balaban & Yaralı, 2025; Yan et al., 2025). Multiple studies have shown that background noise alter the acoustical cues associated with consonants more significantly than vowels, making the identification of consonants more challenging in noise (Parikh & Loizou, 2005; Warrier et al., 2004; Yan et al., 2025). Since consonants convey most of the information necessary for understanding word meaning, accurate neural encoding of the acoustical cues that give rise to consonant perception is important for effective speech understanding in noisy listening conditions (Balaban & Yaralı, 2025; Warrier et al., 2004).

In natural speech, voice onset time (VOT) functions as a critical acoustical cue for differentiation and identification of voiced from voiceless stop consonants such as /d/ and /t/. (Brinca et al., 2016; Whiteside et al., 2003). The accurate perception of VOT depends on the precise temporal alignment of neural activity with the onset of voicing (Sinex & McDonald, 1989; Sinex et al., 1991). Difficulties in the identification of VOT have been reported in clinical populations (Breier et al., 2001; Gooch et al., 2011; Götz et al., 2025; Hestvik et al., 2023; Noordenbos & Serniclaes, 2015), who have a poor understanding speech in noisy conditions (Almeqbel & McMahon, 2015; Anderson, Skoe, et al., 2010; Bradlow et al., 2003; O'Hara & Mealings, 2018; Wible et al., 2002). Studies have suggested deficits in temporal processing as

a reason for the difficulties in understanding speech in noise in these populations (Almeqbel & McMahon, 2015; Talcott et al., 2000; Warrier et al., 2004). Early detection and diagnosis of auditory temporal processing deficits are important to ensure early treatment in children with impaired temporal processing abilities. One of the primary limitations in detecting temporal processing deficits in infants and young children is their inability to provide reliable behavioral responses to tasks designed to assess temporal processing (Dillon & Cameron, 2021b). Also, the behavioral responses usually are influenced by non-auditory factors like attention and language skills as well (Dillon & Cameron, 2021b). One way to eliminate the non-auditory factors is to use Auditory Evoked Potentials (AEPs) to objectively assess the sensitivity to temporal changes at the level of auditory cortex (Almeqbel & McMahon, 2015; Michalewski et al., 2005).

AEPs are neural responses recorded from surface electrodes placed on the scalp and generated in the auditory cortex (Luck, 2012). Cortical AEPs, based on the scalp sites, include two categories: frontocentral and temporal responses (Pang & Taylor, 2000; Ponton et al., 2002; Ponton et al., 2000; Wolpaw & Penry, 1975). Frontocentral cortical evoked potentials (P1, N1, P2, N2) which mainly arise from the primary and secondary auditory cortices (Sharma & Dorman, 1999; Sharma et al., 2000). Temporal cortical evoked potentials or T-complex, including Na-Ta-Tb, is another auditory cortical response, originating from the superior temporal gyrus (Pang & Taylor, 2000; Ponton et al., 2002; Ponton et al., 2000; Wolpaw & Penry, 1975). T-complex and frontocentral responses differ in terms of origins and developmental trajectory. More specifically, the T-complex arises from the secondary auditory cortex in the posterior and lateral parts of the superior temporal gyrus (Brodman areas 42 and 22), indexing the neural processing in belt and para-belt areas of auditory cortex (Howard et al., 2000; Scherg & Von Cramon, 1986; Wolpaw & Penry, 1975). Frontocentral responses mainly originate from the primary auditory areas in Heschl's gyrus (Brodmann 41), in temporal plane (Liegeois-Chauvel et al., 1991; Wolpaw & Penry, 1975).

AEPs have been extensively used in search of neurophysiology underlying temporal processing correlates of voicing perception (Almeqbel & McMahon, 2015; Hestvik et al., 2023; Hoonhorst et al., 2009; Martin & Boothroyd, 1999; Sharma & Dorman, 1999; Tremblay et al., 2003). The majority of AEPs studies evaluating the impact of timing features of speech, like VOTs, have explored the frontocentral responses (Almeqbel & McMahon, 2015; J.-H. Han et al., 2020; Katrina Agung King et al., 2008). In quiet condition, the study of King et al.,2008 showed that P1 and N2 had longer latency to speech sound /ta/ containing longer VOT, than

speech sound /da/. In study of Han et al., 2020, the latency of P2 and N2 were recorded to speech sound /a/ with short VOT (0ms) and long VOT (50ms), in quiet and in noise. Results in quiet conditions showed that there was longer latency to stimuli with long VOT than short VOTs, also in noisy conditons, the latency of the N2 and P2 to longer VOT (50ms) was more prolonged than in response to speech sound with short VOT (0ms) (Almeqbel & McMahon, 2015; J. H. Han et al., 2020). These finding highlighted the more detrimental effects of noise on stimuli with long duration of VOT than short duration of VOT (Almeqbel & McMahon, 2015; J. H. Han et al., 2020).

The effects of VOT on temporal auditory responses in children have been little explored. To the best of our knowledge, only one study (Hoonhorst et al., 2009) has investigated the representation of long and short VOT on the T-complex in quiet conditions and in adults. In this study, the Na was recorded in response to syllables with onset /t/ and /d/ in quiet (Hoonhorst et al., 2009). Results showed that the Na, was double-peaked for long VOT values, while there was one peak for short VOTs (Hoonhorst et al., 2009). To our knowledge this study was the only study reporting representation of VOT by T-complex and has also reported only Na. So, little is known about the Ta and Tb components, in response to long and short VOT, in quiet and in noise in children. Then, more research is needed to evaluate the effect of long and short VOTs on Ta and Tb, especially in noisy conditions. Moreover, T-complex's generators seem to contribute to the representation of temporal dynamics at the onset of syllables (Belin et al., 2002; Belin et al., 2000; Rupp et al., 2022). Neuroimaging research has shown that the superior temporal gyrus, the primary generator of the T-complex, exhibits greater activation in response to voiced compared to voiceless speech sounds (Belin et al., 2002; Belin et al., 2000; Rupp et al., 2022). Given the involvement of the T-complex generators in encoding voice onset time (VOT), examining T-complex responses to syllables with different VOTs may provide valuable insights into how the superior temporal gyrus processes the temporal features of speech. This approach may improve identification and characterization of speech perception difficulties in young children who struggle to understand speech in noisy environments, at the level of secondary auditory cortex.

In order to assess potential deficits in processing of VOT at higher levels of the auditory system indexed by T-complex, it is critical to first collect data on typically developing children. Therefore, this study aimed to investigate the cortical temporal auditory responses in quiet and noise, in a group of typically developing children.

In the current study, both electrophysiological responses and behavioral responses to speech sound /da/ and speech sound /ta/, in quiet and with background noise were recorded. Given the literature and contribution of T-complex generators in encoding the VOTs, it was expected that the T-complex would vary as a function of VOTs. Moreover, it was expected that background noise will reduce the amplitude and increase the latency of the T-complex.

5.2. Method

5.2.1. Ethics approval

The University of Ottawa Research Ethics Board approved this study, Ethic file number=H-07-24-10451. All participants received and signed a copy of the written consent form.

5.2.2. Participants

Fifteen children between 8-12 years old (Mean=10.438, SD=1.315, Male:7) recruited from Ottawa's community participated in this study.

5.2.3. Inclusion and exclusion criteria

All participants had normal hearing detection thresholds at 15 dB HL or less in octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996)'s protocol. Tympanograms were normal (admittance curve with a single peak between +50 to -100daPa using a 226-Hz probe tone). Participants were right-handed and fluent in English for both speaking and listening, as determined by the Language Experience and Proficiency Questionnaire (LEAP-Q) (Marian et al., 2007), as the Babble noise used in this study was in English (Marian et al., 2007).

No history of otological or neurological disorders were reported for any of the children participating in the study. The Auditory processing domain questionnaire (APDQ) was also used to evaluate the auditory processing, attention and language skills. The APDQ is a screening tool, developed in 2018, with 70-85% accuracy and evaluates auditory processing, attention and language skill's of individual between 7-18 years old and can differentiate between disorders with overlapping symptoms including auditory processing disorder (APD), language disorder and attention disorder/hyper activity (ADHD) (O'Hara & Mealings, 2018). The BKB-SIN (Bamford-Kowal-Bench Speech-in-Noise) was also conducted to make sure participants did not have listening difficulties in noise. All included participants had normal performance in BKB (signal-to-noise ratio loss was 0-3dB) and the score of the APDQ were in normal range (>70%). Also, all children had normal cognitive abilities, using the auditory

Continuous Performance Test (ACPT). The ACPT is a cognitive test designed to measure a person's sustained attention and vigilance, particularly in an auditory context (Keith, 1994; Riccio et al., 1996). Participants had minimal musical training, as previous work has shown that music practice can enhance the understanding of speech in noise and enhanced AEPs in the presence of background noise (Anderson et al., 2013; Benjamin Rich Zendel et al., 2015). None of the participants had musical training. All participants consented to participate in this study. Table 1 demonstrated the results of APDQ, BKB and ACPT test.

Table 1. *The results of APDQ, BKB and ACPT tests.*

	Age	AP	LA	ATT	BKB	ACPT-Commission errors 2	ACPT-Omission errors
Valid	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0
Mean	10.881	86.813	90.438	83.000	1.919	3.063	1.688
Std. Deviation	1.270	11.600	10.844	10.545	0.732	1.436	0.704
Minimum	8.700	57.000	71.000	53.000	0.800	1.000	1.000
Maximum	12.900	99.000	100.000	97.000	3.000	5.000	3.000

AP=Auditory processing, ATT=Attention, ACPT=Auditory Continuous Performance Test, BKB=Bamford-Kowal-Bench Speech-in-Noise, LA=Language.

5.2.4. Procedure

During the experiment, after familiarization with the testing equipment and procedure, the participants were seated in a sound-attenuated testing room, where they completed the experiment. Prior to testing, the purpose of the study was explained to each participant and informed consent was obtained from the children and their parents. Participants went through behavioral test and electrophysiological recording. The behavioral test was a discriminating task, and the electrophysiological recording included a passive auditory paradigm. The details of experiments are as follows.

5.2.5. Stimuli

Stimuli were speech stimuli including syllables /da/ and /ta/ with a duration of 250ms (Dubno & Schaefer, 1992). These stimuli were natural, recorded with a male voice and used in the study of Dubno & Schaefer in 1992. The syllables /da/ and /ta/ have similar articulation place (Digeser et al., 2009) and contain similar formant frequencies (Figure 1and 2). They differ mainly in consonant voicing which is cued by voice onset time (VOT), speech sound /da/ has a fast temporal dynamic at the onset of the syllables (short voicing time, around 20ms) and speech sound /ta/ has a slow temporal dynamic at the onset of the syllables (long voicing time,

around 100ms). In the current manuscript, the /ta/ is referred to speech sound and Ta is referred to the positive component of T-complex.

Stimuli were presented using E-prime software via insert earphones (EARTone 3A) in two listening conditions: quiet and Babble noise. The Babble noise was an English-four-talker masker previously developed and employed by Zendel et al, 2015 (Benjamin Rich Zendel et al., 2015). Stimuli were equalized for root mean square (rms) amplitude and then played at 80 dB SPL. For noisy conditions, signal-to-noise ratio (SNR) was kept as +5dB, by reducing the intensity of noise to 75dB SPL. Interstimulus interval (ISI) was jittered randomly between 1100-1250ms.

5.2.6. Behavioral measurements

Behavioral task included two blocks corresponding to quiet and Babble noise conditions, with the babble noise presented at SNR of +5 dB. Each block included 75 presentations of /ta/ and 75 presentations of /da/. Stimulus order within each block was randomized, and block order was also randomized across participants. After each trial, participants indicated whether they perceived /ta/ or /da/ by pressing a button on a response box. Accuracy was calculated based on the number of hits (i.e., /da/ presented and correctly identified as /da/) minus misses (/da/ presented but identified as /ta/). Reaction time was also measured. Accuracy and Reaction time for /da/ were compared with those for /ta/.

5.2.7. Electrophysiological recording

The EEG data were recorded during a passive listening paradigm. Participants were instructed to ignore the auditory stimuli and direct their attention to a silent, subtitled movie. Stimuli were delivered in two blocks (quiet and noise), each block included 150 presentations of /da/ and 150 presentations of /ta/. Electrical responses were recorded using 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). Electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp according to the international 10-20 standard system. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were monitored using an electrode placed on the infra-orbital ridge of the left eye. The tip of the nose served as an online reference for all channels, including the electro-oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for offline analyses.

5.2.8. Electrophysiological data analysis

EEG data were analysed using Brain Products' Analyzer2 software. Continuous data were digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as an off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts were corrected using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1996). Vertical EOG activity was computed by subtracting the inferior orbital electrode from the FP2 channel and horizontal eye movements were computed by subtracting the FT9 from the FT10 channel (Duda-Milloy et al., 2019). ICA assumes that the recoded EEG signals represent a linear mixture of statistically independent neural and non-neural sources. ICA assumes that the recorded EEG signals reflect a linear mixture of neural and non-neural sources. Using blind source separation, the algorithm was trained to identify each participant's ocular artifact components, which were subsequently removed from the EEG data. Following ICA correction, the data were visually inspected to confirm adequate artifact rejection.

EEG epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset. Epochs containing amplitudes exceeding ±100 μV at any electrode were rejected, resulting in the exclusion of fewer than 10% of trials. Artifact-free epochs were averaged for each participant and subsequently across participants to generate grand-average waveforms for each voice onset time (VOT) condition, time-locked to the onset of /da/ (short VOT) and /ta/ (long VOT). All averaged waveforms were baseline corrected using the 100ms pre-stimulus interval. The T-complex was extracted from the temporal electrodes sites T7 and T8 (Wolpaw & Penry, 1975). These sites are considered optimal for measuring the T-complex as they reflect minimal contribution from the neural generators underlying the P1, N1 and P2 responses (Tonnquist-Uhlen et al., 2003).

Peak detection was performed for each participant to measure responses latency. Ta was defined as the most positive deflection occurring between 80 and 150ms, and Tb was defined as the most negative deflection following Ta, occurring between 150 and 250ms. Mean amplitudes were calculated with in a 50ms window centered on each peak (±25ms). These time windows were determined based on the grand average AEPs waveforms at the T7 and T8 electrode sites (J. A. Hämäläinen et al., 2011). The latency intervals used for mean amplitude measurements are reported in Table 2. Mean amplitude, rather than peak amplitude, was used because peaks are more susceptible to noise (Luck, 2012). As the Na wave was not reliably identified in most participants, only Ta and Tb are reported in the results. Additionally, Tb was

not identifiable in the babble noise condition, so this condition was excluded from Tb amplitude and latency analyses.

Table 2. *Latency intervals over which each of the mean amplitudes were calculated.*

Peak-site	listening condition- Stimuli			
	Quiet-/ta/	**Quiet-/da/**	**Babble noise-/ta/**	**Babble noise -/da/**
Ta-T7	134(119-169)	138(123-173)	310(285-335)	256(231-281)
Tb-T7	196(171-221)	194(169-219)	Not identifiable	Not identifiable
Ta-T8	132(107-157)	138(123-173)	302(277-327)	252(227-277)
Tb-T8	196(171-221)	196(171-221)	Not identifiable	Not identifiable

5.2.9. Statistical analysis

Statistical analyses were conducted using JASP software (version 0.19.1.0; Jeffreys's Amazing Statistics Program). A repeated-measures analysis of variance (ANOVA) was used to examine differences in AEPs amplitude and latency. Prior to conducting the ANOVAs, all data were inspected to ensure compliance with statistical assumptions. Normality of residuals was evaluated using Q–Q plots generated in JASP, which indicated approximately normal distributions, as data points closely aligned with the diagonal reference line. When Mauchly's test indicated a violation of sphericity, Greenhouse–Geisser corrections were automatically applied to adjust the degrees of freedom.

Separate repeated-measures ANOVAs were performed for each AEPs component, with amplitude and latency analyzed independently. For the Ta and Tb responses, three-factor repeated-measures ANOVAs were conducted separately. The within-subjects factors included Listening conditions (Quiet, Babble noise), Stimulus (/da/, /ta/), and Electrode Location (T7, T8). This design enabled the assessment of main effects and interactions among Listening conditions, Stimuli, and Location on amplitude and latency of Ta and Tb.

Regarding behavioral responses, two repeated measurements ANOVA were conducted separately for accuracy and reaction time. The within-subjects factors included Listening Condition (Quiet, Babble noise) and Stimulus (/da/, /ta/). To examine the relationship between cortical auditory responses and behavioral performance, Pearson correlations analyses were conducted between Ta and Tb peaks (latency and amplitude) and speech perception measures. To account for differing measurement scales, all variables were z-scored across participants prior to analysis. Effect sizes were indexed using the correlation coefficient (r or ρ), with r^2 reported as the proportion of variance explained. Given the modest sample size, effect sizes

and the direction of associations were interpreted alongside p-values to characterize the strength of AEPs and behavior relationships, particularly in cases where findings trended toward significance.

5.3. Results

5.3.1. Behavioral results

Table 3 and 4 showed the significant effects of Listening conditions on accuracy (*F (1, 14) = 18.346, p < .001, η^2= 0.294)*. However, there was no significant effect of Stimulus (*F (1, 14) = 0.019, p =0.892, η^2= 5.364×10^{-4})* or interaction between factors (*F (1, 14) = 0.175, p < .682, η^2= 0.001)*. The effect of listening conditions was confirmed with a follow up test showing lower accuracy in noise than in quiet for both stimuli (*p<0.001*). For reaction time (Table 3), there was a significant effect of Listening condition (*F (1, 14) = 75.081, p < .001, η^2= 0.692)*, and significant effects of Stimulus (*F (1, 14) = 62.196, p < .001, η^2= 0.121),* but the interaction between factors was not significant (*F (1, 14) = 1.644, p < .221, η^2= 0.003)*. Follow up test showed longer reaction time in noise than when in quiet (*p< .001*). Also, there was longer reaction time in response to speech sound /ta/ than speech sound /da/, in both listening conditions (*p< .001*).

Correlation analyses did not reveal any statistically significant associations between Ta and Tb measurements and behavioral performance (all p > .05) (Tables have been inserted in Appendix). However, effect size estimates indicated a relatively moderate to strong negative association between Ta latency at electrode T7 for the speech sound /ta/ and speech perception accuracy (r = −0.54) when it was recorded in the quiet condition. A similar negative association was observed between Tb latency at electrode T7 for the speech sound /ta/ and speech perception accuracy (r = −0.43) when it was recorded in the quiet condition, indicating that longer cortical response latencies were associated with lower accuracy.

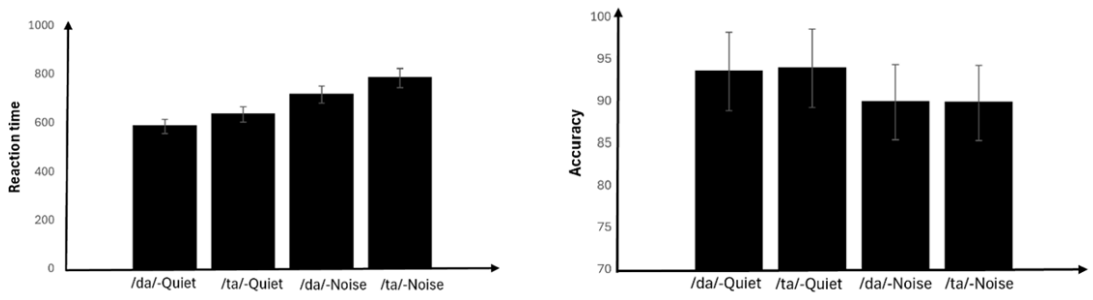

Figure 1. *The average of reaction time (RT) and accuracy (ACC) to /da/ and /ta/, in quiet (Q) and in noise (B). Reaction time was impacted by Stimuli; RT was longer to speech sound /ta/ than to speech sound /da/. There was a longer reaction time in noise, versus quiet for both speech sounds, also, there was less accuracy in noise than quiet for both stimuli.*

Table 3. *The results of ANOVA to assess the effect of Listening conditions (Quiet and Noise) and Stimuli (/da/ and /ta/) on Accuracy.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	228.150	1	228.150	18.346	< .001	0.294
Residuals	174.100	14	12.436			
Stimuli	0.417	1	0.417	0.019	0.892	5.364×10^{-4}
Residuals	307.833	14	21.988			
Listening condition ✻ Stimuli	0.817	1	0.817	0.175	0.682	0.001
Residuals	65.433	14	4.674			

Note. Type III Sum of Squares

Table 4. *The results of ANOVA to assess the effect of Listening conditions (Quiet and Noise) and Stimuli (/da/ and /ta/) on Reaction time.*

Cases	Sum of Squares	df	Mean Square	F	p	η^2
Listening condition	297334.401	1	297334.401	75.081	< .001	0.692
Residuals	55442.690	14	3960.192			
Stimuli	51844.549	1	51844.549	62.169	< .001	0.121
Residuals	11675.047	14	833.932			
Listening condition ✻ Stimuli	1421.748	1	1421.748	1.644	0.221	0.003
Residuals	12104.789	14	864.628			

Note. Type III Sum of Squares

5.3.2. Electrophysiological results

5.3.2.1. Amplitude, Ta

A repeated measures ANOVA showed no significant effect of Listening condition (*F (1, 14) = 2.423, p = 0.142, η^2= 0.046*, but a significant effect of Stimulus (*F (1, 14) = 37.312, p < .001, η^2= 0.162*) and marginally significant effects of Location (*F (1,14) = 3.606, p=.078, η^2=0.039*). There was marginally significant interaction between Listening condition and Stimulus (*F (1,14) =3.726, p = .074, η^2=0.018*), and a significant interaction between Stimulus and Location (*F (1,14) =5.629, p=.033, η^2=0.016*), but no significant interaction between

Listening condition and Location (*F (1,14) =0.152, p = .702, η²=0.001*). Follow up t-tests with a Bonferroni correction were calculated to investigate the source of interactions. For the Stimuli by Location interaction, the amplitude of Ta was larger to speech sound /ta/ than to the speech sound /da/ at both sites, and the impact of Stimulus (VOT) was larger at electrode T8 (*p < .001*) compared to T7 (*p=.03*). Regarding the Stimuli by Listening conditions interaction, the Stimuli effect was significant in quiet (*p< .004*), and not in noise (*p= .089*)

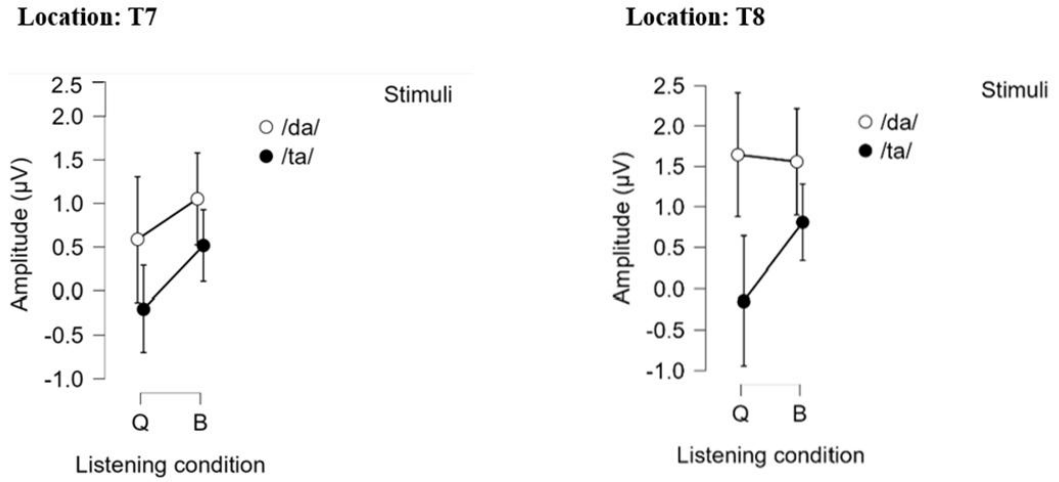

Figure 9 *The mean amplitude of Ta, in response to speech sounds /da/ and /ta/, in quiet and noise, at electrodes T7 and T8. Ta had larger amplitude to speech sound /da/ than speech sound /ta/, in quiet condition, especially at electrode T8. There was no difference in amplitude of Ta between speech sound /da/ and speech sound /ta/ in noise.*

5.3.2.2. Amplitude, Tb

A repeated measures ANOVA showed a significant effect of Stimulus (*F (1, 14) = 13.178, p = 0.003, η²= 0.155*) but no significant effects of Location (*F (1,14) = 0.015, p = .905, η²*=6.026×10^{-4}). The interactions between Stimulus and Location were significant (*F (1, 14) = 9.692, p = .008, η²= 0.047*). Follow up test confirmed this interaction and showed a larger amplitude of Tb to speech sound /ta/ than speech sound /da/ at electrode T8 (Figure 3) ((*t* (14) = -5.163, *p* < .001, Cohen's *d* = -0.970)) not at electrode T7 (*t* (14) = 1.097, *p* = 1.000, Cohen's *d* = 0.383) (Figure 3). As the Tb was not detectable in babble noise, the factor of listening conditions has not been calculated.

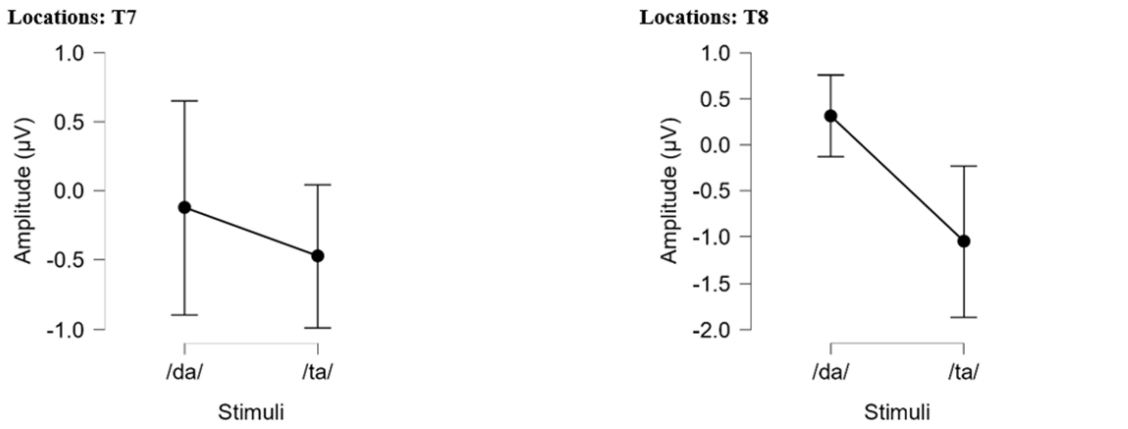

Figure 10 *The mean amplitude of Tb in response to speech sound /da/ and speech sound /ta/, in quiet, at electrodes T7 and T8. Tb had larger amplitude to speech sounds /ta/ than /da/ at electrode T8.*

5.3.2.3. Latency, Ta

A repeated measures ANOVA showed a significant effect of Listening condition (*F (1, 14) = 1011.875, p < .001, η^2= 0.851*), significant effect of Stimulus (*F (1, 14) = 41.024, p = <.001, η^2= 0.042*) and no significant effect of Location (T7 vs. T8) (*F (1, 14) =1.595, p < .227, η^2= 0.001*). There were no significant interactions between three factors, but the interaction between Listening conditions and Stimulus was significant (*F (1, 14)* = 43.157*, p < .001, η^2= 0.037*). The follow-up tests also showed there was no significant difference between the latency of Ta in response to speech sounds /da/ and /ta/ in quiet (*p = .145*), but the difference was significant in noise (*p < .001*). Ta had longer latency to speech sound /ta/ than speech sound /da/ in noise (Figure 4).

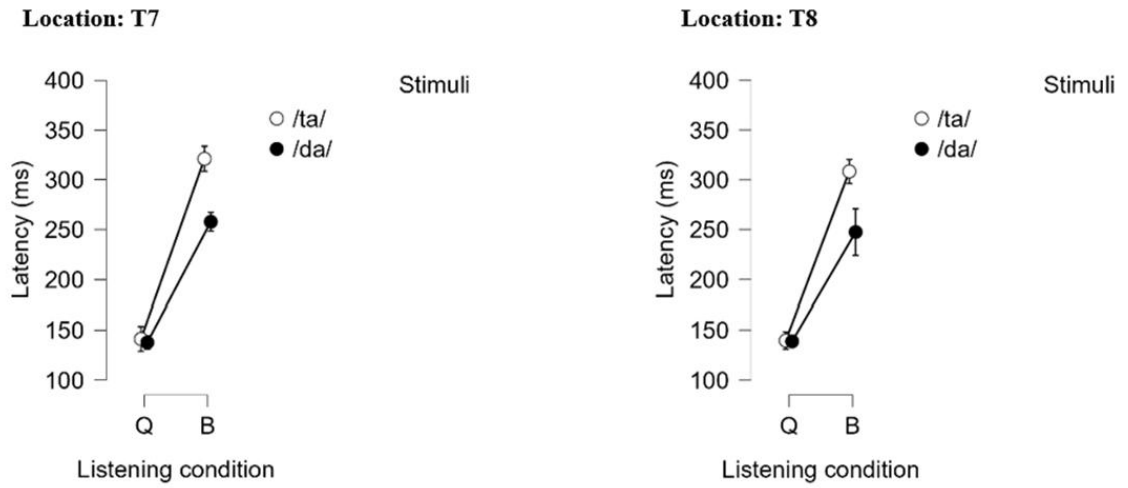

Figure 4. *The mean latency of Ta in response to speech sounds /da/ and /ta/, in quiet and noise, at electrodes T7 and T8. There was a longer latency of Ta when it was evoked to speech sound /ta/ than speech sound /da/, in noisy conditions.*

5.3.2.4. Latency, Tb

Repeated measures ANOVA showed no significant effects of Stimulus ($F\ (1, 14) = 5.386\times10^{-4}$, $p = .982$, $\eta^2 = 9.235\times10^{-6}$), and no significant effect of Location ($F\ (1, 14) = 0.016$, $p = .900$, $\eta^2 = 4.525\times10^{-4}$). The interaction between Stimuli and Location was not significant ($F\ (1, 14) = 0.017$, $p = .898$, $\eta^2 = 4.525\times10^{-4}$) (Figure 5). As the Tb was not detectable in babble noise, the factor of listening conditions has not been calculated.

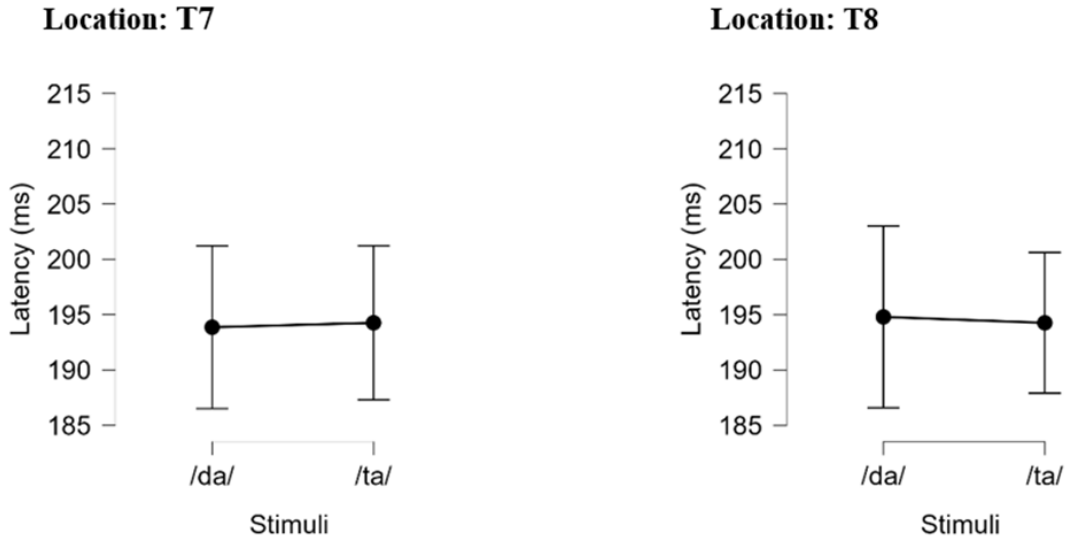

Figure 5. *The mean latency of Tb, in response to speech sounds /da/ and /ta/ at electrodes T7 and T8. There was no difference in latency of Tb between speech sound /da/ and speech sound /ta/.*

5.4. Discussion

This study aimed to evaluate the representation of long and short VOT using the T-complex, an auditory response originating from the superior temporal gyrus, in normal-hearing children. In addition, behavioral measures of discrimination performance-including accuracy and reaction time for short and long VOTs- were assessed.

Reaction time was impacted by stimulus type, with longer response time for /ta/ compared to /da/. Consistent with this behavioral finding, Ta latency was prolonged for the speech sound /ta/ relative to /da/ when stimuli were presented in noise. Additionally, both T-complex components, Ta, in both quiet and noise conditions, and Tb in quiet conditions, differentiated /ta/ from /da/, in both quiet and noise conditions based on amplitude measures. In quiet, there was larger amplitude of Ta amplitudes were larger for speech sound /da/ than /ta/, whereas Tb showed larger amplitude for /ta/ than for /da/ at electrode T8. These findings are explored in greater detail in the following sections.

5.4.1. *Difference between representation of /da/ and /ta/ in quiet*

As presented in Figures 2, 3 and 6, the findings revealed distinct effects of VOT on temporal auditory evoked potentials. Specifically, Ta amplitudes were larger in response to short VOT in both hemispheres, whereas, Tb amplitudes were larger for long VOTs, with a stronger effect observed in the right hemisphere.

The Tb results showed a right-hemispheric lateralization for long VOT, as responses to short VOT were comparatively smaller (Figure 6). Hemispheric asymmetries in the encoding of long and short voicing times have been widely reported, with larger right-hemisphere responses to long VOTs and larger left-hemisphere responses to short VOTs in adults (Sandmann et al., 2007; Segalowitz & Cohen, 1989; Steinschneider et al., 1999). Consistent with this literature, the result of the current study replicates the pattern of a right hemisphere advantage for the long VOT syllables in children.

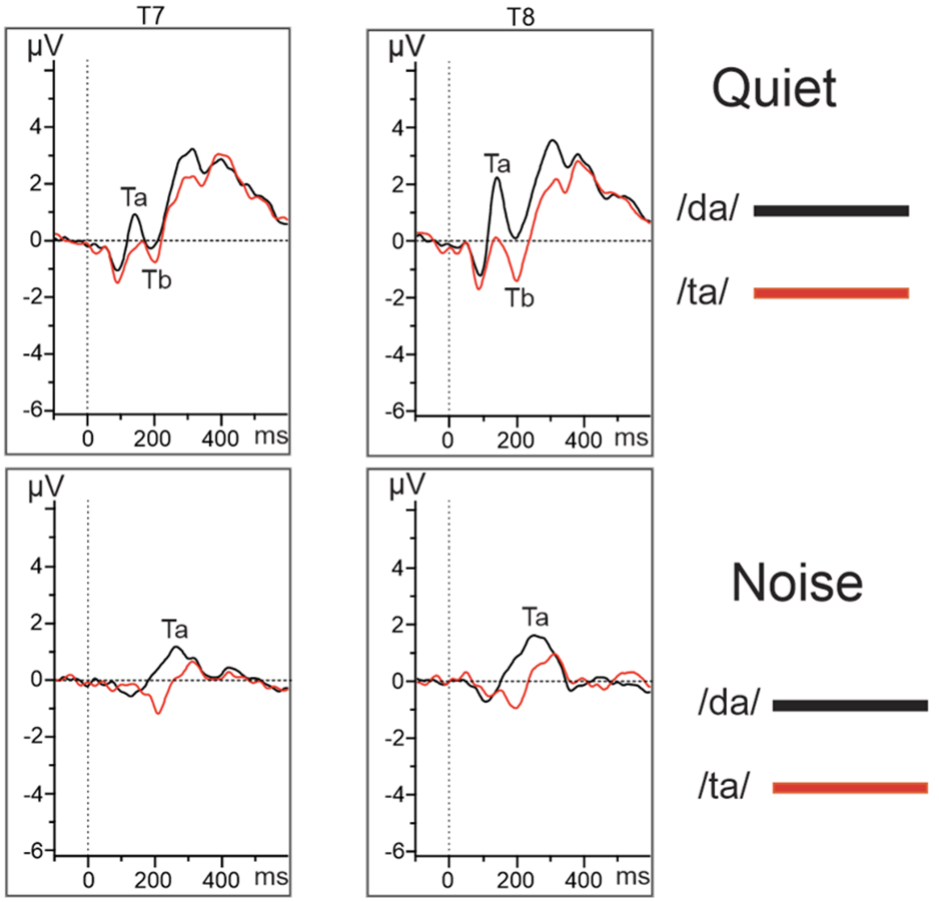

Figure 6. *The waveforms of Ta and Tb responses to speech sounds /da/ and /ta/, in quiet (upper panel) and in noise (lower panel). Black curve is T-complex response to speech sound /da/ and red curve is T-complex response to /speech sound ta/. In quiet condition, Tb showed larger response, at electrode T8 when it was evoked by speech sound /ta/ than speech sound /da/. Tb seems to be disappeared in noise. Ta showed larger response when it was evoked by speech sound /da/ than when it was*

evoked by speech sound /ta/, in quiet condition. In noise, Ta had shorter latency and smaller amplitude to speech sound /ta/ than speech sound /da/.

Tb, the second component of the T-complex, is thought to originate from secondary auditory areas (Ponton et al., 2002; Wolpaw & Penry, 1975). Previous studies using paired-tone paradigms in both adults and children have shown that Tb indexes fast and slow temporal processing (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). In particular, Clunies-Ross et al., (2018), recorded T-complex responses to tone-pairs with interstimulus intervals of 25, 50, 100 and 200 ms in children between 7-9 years old reporting larger Tb amplitudes in the right hemisphere for longer intervals (100 and 200 ms) and in the left hemisphere for shorter intervals (25 and 50 ms). These finding suggest the presence of hemispheric preference for slow and fast temporal modulation early in development and are consistent with reports of temporal processing asymmetries in young children (Thompson et al., 2016). Thompson et al., (2016) reported a lateralization of high-frequency (20–50 Hz) endogenous cortical oscillations, even in younger children aged 3–5 years. Presumably, it is possible to observe hemispheric differences in response to short and long duration VOTs with Tb in the children aged between 8-12 years, as in the current study. These findings support the contribution of Tb's neural generators in the representation of the temporal dynamics of auditory stimuli at the level of secondary auditory cortex in children.

In contrast, Ta amplitudes were larger when evoked by the speech sound /da/ compared to the speech sound /ta/. This finding was consistent with previous studies (Digeser et al., 2009; Kuruvilla-Mathew et al., 2015; Toscano et al., 2010) showing that low-frequency stop consonants (/di/ and /gi/) elicit larger amplitude responses than high-frequency emphasis stops (/ti/ and /pi/) (Kuruvilla-Mathew et al., 2015). This pattern indicates with evidence that high-frequency sounds evoke smaller AEPs than low-frequency sounds of equivalent intensity (Picton et al., 1978; Yrttiaho et al., 2008). Furthermore, aperiodic speech sounds like /ta/ have been shown to elicit smaller N1 responses than periodic sounds like /da/ (Yrttiaho et al., 2008).

It was worth mentioning that, contrary to Tb, it seems that the larger amplitude of Ta to speech sound /da/ than /ta/ was stronger at the right hemisphere than in the left (Table 2). While this finding may suggest the rightward lateralization of fast temporal processing at the level of Ta's generators, it does not support the previous findings (Karen L Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). Clunies-Ross et al., 2015 reported a symmetric hemisperic Ta response to short and long temporal interval in adults, and suggested that it is reasonable to see a symmetric pattern of Ta between right and left hemispheres to fast and slow temporal

processing because Ta generates mainly from primary auditory cortex. This idea is consistent with the theory of Asymmetric Sampling in Time (AST) propsed by Poeppel (2003) proposing that the auditory structure in primary auditory cortex right and left hemispheres contribute to symmetric auditory temporal processing (Bishop et al., 2011). However, it seems there is a developmental effect on symmetric pattern of temporal processing in auditory cortex (Clunies-Ross et al., 2018). Clunies-Ross et al., (2018) using the same experiment as their study from 2015 (Karen L Clunies-Ross et al., 2015) recorded the Ta to a pair of tones with short and long intervals (25 vs 100ms), in children at age 7 and 9. Results showed that there was no significant difference between Ta responses across all intervals over the left hemisphere. This findings was similar to the pattern of Ta in an adult population (Karen L Clunies-Ross et al., 2015) suggesting that the associated auditory areas in the left hemisphere may function according to a short temporal integration window, and also that the generator of the Ta in the left hemisphere may be mature at age 7. However, contrary to the left hemisphere, there was a attenuated amplitude of Ta to tone-pairs with short interval compared to long intervals at the right hemisphere in children (Clunies-Ross et al., 2018). Author interpeted that this pattern firstly shows that the generators of the Ta in the right hemisphere are functioning according to a longer integration window of approximately 100 ms in children. Secondly, as this finding was not in line with data from adults (Karen L Clunies-Ross et al., 2015), it was proposed that the sensitivity to longer intervals could reflect the later development of the Ta generators in the right hemisphere. This idea supports the reports of a previous study such as Mahajan and McArthur (2013) who found that the Ta peak over the right hemisphere continued to develop throughout adolescence, suggesting that the functional development of the left and right hemispheres differs (Mahajan and McArthur, 2013).

Given the inconsistencies across studies and with current results, as well as the variability in reported neural generators of Ta (Clunies-Ross et al., 2018), the functional implications of hemispheric asymmetries in Ta during development remains unresolved and require further investigation.

Overall, the results in quiet conditions showed the encoding of both long and short VOTs at the level of secondary auditory cortex, with a clear rigtward lateralizarion for long VOTs in children aged 8-12 years old. These findings provide further insights into temporal processing at higher level of the auditory cortex and suggest that Tb, in particular, may serve as a useful electrophysiological marker of temporal processing in children, including those with auditory processing disorders.

5.4.2. Difference between representation of /da/ and /ta/ in noise

Another key finding of the current study was a longer Ta latency in resonse to the speech sound /ta/ compared to /da/ in noisy conditions (Figure 4). This result suggests that noise has a more detrimental effect on auditory responses to long VOT syllables than to short VOT syllables.

Compared to /d/, the consonant /t/ contains greater high-frequency onset energy and is characterized by a slower rise time and reduced burst energy, making it more vulnerable to masking by noise (Kuruvilla-Mathew et al., 2015). In more detail, in syllables with voiced consonants like /da/, the voiced consonant /d/ possess a short VOT, and contains an onset burst with similar spectral energy as the following vowel's formant frequencies (vowel a) (Kuruvilla-Mathew et al., 2015). While, the syllables with voiceless consonants like /ta/, have longer VOT and does not have similar-to-vowels spectral energy at the onset, they contain little energy in the first formant (F1) frequency region before the onset of the vowel (Kuruvilla-Mathew et al., 2015). The smaller energy at the onset probably makes the voiceless syllables more vulnerable to noise, which was observed in longer latency.

5.4.3. Correlation between behavioral responses and AEPs

Altough the results of AEPs obtained in noise revealed greater suppression of auditory responses to the speech sound /ta/ than those recorded to speech sound /da/, this effect was not reflected in behavioral results. Specifically, a significant interaction between listening conditions and stimulus was observed for Ta latency, with longer latencies to speech sound /ta/ than to /da/ only in noise. In contrast, the significant effect of stimulus on reaction time did not interact with listening condition, such that reaction times to speech sound /ta/ were consistently longer than to speech sound /da/ in both quiet and noise listening conditions.

The AEP and behavioral correlations are typically characterized by small-to-moderate effect sizes. Givent the non signifcant correlations and the weak pearson r (see Appenndix), the present study may have been underpowered to detect consistent associations across all conditions. Larger samples will be necessary to determine whether the observed pattern generalizes across stimuli and listening environments.

5.5. Future research

This study examined speech processing at a single SNR; future research incorporating multiple SNR levels would provide deeper insight into the neural processing of temporal

features in speech stimuli. In addition, further studies using the T-complex in populations with poor speech perception in noise may shed light on underlying temporal neural processing impairments in these groups.

5.6. Conclusion

This study demonstrated the representation of both long and short voice onset times (VOTs) in the auditory system, as indexed by the T-complex. A right-hemispheric advantage was observed for long VOTs compared to short VOTs, as reflected in the Tb component, the latest peak of the T-complex. These findings indicate hemispheric lateralization of temporal processing at the level of the secondary auditory cortex and contribute to a more refined understanding of asymmetric temporal processing at higher level of the auditory pathway. Importantly, these results suggest potential clinical relevance by supporting the use of a non-invasive electrophysiological measure to objectively assess temporal processing abilities. Such measures, using stimuli varying in VOT and SNR, may be particularly useful in at-risk young children with temporal processing disruption, including those with auditory processing disorder and dyslexia.

5.7. CRediT authorship contribution statement

Zohreh Ahmadi: Writing – review & editing, Writing – original draft, Visualization, Methodology, Project administration, Investigation, Formal analysis, Conceptualization. Amineh Koravand: Writing – review & editing, Visualization. Shanna Kousaie: Writing – review & editing, Methodology, Funding acquisition, Formal analysis. Benjamin Rich Zendel: Writing – review & editing, Supervision, Methodology, Resources, Project administration.

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5.9. Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper**.**

5.10. Data availability

Data will be made available on request.

5.11. Declaration of generative AI and AI-assisted technologies in the writing process

No AI tools or services were used during the preparation of this work.

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5.13. Appendix

Table 1. Pearson's Correlations between the latency of Ta and behavioral measurements, in response to speech sound /da/ in quiet.

Variable		Ta7- _z	Ta8- z	/da/ACQ_z	RT-/da/Q_z
1. Ta7 _z	Pearson's r	—			
	p-value	—			
2. Ta8 _z	Pearson's r	0.262	—		
	p-value	0.345	—		
3. /da/ACQ_z	Pearson's r	0.075	0.261	—	
	p-value	0.790	0.347	—	
4. RT-/da/Q_z	Pearson's r	-0.040	-0.117	0.321	—
	p-value	0.889	0.679	0.243	—

Table 2. Pearson's Correlations between the latency of Ta and behavioral measurements, in response to speech sound /da/, in Babble noise.

Variable		Ta7- z	Ta8- _z	/da/ACB_z	RT-/da/B_z
1. Ta7 z	Pearson's r	—			
	p-value	—			
2. Ta8 z	Pearson's r	0.226	—		
	p-value	0.418	—		
3. /da/ACB_z	Pearson's r	0.111	-0.152	—	
	p-value	0.694	0.589	—	
4. RT-/da/B_z	Pearson's r	-0.262	0.176	0.063	—
	p-value	0.346	0.529	0.823	—

Table 3. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /da/ in quiet.

Variable		Tb7- _z	Tb8- z	/da/ACQ_z	RT-/da/Q_z
1. Tb7 z	Pearson's r	—			

Table 3. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /da/ in quiet.

Variable		Tb7- _z	Tb8- z	/da/ACQ_z	RT-/da/Q_z
	p-value	—			
2. Tb8 z	Pearson's r	0.659	—		
	p-value	0.008	—		
3. /da/ACQ_z	Pearson's r	-0.179	0.161	—	
	p-value	0.524	0.568	—	
4. RT-/da/Q_z	Pearson's r	-0.085	0.003	0.321	—
	p-value	0.764	0.991	0.243	—

Table 4. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /da/, in Babble noise.

Variable		Tb7 _z	Tb8 _z	/da/ACB_z	RT-/da/B_z
1. Tb7 z	Pearson's r	—			
	p-value	—			
2. Tb8 _z	Pearson's r	0.572	—		
	p-value	0.026	—		
3. /da/ACB_z	Pearson's r	0.078	-0.105	—	
	p-value	0.783	0.710	—	
4. RT-/da/B_z	Pearson's r	0.103	0.303	0.063	—
	p-value	0.715	0.272	0.823	—

Table 5. Pearson's Correlations between the latency of Ta and behavioral measurements, in response to speech sound /ta/ in quiet.

Variable		Ta7- _z	Ta8- z	/ta/ACQ_z	RT-/ta/Q_z
1. Ta7 z	Pearson's r	—			
	p-value	—			
2. Ta8 _z	Pearson's r	0.237	—		
	p-value	0.394	—		
3. /ta/ACQ_z	Pearson's r	-0.538	0.441	—	
	p-value	0.038	0.100	—	
4. RT-/ta/Q_z	Pearson's r	-0.045	0.260	0.260	—
	p-value	0.872	0.349	0.350	—

Table 6. Pearson's Correlations between the latency of Ta and behavioral measurements, in response to speech sound /ta/ in Babble noise.

Variable		Ta7- _z	Ta8 _z	/ta/ACB_z	RT-/ta/B_z
1. Ta7 _z	Pearson's r	—			
	p-value	—			
2. Ta8 z	Pearson's r	0.179	—		
	p-value	0.523	—		
3. /ta/ACB_z	Pearson's r	-0.162	0.459	—	
	p-value	0.565	0.085	—	
4. RT-/ta/B_z	Pearson's r	-0.102	-0.201	-9.215×10^{-4}	—
	p-value	0.719	0.473	0.997	—

Table 7. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /ta/ in quiet

Variable		Tb7- _z	Tb8- _z	/ta/ACQ_z	RT-/ta/Q_z
1. Tb7 z	Pearson's r	—			
	p-value	—			
	Spearman's rho	—			
	p-value	—			
2. Tb8 z	Pearson's r	0.288	—		
	p-value	0.299	—		
	Spearman's rho	0.377	—		
	p-value	0.166	—		
3. /ta/ACQ_z	Pearson's r	-0.430	0.256	—	
	p-value	0.110	0.357	—	
	Spearman's rho	-0.329	0.203	—	
	p-value	0.232	0.467	—	
4. RT-/ta/Q_z	Pearson's r	-0.366	0.019	0.260	—
	p-value	0.180	0.947	0.350	—
	Spearman's rho	-0.108	-0.014	0.398	—
	p-value	0.702	0.960	0.142	—

Table 8. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /ta/ in Babble noise.

Variable		Tb7- _z	Tb8- _z	/ta/ACB_z	RT-/ta/B_z
1. Tb7 z	Pearson's r	—			
	p-value	—			
2. Tb8 z	Pearson's r	0.044	—		
	p-value	0.878	—		
3. /ta/ACB_z	Pearson's r	-0.235	0.304	—	
	p-value	0.399	0.270	—	

Table 8. Pearson's Correlations between the latency of Tb and behavioral measurements, in response to speech sound /ta/ in Babble noise.

Variable		Tb7- _z	Tb8- _z	/ta/ACB_z	RT-/ta/B_z
4. RT-/ta/B_z	Pearson's r	0.051	0.337	-9.215×10^{-4}	—
	p-value	0.857	0.219	0.997	—

Table 9. Pearson's Correlations between the amplitude of Ta and behavioral measurements, in response to speech sound /da/ in Quiet.

Variable		Ta7- z	Ta8- _z	/da/ACQ_z	RT-/da/Q_z
1. Ta7- _z	Pearson's r	—			
	p-value	—			
2. Ta8- _z	Pearson's r	0.341	—		
	p-value	0.213	—		
3. /da/ACQ_z	Pearson's r	-0.158	-0.255	—	
	p-value	0.574	0.358	—	
4. RT-/da/Q_z	Pearson's r	0.016	-0.277	0.321	—
	p-value	0.955	0.318	0.243	—

Table 10. Pearson's Correlations between the amplitude of Ta and behavioral measurements, in response to speech sound /da/, in Babble noise.

Variable		Ta7 _z	Ta8- _z	/da/ACB_z	RT-/da/B_z
1. Ta7- z	Pearson's r	—			
	p-value	—			
2. Ta8- z	Pearson's r	0.489	—		
	p-value	0.064	—		
3. /da/ACB_z	Pearson's r	-0.216	-0.262	—	
	p-value	0.439	0.346	—	
4. RT-/da/B_z	Pearson's r	0.238	0.234	0.063	—
	p-value	0.392	0.401	0.823	—

Table 11. Pearson's Correlations between the amplitude of Tb and behavioral measurements, in response to speech sound /da/ in Quiet.

Variable		Tb7 _z	Tb8- _z	/da/ACQ_z	RT-/da/Q_z
1. Tb7- _z	Spearman's rho	—			
	p-value	—			
2. Tb8- z	Spearman's rho	0.510	—		

Table 11. Pearson's Correlations between the amplitude of Tb and behavioral measurements, in response to speech sound /da/ in Quiet.

Variable		Tb7 _z	Tb8- _z	/da/ACQ_z	RT-/da/Q_z
	p-value	0.052	—		
3. /da/ACQ_z	Spearman's rho	-0.074	-0.358	—	
	p-value	0.794	0.189	—	
4. RT-/da/Q_z	Spearman's rho	-0.250	-0.132	0.267	—
	p-value	0.368	0.638	0.336	—

Table 12. Pearson's Correlations between the amplitude of Tb and behavioral measurements, in response to speech sound /da/, in Babble noise.

Variable		Tb7- z	Tb8- z	/da/ACB_z	RT-/da/B_z
1. Tb7- z	Pearson's r	—			
	p-value	—			
2. Tb8- z	Pearson's r	0.530	—		
	p-value	0.042	—		
3. /da/ACB_z	Pearson's r	-0.092	-0.353	—	
	p-value	0.744	0.197	—	
4. RT-/da/B_z	Pearson's r	0.487	0.092	0.063	—
	p-value	0.066	0.745	0.823	—

Table 13. Pearson's Correlations between the amplitude of Ta and behavioral measurements, in response to speech sound /ta/ in Quiet.

Variable		Ta7z	Ta8- z	/ta/ACQ_z	RT-/ta/Q_z
1. Ta7- z	Pearson's r	—			
	p-value	—			
2. Ta8- z	Pearson's r	0.331	—		
	p-value	0.228	—		
3. /ta/ACQ_z	Pearson's r	0.127	0.071	—	
	p-value	0.653	0.800	—	
4. RT-/ta/Q_z	Pearson's r	0.231	-0.440	0.260	—
	p-value	0.408	0.100	0.350	—

Table 14. Pearson's Correlations between the amplitude of Ta and behavioral measurements, in response to speech sound /ta/, in Babble noise.

Variable		Ta7z	Ta8- z	/ta/ACB_z	RT-/ta/B_z
1. Ta7- z	Pearson's r	—			
	p-value	—			
2. Ta8- z	Pearson's r	0.046	—		
	p-value	0.871	—		
3. /ta/ACB_z	Pearson's r	-0.144	-0.238	—	
	p-value	0.608	0.393	—	
4. RT-/ta/B_z	Pearson's r	0.055	0.031	-9.215×10^{-4}	—
	p-value	0.847	0.912	0.997	—

Table 15. Pearson's Correlations between the amplitude of Tb and behavioral measurements, in response to speech sound /ta/ in Quiet.

Variable		Tb7- _z	Tb8- z	RT-/ta/Q_z	/ta/ACQ_z
1. Tb7- z	Pearson's r	—			
	p-value	—			
2. Tb8- z	Pearson's r	0.349	—		
	p-value	0.202	—		
3. RT-/ta/Q_z	Pearson's r	-0.037	-0.226	—	
	p-value	0.897	0.418	—	
4. /ta/ACQ_z	Pearson's r	-0.057	0.148	0.260	—
	p-value	0.839	0.599	0.350	—

Table 16. Pearson's Correlations between the amplitude of Tb and behavioral measurements, in response to speech sound /ta/ in Babble noise.

Variable		Tb7 _z	Tb8- z	/ta/ACB_z	RT-/ta/B_z
1. Tb7- z	Pearson's r	—			
	p-value	—			
2. Tb8- z	Pearson's r	0.102	—		
	p-value	0.719	—		
3. /ta/ACB_z	Pearson's r	0.006	-0.255	—	
	p-value	0.983	0.359	—	
4. RT-/ta/B_z	Pearson's r	0.176	-0.212	-9.215×10^{-4}	—
	p-value	0.531	0.449	0.997	—

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Chapter 4. APD study

Evaluation of frontocentral and temporal auditory evoked potentials in children with auditory processing disorder, in quiet and in noise, using stimuli with various temporal features

6.1. Introduction

Auditory processing disorder (APD) manifests with difficulties in auditory processing skills such as binaural processing, listening in adverse listening conditions, and temporal processing (ASHA, 2005). The prevalence of APD is reported by different studies to vary from 0.5% to 10% (Bamiou et al., 2001; Hind et al., 2011; Nagao et al., 2016). Speech perception in the presence of background noise is known to be the most challenging condition for children with APD (Hind et al., 2011; Muthuselvi & Yathiraj, 2009). Since learning in schools often occurs in noisy environments, these speech-in-noise (SIN) deficits have debilitating effects on learning skills and the academic achievements of children with APD (Alanazi, 2023; Bamiou et al., 2001; Canale et al., 2014; Cestnick & Jerger, 2000; Del Zoppo et al., 2015; Drosos, 2024; Hunsaker, 1983; Sperling et al., 2005).

Considering the prevalence and detrimental effects of APD, identification of APD is highly recommended (ASHA. 2005). A comprehensive evaluation of auditory processing disorder is recommended to include both subjective and objective measurements (Dillon & Cameron, 2021b). Behavioral techniques or subjective methods assesses specific auditory mechanisms, such as dichotic listening, temporal resolution, and speech perception in noise (ASHA, 2005). However, the interpretation of these behavioral assessments can be complicated by non-auditory factors, such as motivation (Geffner & Ross-Swain, 2014). For this reason, there has been increasing emphasis on incorporating objective physiological and electrophysiological tests measures into APD assessment (Alanazi, 2023; Dillon & Cameron, 2021a; Ferre, 1997; Maggu & Overath, 2021).

Maggu & Overath, 2021, recently proposed an objective test battery, referred to as *site-based APD evaluation*, to assess auditory processing using physiological and neurophysiological measures (refere to table 1 in chapter 1 for review). This test battery evaluates the responses from the peripheral auditory system up to central auditory system at levels of subcortical areas or brainstem (Maggu & Overath, 2021). Although this assessment battery offers valuable insight into the locus and nature of auditory pathway dysfunction, it mainly targets subcortical and brainstem processing mechanisms. Consequently, there is a compelling need to extend evaluation to cortical levels of auditory processing. Incorporating neurophysiological measures of cortical responses alongside subcortical indices may yield a more comprehensive and integrated understanding of auditory processing deficits, particularly in individuals with auditory processing disorder (APD).

Multiple studies have evaluated the utility of cortical auditory responses in identification of children with APD (Fox-Thomas, 2003; Koravand et al., 2017; Tone Stokkereit Mattsson et al., 2019; Morlet et al., 2019), however, a high degree of variability in the reported patterns of cortical responses across these studies makes it challenging to identify consistent trends or draw definitive conclusions (Macaskill et al., 2022). For example, some studies have reported no significant differences in the latencies or amplitudes of frontocentral components between children with APD and their typically developing (TD) peers (Fox-Thomas, 2003; T. S. Mattsson et al., 2019). Macaskill et al., 2022 has mentioned the variability in findings may be partially attributed to heterogeneous population and heterogeneous methodology.

6.1.1. Heterogeneous population

Across the AEP studies in children with APD, diagnostic criteria vary substantially (Macaskill et al., 2022). While some studies relied on ASHA diagnostic descriptions (Jutras et al., 2019; Koravand et al., 2017; T. S. Mattsson et al., 2019), other studies have used test batteries defined in the scope of their research project, typically made up of a wide range of auditory processing tests (Hassaan, 2015; Liasis et al., 2003; Purdy et al., 2002). It other words, these studies probably have included APD children with impairments in different underlying auditory mechanisms. This lack of consistency in diagnostic approaches likely contributes to variability in reported electrophysiological findings. Moreover, exclusion criteria and handling of comorbid conditions were not consistent. Some studies have used the specific test for excluding participant with impaired cognitive abilities (Jutras et al., 2019; Koravand et al., 2017; T. S. Mattsson et al., 2019; Purdy et al., 2002), however, others have relied on reports from parents or educators (Hassaan, 2015; Liasis et al., 2003; Sharma et al., 2006). Comorbidity with attention and language impairments is another factor that may result in varying findings (de Wit et al., 2018). As a result, applying a more holistic inclusion criteria for including participants with similar underlying auditory processing impairments and eliminating comorbidity with other neurodevelopmental disabilities may load to more consistent results in AEP studies.

6.1.2. Heterogeneous Methodology

Several methodological limitations may contribute to the high degree of variability observed in AEP findings in children with APD. The listening condition and stimuli type are likely the influencing factors for inconsistency in results. Regarding the listening conditions, most AEP studies in APD population have been conducted exclusively under quiet listening

conditions (Fox-Thomas, 2003; Koravand et al., 2017; Liasis et al., 2003; Purdy et al., 2002). Considering that the poor speech-in-noise ability is hallmark of APD, applying background noise may lead to more consistent significant differences in AEPs between APD and normal children (Anderson & Kraus, 2010; Cunningham et al., 2002; Russo et al., 2005). Moreover, among the studies using noise to assess the cortical AEPs in children with APD, only one type of noise has been used; white noise or babble noise (Hussain et al., 2022; T. S. Mattsson et al., 2019; Morlet et al., 2019). Comparing auditory cortical responses across different types of background noise including white noise and babble noise at the same time, could yield important insights into the neurophysiology of auditory processing in noise.

Regarding the stimuli type, it appears that most of the AEP studies have used simple tones or speech stimuli with similar spectral-temporal features like /da/ or/and /ba/ (Hussain et al., 2022, Fox-Thomas, 2003; Koravand et al., 2017, Mattsson et al., 2019, Morlet et al., 2019, Macaskill et al., 2022). It has been mentioned that speech sounds versus simple pure tones seems to be more effective in identification of APD (Omidvar et al., 2023). Also, given that poor temporal processing is one the most fundamental dysfunction in children with APD leading to difficulties in speech perception in noise (Almeqbel & McMahon, 2015; Dillon & Cameron, 2021b; Talcott et al., 2000; Warrier et al., 2004), using different speech stimuli with distinct temporal dynamic may be promising to evaluate temporal processing more efficiently than using speech with similar temporal features.

Voice onset time (VOT) is one of the important temporal cues to discriminate stop consonants and understanding speech in noisy environments (Sharma & Dorman, 1999; Warrier et al., 2004). VOT has been widely used to evaluate temporal processing in children with listening difficulties (Almeqbel & McMahon, 2015; Talcott et al., 2000; Warrier et al., 2004). To the best of our knowledge, only one study has assessed the representation of VOT in children with APD (Hestvik et al., 2023). Hestvik et al., 2023 recorded Mismatch Negativity (MMN) and identified impairments in representation of short and long VOTs in APD populations. Although this study suggests impaired temporal processing in APD populations, it was conducted only in quiet and did not report other cortical responses which arise from different generators like temporal auditory cortex (Ceponiene et al., 1998; Deouell et al., 1998).

Cortical auditory responses based on scalp distribution, include two main categories, frontocentral and temporal auditory responses (T-complex) (Tonnquist-Uhlen et al., 2003; Wolpaw & Penry, 1975). These two responses differ in their neural generators and trajectory

of maturation (Ponton et al., 2000; Tonnquist-Uhlen et al., 2003) While frontocentral responses arise mainly from primary auditory areas, superior temporal plane, and mature around age 9-10, T-complex seems to be mature earlier (4-5 years old) and arises mainly from secondary auditory areas, lateral superior temporal gyrus (Pang & Taylor, 2000; Ponton et al., 2000; Tonnquist-Uhlen et al., 2003). Unlike frontocentral responses that have been widely used to assess auditory processing in APD populations, there is no study that investigates T-complex in children with APD (Ahmadi et al., 2024).

Multiple studies have consistently shown the utility of T-complex in identification of auditory processing impairments in neurodevelopmental disorders (D. V. Bishop et al., 2012; Rinker et al., 2021; Shafer et al., 2011; Tonnquist-Uhlén, 1996b). Interestingly, some studies have shown atypical pattern of T-complex in children with dyslexia while there was no difference in frontocentral responses between children with APD and typically developing (TD) children (Bruneau et al., 1999; Gomes et al., 2012). Collectively, given different neural generators and existence of distinct patterns between frontocentral and temporal response, examining both frontocentral responses and T-complex waveform, in individuals with APD might provide information that is complementary to the more commonly reported frontocentral AEP components. Such research could bring valuable insights into the nature and locus of auditory processing impairments, particularly in determining whether observed deficits are more closely associated with activity in the superior temporal plane or the lateral superior temporal gyrus (Wagner et al., 2017).

Considering the previously mentioned methodological limitations in AEPs studies, the current study aimed to explore the utility of cortical auditory response in children with APD using different stimuli and noise as well as exploring different types of cortical responses including frontocentral and temporal responses. By incorporating diverse stimuli and recording sites, this chapter aims to provide a more comprehensive understanding of cortical auditory processing in APD. The specific objectives of this chapter are presented in the following section.

The first objective of the APD study was to examine differences in the morphology of frontocentral and temporal auditory responses (T-complex) between children with APD and typically developing children. These comparisons were conducted across quiet and different noise conditions, using multiple stimulus types to assess potential group-related variations in cortical response patterns. Based on the existing literature, it was hypothesized that children

with APD would exhibit atypical patterns in both frontocentral and temporal AEPs compared to typically developing children, particularly when responses were elicited by speech stimuli in the presence of babble noise. Furthermore, it was hypothesized that the T-complex in children with APD would demonstrate response patterns distinct from those observed in frontocentral components, reflecting potential differences in underlying cortical processing mechanisms.

The second objective of the APD study was to examine the cortical representation of long and short voice onset times (VOTs) using the T-complex in children with APD. In addition, behavioral performance for long and short VOT contrasts was assessed to complement the electrophysiological findings. Given the documented temporal processing deficits in children with APD, it was hypothesized that differences in VOT representation would be observed between children with APD and typically developing children, both at the cortical and behavioral levels.

6.2. Method

6.2.1. Participants

Two groups including five typically developing (TD) children and five children with APD. All participants TD and APD groups had normal hearing detection thresholds at 15 dB HL or less in octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996)'s protocol. Tympanograms were normal in all participants (admittance curve with a single peak between +50 to -100daPa using a 226-Hz probe tone). All groups were right-handed and it was determined according to an adapted protocol to assess laterality (De Agostini & Dellatolas, 1988). No history of otological or neurological disorders was reported by any of the normal participants. In addition, participants with musical training were not included, as previous studies have shown that music practice can enhance speech-in-noise perception and increase the amplitude of AEPs in the presence of background noise compared to quiet (Anderson et al., 2013; Benjamin Rich Zendel et al., 2015). The children in APD group also did not have musical training.

Auditory processing domain questionnaire (APDQ) was used to assess auditory processing, attention and language abilities of children in the TD group. APDQ is a screening tool with 70-85% accuracy and evaluates the auditory processing, attention and language skills of individual between 7-18 years old (O'Hara & Mealings, 2018). Considering that the poor perception of speech in noise is the hallmark of APD, the ability to understanding speech in

noise was also evaluated in the normal group to make sure there were not listening difficulties among the children included TD group. Bamford-Kowal-Bench Sentences in Noise (BKB-SIN) (BKB-SIN, 2005) was used for this purpose. The BKB-SIN test is designed to estimate the required listener's SNR to obtain 50% of key words correct. The test consists of 18 list pairs (e.g., lists 1a and 1b are to be used together) of 10 sentences each spoken by a male speaker and in the presence of four-talker babble. The participants needed to repeat the whole sentence. In each list, the sentences are presented at pre-recorded SNRs that decrease 3-dB steps from a +21 (for the first sentence) to a -6 dB SNR (for the last sentence). Each sentence is prefaced with ''ready'' and has three target words, except for the first sentence which has four target words. Each list of 10 sentences requires 95s to administer. The following is one example of sentences in the BKB-SIN: The doge made and angry noise. All five children in TD group had normal performance in the BKB-SIN test.

The APD group were referred from a private hearing center, and they were already diagnosed with APD by the center. To reduce the influence of comorbid conditions and enhance sample homogeneity, only children without a prior diagnosis of ADHD, language impairment (LI), or other neurodevelopmental disorder were included in the APD group. Furthermore, all participants in the APD group exhibited documented deficits on temporal processing assessments and poor speech-in- noise performance, ensuring greater consistency in the auditory profile of the sample. Children either in TD or APD groups also needed to have normal performance in a cognitive ability test. In this regard, the auditory Continuous Performance Test (ACPT) was used, which is a cognitive task designed to measure a person's sustained attention and vigilance, particularly in an auditory context (Keith, 1994; Riccio et al., 1996). The results of the BKB, APDQ and ACPT have been inserted into table 1.

Table 1. *Demographic and Behavioral Characteristics of TD and APD Participants.*

Group	Gender	Age (years)	ACPT	BKB	AP (%)	LA (%)	TA (%)
TD	Male	11.4	4	2.5	85	78	88
TD	Female	12.2	5	1.5	95	85	99
TD	Male	10.5	6	1.5	100	100	86
TD	Male	8.6	3	0.5	100	95	100
TD	Male	10.2	2	2	100	98	94
APD	Female	12.1	4	5.5	DNT	DNT	DNT
APD	Male	10.3	3	7	DNT	DNT	DNT
APD	Male	11.5	4	4	DNT	DNT	DNT
APD	Male	11.5	7	4.5	DNT	DNT	DNT
APD	Male	9.2	3	6	DNT	DNT	DNT

AP=Auditory processing, APD=Auditory processing disorder, ATT=Attention, ACPT=Auditory Continuous Performance Test, BKB=Bamford-Kowal-Bench Speech-in-Noise, DNT=Did not test, LA=Language. TD=Typically developing.

6.2.2. Procedure

The same procedure that has been used for normal adult and children study, was applied in APD experiment as well. Children were seated and watched a silent movie, while auditory stimuli were delivered.

6.2.3. Stimuli

Stimuli were delivered binaurally in a passive non-oddball, homogenous paradigms. The non-speech stimulus was a pure tone 1kHz that was 250ms in duration and had a 10ms rise and fall time, created in Audacity software (Audacity Team, 1999). The speech stimuli were natural speech sounds /da/ and /ta/, that were originally recorded by a male voice and used in the study of Dubno & Schaefer in 1992. The speech stimuli had the same duration (250ms) and rise-fall time (10ms) as the non-speech stimuli. Stimuli were presented in three listening conditions including quiet, in the presence of babble noise and in the presence of white noise. For the non-speech paradigm, there were three blocks of 1kHz tones in the three listening conditions including quiet, white noise, and babble noise, each block contains 300 presentations of 1kHz. For the speech paradigm, there were three blocks for three listening conditions and each blocks contain 300 presentations of /da/ and 300 presentations of /ta/. The order of the listening conditions was randomized between subjects.

Stimuli were equalized for root mean square (rms) amplitude and then played at 80 dB SPL. For noisy conditions, signal-to-noise ratio was kept as +5dB, by reducing the intensity of noise to 75dB SPL. Interstimulus interval (ISI) as calculated from the offset of the preceding stimulus to the onset of the next stimulus, was jittered randomly between 1100-1250ms, as it has been shown that a slower stimulation rate results in more robust AEP waveforms in the immature auditory nervous systems (Phillip M. Gilley et al., 2005). The whole study including the two experimental paradigms took around two hours; 30 minutes for the non-speech experiment and 60 minutes for the speech stimuli experiment with a few short breaks between the blocks and experiments to decrease the risk of fatigue.

6.2.4. Behavioral evaluation

The behavioral test was done before the EEG recording. In the behavioral experiment, the participant was instructed to discriminate between /ta/ and /da/. They needed to press the right button when they heard ta and the left button when they are heard da. There were 80 presentations of /da/ and 80 presentations of /ta/. Accuracy and Reaction time were calculated

as behavioral measures. The Accuracy and Reaction time for the stimulus /da/ were compared with results for the stimulus /ta/. Accuracy was calculated according to of the number of hits (i.e., participant was presented /da/ and reported /da/) minus misses (participant was presented /da/ but reported /ta/).

6.2.5. Electrophysiological recording

The electrical responses were recorded from 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). The electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp, using the international 10-20 system of electrode placement. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. The tip of the nose served as an online reference for all channels, including the electro-oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. The EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for later analyses.

6.2.6. Electrophysiological data analysis

The data were analysed using Brain Products' Analyzer2 software. The signals were digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts were corrected in the EEG using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1995). To do so required a computation of vertical and horizontal EOG activity. A vertical EOG was computed by subtracting the inferior orbital and the FP2 channels and horizontal eye movements were computed by subtracting the FT9 and FT10 channels (Duda-Milloy et al., 2019). The ICA model assumes that the observed electrophysiological signals represent a linear mixture of neural sources and artefacts that are unknown but are mutually statistically independent. The algorithm relies on blind source separation which was trained to recognize each participant's ocular artefact signature which was then partitioned out of the EEG traces. Epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset. Segmented waveforms were baseline corrected around the 100ms pre-stimulus interval and averaged based on condition. Epochs were excluded if they had amplitudes exceeding ±100μV at any site. Less than 10% of trials were excluded. Moreover, the EEG was visually inspected to ensure adequate artifact rejection. Averaged waveforms were computed for each subject and then across subjects to obtain grand averaged waveforms for each condition. Although both Cz and Fz were selected for the detection of

frontocentral responses (P1-N1-P2-N2), only the responses at Cz are reported as there were more robust responses at Cz compared to Fz (Näätänen & Picton, 1987). This method is consistent with previous studies that have reported the P1-N1-P2-N2 responses at Cz, because this electrode showed the largest amplitude values, and hence were more susceptible to reveal statistical findings in children (Duquette-Laplante et al., 2024; Hussain et al., 2022; T. S. Mattsson et al., 2019; Morlet et al., 2019). Additionally, the amplitude of the N1 at Cz has been considered the strongest predictor of individual speech-in-noise perception thresholds (Billings et al., 2013). The T-complex was extracted from the temporal regions, T7 and T8 (Wolpaw & Penry, 1975). Tonnquist-Uhlen and colleagues (2003) also suggest that T7 and T8 are optimal for measuring the T-complex since they reflect little activity from the sources underlying the P1, N1 and P2. Regarding T-complex components, as Na was not detected clearly in most of the participants; accordingly, only Ta and Tb are reported in the results. In both groups, Tb was not identifiable in babble noise in response to /da/ and the P1, N2 and Tb were not identifiable in response to /ta/ in babble noise, also in APD group N2 was not identified in response to /da/ in-babble noise, so these conditions were removed from the analysis.

A peak detection procedure was performed for each participant to measure peak latency for each component. Regarding amplitude measurements a mean amplitude method was applied. Mean amplitude rather than peak amplitude is less affected by noise, leading to more efficient responses (Clayson et al., 2013; Luck, 2012). The mean amplitudes were calculated based on an interval between two points (25ms) on either side of the peak (i.e., a 50ms window) for each peak. This time window was based on the grand averaged ERPs at sites Cz, T7 and T8 (Anderson, Chandrasekaran, et al., 2010; J. A. Hämäläinen et al., 2011). The latency intervals over which each of the mean amplitudes were calculated are reported in Table2 and 3.

Table 2. *Latency intervals over which each of the mean amplitudes were calculated in TD children, in three listening conditions including quiet (Q), white noise (W) and babble noise (B), in response to three stimuli including 1kHz, /da/ and /ta/.*

Peak	Stimuli-listening conditions								
	1 kHz Q	**1 kHz W**	**1 kHz B**	**/da/ Q**	**/da/ W**	**/da/ B**	**/ta/ Q**	**/ta/ W**	**/ta/ B**
P1	63–113ms	65–115ms	65–115ms	63–113ms	95–145ms	116–169ms	57–107ms	147–197ms	171–221ms
N1	103–153ms	109–159ms	113–163ms	109–159ms	133–183ms	243–291ms	103–153ms	215–265ms	311–361ms
P2	141–191ms	161–211ms	161–211ms	147–197ms	189–239ms	341–391ms	177–227ms	287–337ms	Not identified
N2	245–295ms	245–295ms	243–293ms	245–295ms	285–335ms	435-460ms	371–421ms	415–465ms	Not identified

Ta-T7	103–153ms	99–149ms	99–149ms	137–187ms	139–189ms	271–321ms	133–183ms	219–269ms	341–391ms
Tb-T7	157–207ms	139–189ms	145–195ms	169–219ms	203–253ms	Not identified	163–213ms	285–335ms	Not identified
Ta-T8	103–153ms	109–159ms	109–159ms	129–179ms	141–191ms	237–287	137–187ms	219–269ms	295–345ms
Tb-T8	153–203ms	163–213ms	161–211ms	169–219ms	199–249ms	Not identified	177–227ms	287–337ms	Not identified

Table 14 *Latency intervals over which each of the mean amplitudes were calculated in APD children, in three listening conditions including quiet (Q), white noise (W) and babble noise (B), in response to three stimuli including 1kHz, /da/ and /ta/.*

Peak	**Stimuli-listening conditions**								
	1 kHz Q	**1 kHz W**	**1 kHz B**	**/da/ Q**	**/da/ W**	**/da/ B**	**/ta/ Q**	**/ta/ W**	**/ta/ B**
P1	61–111ms	50–105ms	65–117ms	69–119ms	93–143ms	121–171ms	73–123ms	175–225ms	177–227ms
N1	103–153ms	105–150ms	103–153ms	115–165ms	141–191ms	271–321ms	135–185ms	219–269ms	241–291ms
P2	127–177ms	159–209ms	151–205ms	141–191ms	173–233ms	339–389ms	173–223ms	243–293ms	Not identified
N2	239–289ms	235–285ms	243–293ms	215–265ms	261–311ms	Not identified	243–293ms	335–385ms	Not identified
Ta-T7	101–151ms	93–143ms	93–143ms	133–183ms	139–189ms	265–315ms	140–190ms	217–267ms	355–405ms
Tb-T7	139–289ms	145–195ms	143–193ms	159–209ms	199–249ms	Not identified	171–221ms	285–335ms	Not identified
Ta-T8	107–157ms	113–163ms	125–175ms	120–170ms	139–189ms	260–285ms	113–163ms	217–267ms	283–333ms
Tb-T8	159–209ms	163–213ms	170–210ms	170–220ms	199–249ms	Not identified	163–213ms	275–325ms	Not identified

6.2.7. Statistical analysis

Given the sample size, nonparametric statistical tests were employed to address the objectives of the APD study. Group differences in amplitude and latency of frontocentral and temporal responses between the APD and typically developing (TD) groups were examined using the Mann–Whitney U test for independent samples. To evaluate the effect of stimulus type (VOT) within each group, Wilcoxon signed-rank tests were conducted to compare the amplitude and latency of Ta and Tb responses to /da/ and /ta/. Due to the small sample size and the exploratory nature of the study, separate analyses were conducted to reduce model complexity and avoid overfitting. However, we acknowledge that a single omnibus mixed-design ANOVA including Group and VOT factors could provide additional information

regarding interaction effects and should be considered in future studies with larger sample sizes.

The outcomes of these analyses are presented in relation to the effect of noise and stimuli effects in the following section. The descriptive figures of results and the grand average of auditory evoked potentials have been inserted in the following.

6.3. Results of first objective (effect of noise experiment)

6.3.1. Results of Amplitude, T-complex

The results of Mann-Whitney showed a significant difference in amplitude of Ta, between APD and TD children (Table 1 of appendix). Ta had a significantly smaller amplitude (reduced positivity) in APD than TD, at electrode T7, in response to 1kHz in white noise listening condition (Figure 1-18). There was no significant difference in amplitude of Tb between TD and APD groups.

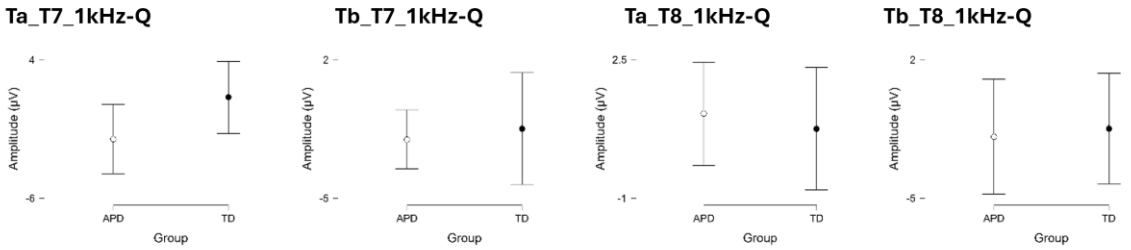

Figure 1. *The amplitude of Ta and Tb recorded at T7 and T8, in response to 1kHz in quiet, in TD and APD groups.*

Figure 2. *The grand average of temporal auditory evoked potentials in response to 1kHz, in quiet, in children with APD (red curve) and TD children (black curve).*

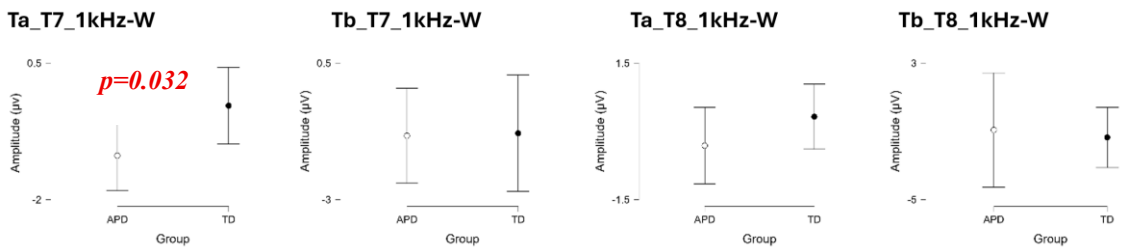

Figure 3. *The amplitude of Ta and Tb recorded at T7 and T8, in response to 1kHz, in white noise, in TD and APD groups. Ta had a smaller amplitude in children with APD than TD, at T7.*

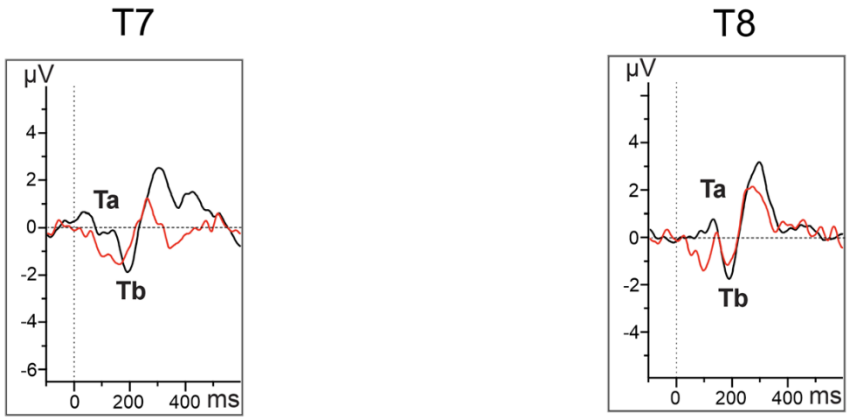

Figure 4. *The grand average of temporal auditory evoked potentials in response to 1kHz, in white noise, in children with APD (red curve) and TD children (black curve).*

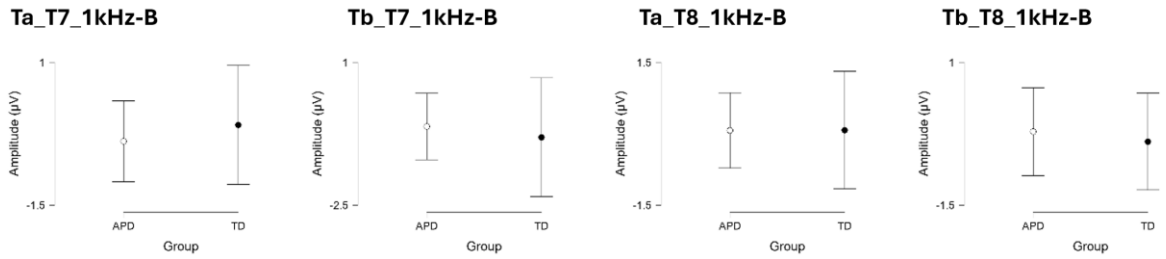

Figure 5. *The amplitude of Ta and Tb, recorded at T7 and T8, in response to 1kHz, in babble noise, in TD and APD groups.*

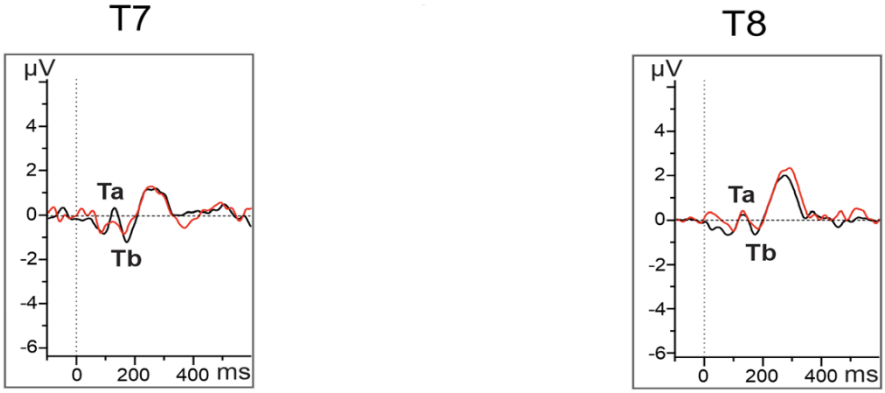

Figure 6. *The grand average of auditory evoked potentials in response to 1kHz, in babble noise, in children with APD (red curve) and TD children (black curve).*

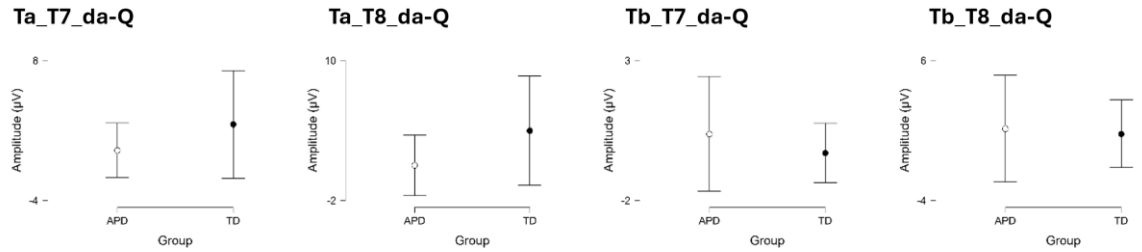

Figure 7. *The amplitude of Ta and Tb, recorded at T7 and T8, in response to /da/ in quiet, in TD and APD groups.*

Figure 8. *The grand average of auditory evoked potentials in response to /da/, in quiet, in children with APD (red curve) and TD children (black curve).*

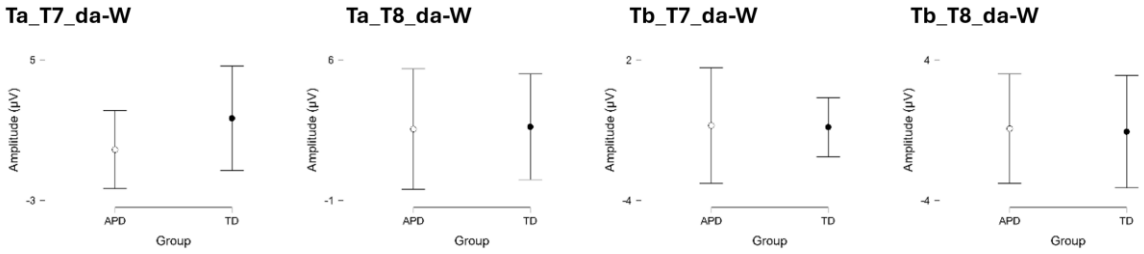

Figure 9. *The amplitude of Ta and Tb, recorded at T7 and T8, in response to /da/ in white noise, in TD and APD groups.*

Figure 10. *The grand average of temporal auditory evoked potentials in response to /da/, in white noise, in children with APD (red curve) and TD children (black curve).*

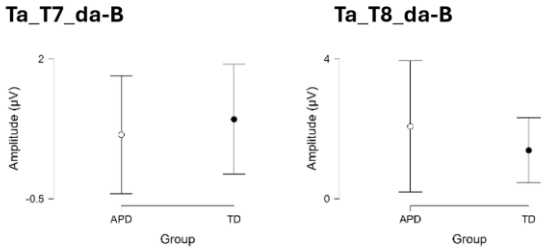

Figure 11. *The amplitude of Ta, recorded at T7 and T8, in response to /da/, in babble noise, in TD and APD groups.*

Figure 12. *The grand average of temporal auditory evoked potentials in response to /da/, in babble noise, in children with APD (red curve) and TD children (black curve).*

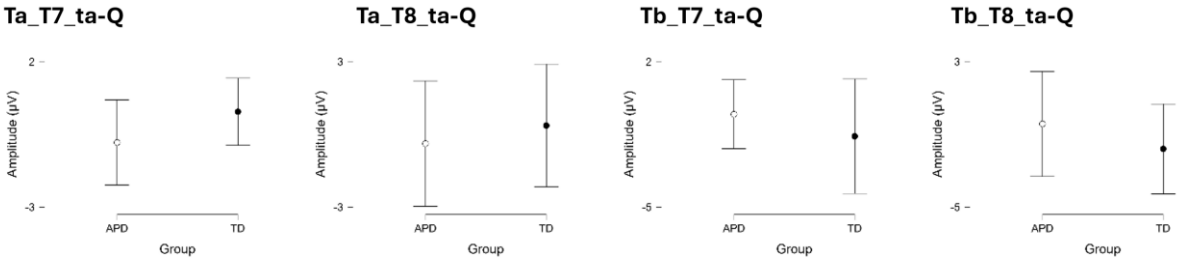

Figure 13. *The amplitude of Ta and Tb, recorded at T7 and T8, in response to /ta/ in quiet, in TD and APD groups*

Figure 14. *The grand average of temporal auditory evoked potentials in response to /ta/, in quiet, in children with APD (red curve) and TD children (black curve).*

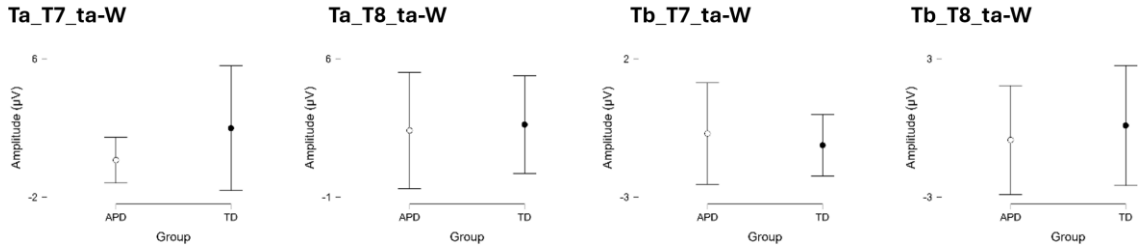

Figure 15. *The amplitude of Ta and Tb, recorded at T7 and T8, in response to /ta/ in white noise, in TD and APD groups*

Figure 16. *The grand average of temporal auditory evoked potentials in response to /ta/, in white noise, in children with APD (red curve) and TD children (black curve).*

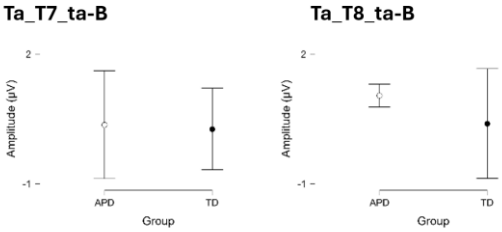

Figure 17. *The amplitude of Ta, recorded at T7 and T8, in response to /ta/ in babble noise, in TD and APD groups*

Figure 18. *The grand average of temporal auditory evoked potentials in response to /ta/, in babble noise, in children with APD (red curve) and TD children (black curve)*

6.3.2. Results of Amplitude, Frontocentral responses

Regarding the frontocentral responses, the significant difference was observed in amplitude of P1 and N2. There was a significantly larger amplitude of P1 in APD than TD group, in white noise using 1kHz (Table 2 of appendix). With the same stimuli, N2 had significantly a smaller amplitude in white noise condition, and a near to significant larger amplitude in babble noise, in APD versus TD group (Figure 19-36).

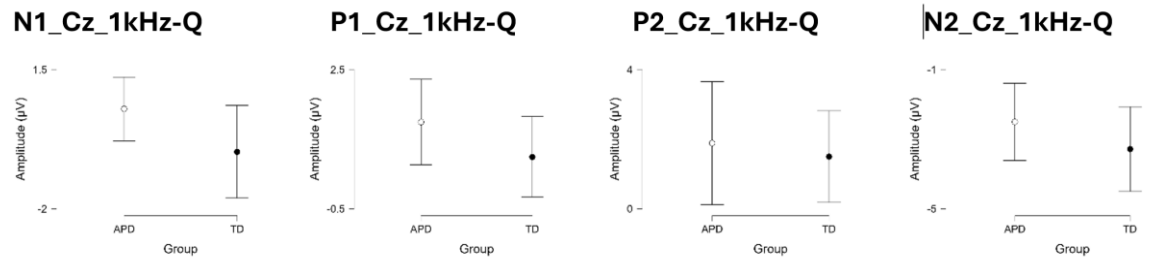

Figure 19. *The amplitude of frontocentral responses, recorded at Cz, in response to 1kHz in quiet, in TD and APD groups.*

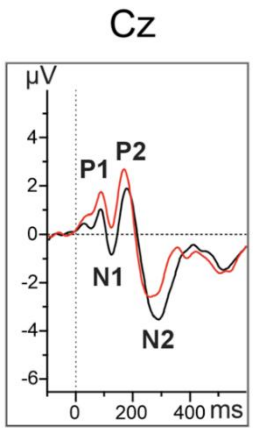

Figure 20. *The grand average of frontocentral auditory evoked potentials in response to 1kHz, in quiet, in children with APD (red curve) and TD children (black curve).*

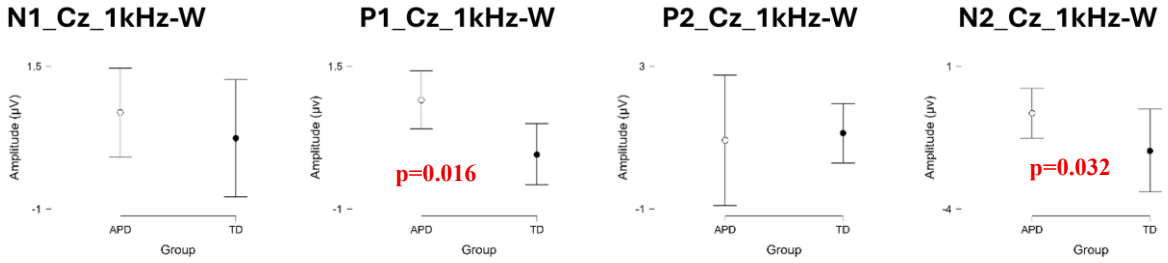

Figure 21. *The amplitude of frontocentral responses, recorded at Cz, in response to 1kHz in white noise, in TD and APD groups. The amplitude of P1 and N2 were larger and smaller, respectively, in APD than TD, in response to 1kHz in the presence of white noise.*

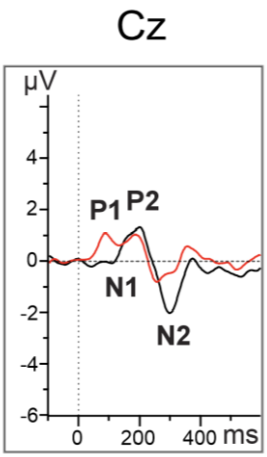

Figure 22. *The grand average of frontocentral auditory evoked potentials in response to 1kHz, in white noise, in children with APD (red curve) and TD children (black curve).*

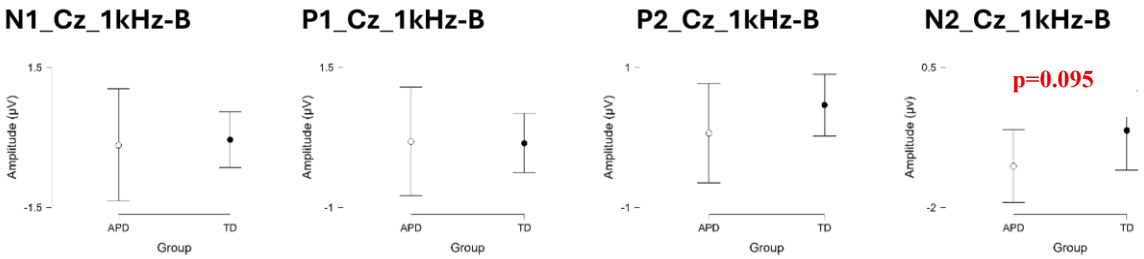

Figure 23. *The amplitude of frontocentral responses, recorded at Cz, in response to 1kHz in babble noise, in TD and APD groups. The amplitude of N2, in response to 1kHz, in the presence of babble noise was larger in APD than TD group.*

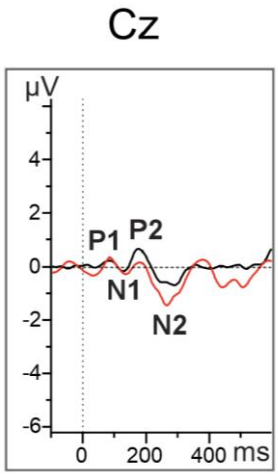

Figure 24. *The grand average of frontocentral auditory evoked potentials in response to 1kHz, in babble noise, in children with APD (red curve) and TD children (black curve).*

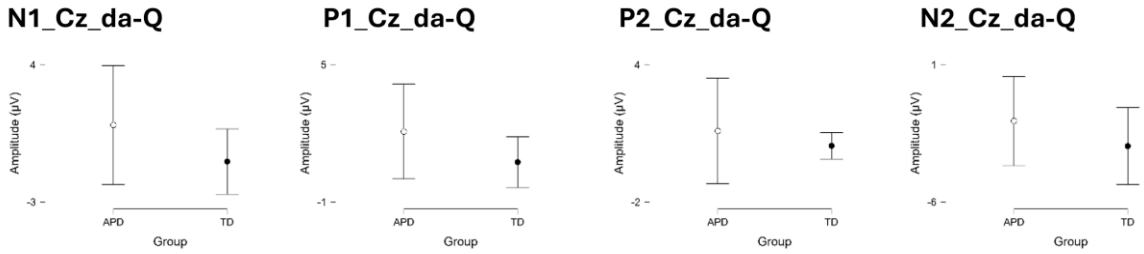

Figure 25. *The amplitude of frontocentral responses, recorded at Cz, in response to /da/ in quiet, in TD and APD groups.*

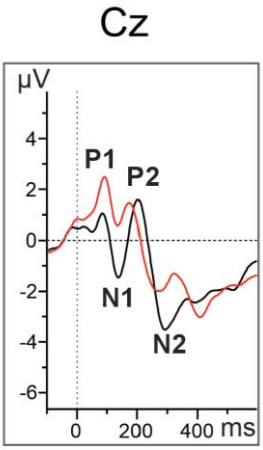

Figure 26. *The grand average of frontocentral auditory evoked potentials in response to /da/ in quiet, in children with APD (red curve) and TD children (black curve).*

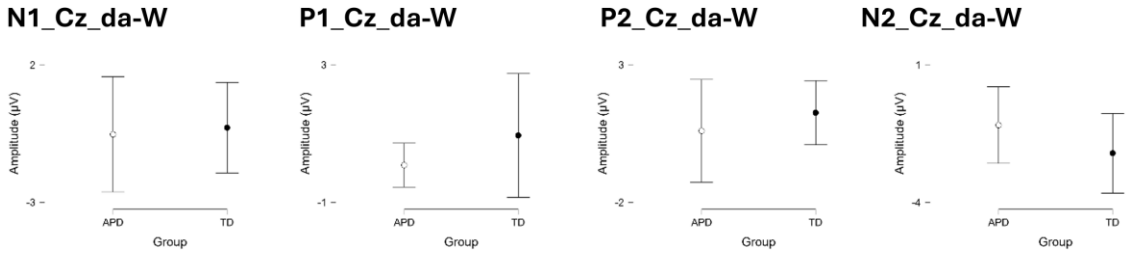

Figure 27. *The amplitude of frontocentral responses, recorded at Cz, in response to /da/ in white noise, in TD and APD groups.*

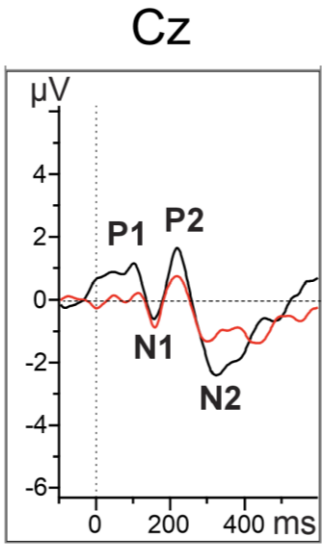

Figure 28. *The grand average of frontocentral auditory evoked potentials in response to /da/ in white noise, in children with APD (red curve) and TD children (black curve).*

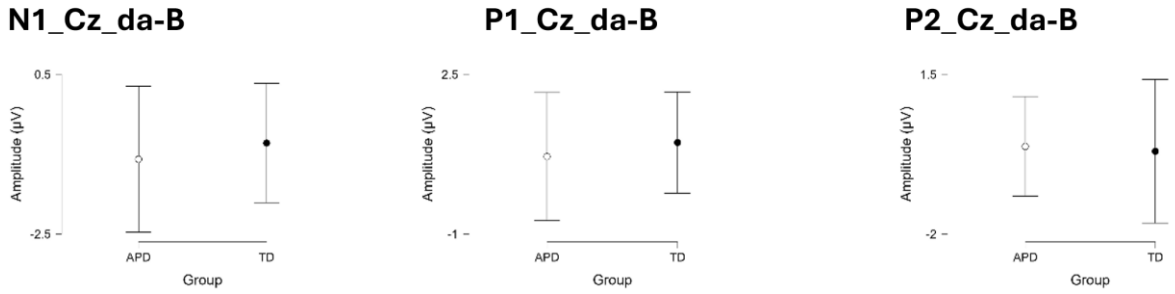

Figure 29. *The amplitude of frontocentral responses, recorded at Cz, in response to /da/, in babble noise, in TD and APD groups.*

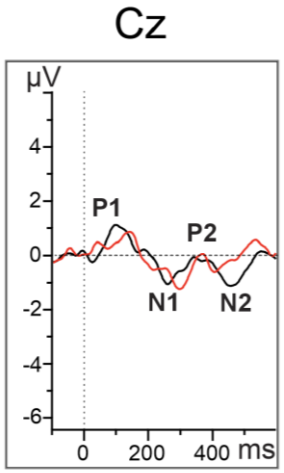

Figure 30. *The grand average of frontocentral auditory evoked potentials in response to /da/, in babble noise in quiet , in children with APD (red curve) and TD children (black curve).*

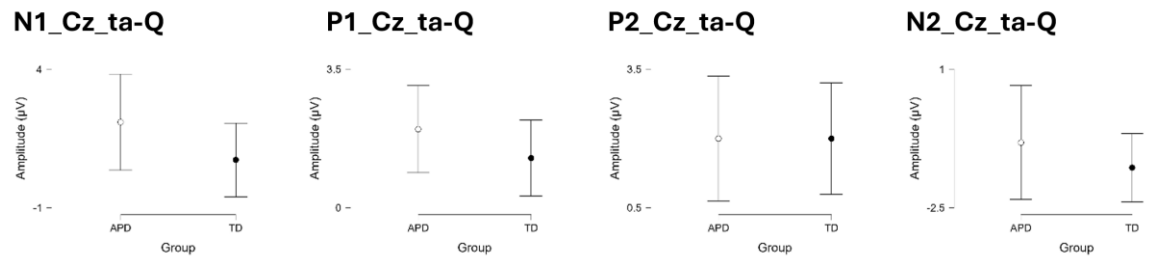

Figure 31. *The amplitude of frontocentral responses, recorded at Cz, in response to /da/, in quiet, in TD and APD groups.*

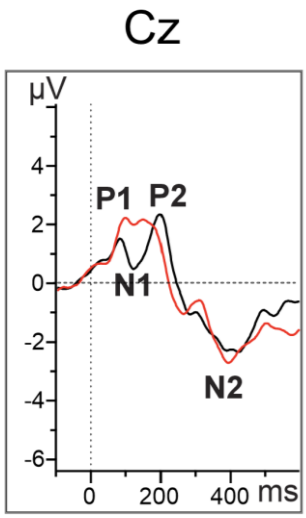

Figure 32. *The grand average of frontocentral auditory evoked potentials in response to /ta/, in quiet, in children with APD (red curve) and TD children (black curve).*

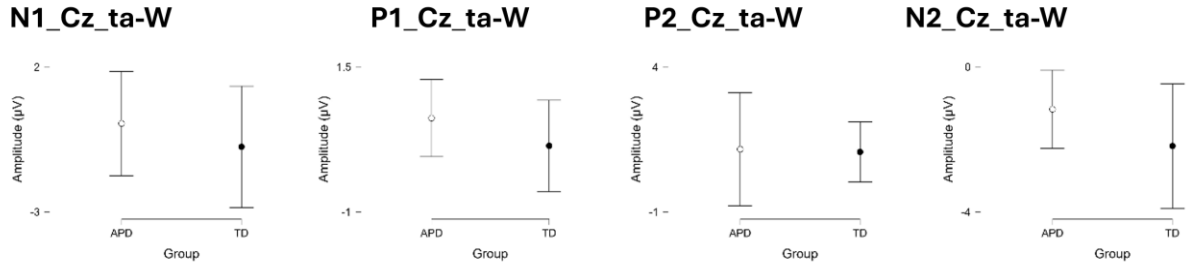

Figure 33. *The amplitude of frontocentral responses, recorded at Cz, in response to /ta/, in white noise, in TD and APD groups.*

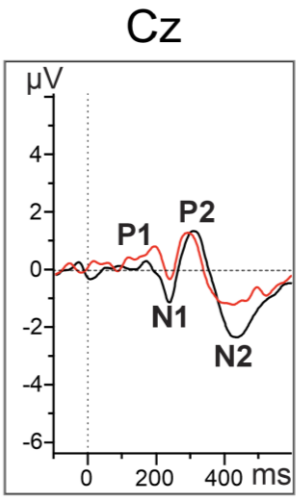

Figure 34. *The grand average of frontocentral auditory evoked potentials in response to /ta/, in white noise, in children with APD (red curve) and TD children (black curve).*

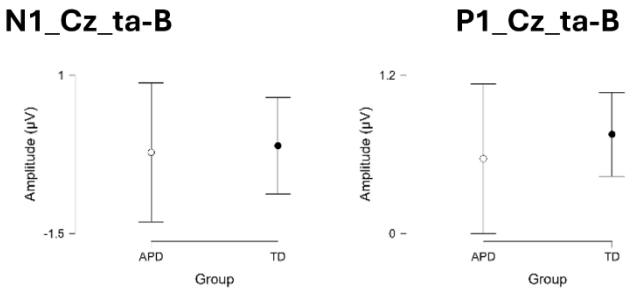

Figure 35. *The amplitude of frontocentral responses, recorded at Cz, in response to /ta/, in babble noise, in TD and APD groups.*

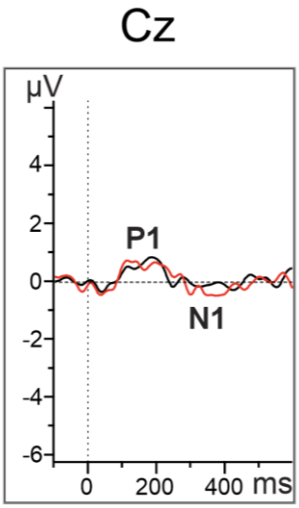

Figure 36. *The grand average of frontocentral auditory evoked potentials in response to /ta/, in babble noise, in children with APD (red curve) and TD children (black curve).*

6.3.3. Results of Latency, T-complex

The results of Mann-Whitney test showed that there was significant difference in latency of Ta and Tb, between APD and TD children (Table 3 of appendix). Tb had a shorter latency at electrode T7 using 1kHz in babble noise ($p < 0.05$) in APD group than TD group. Ta showed a longer latency at electrode T7 ($p = 0.011$) and at electrode T8 (*near to significant,* $p = 0.057$) in response to speech sound /ta/ in the presence of babble noise (Figure 37-54).

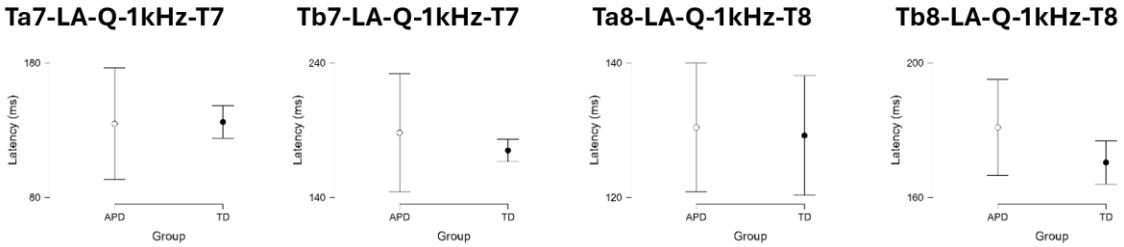

Figure 11 *The latency of Ta and Tb in response to 1kHz in quiet, recorder at T7 and T8, in children with APD and TD.*

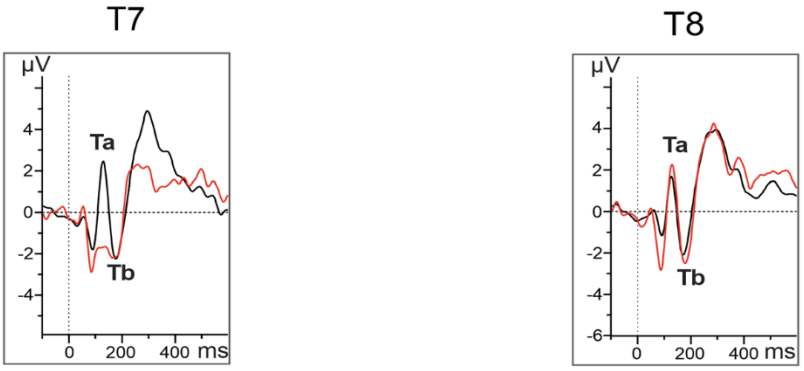

Figure 38. *The grand average of temporal auditory evoked potentials in response to 1kHz, in quiet, in children with APD (red curve) and TD children (black curve).*

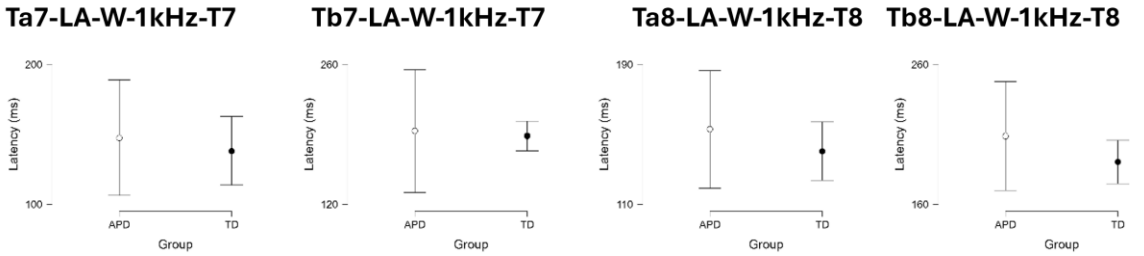

Figure 12 *The latency of Ta and Tb in response to 1kHz in white noise, recorder at T7 and T8, in children with APD and TD.*

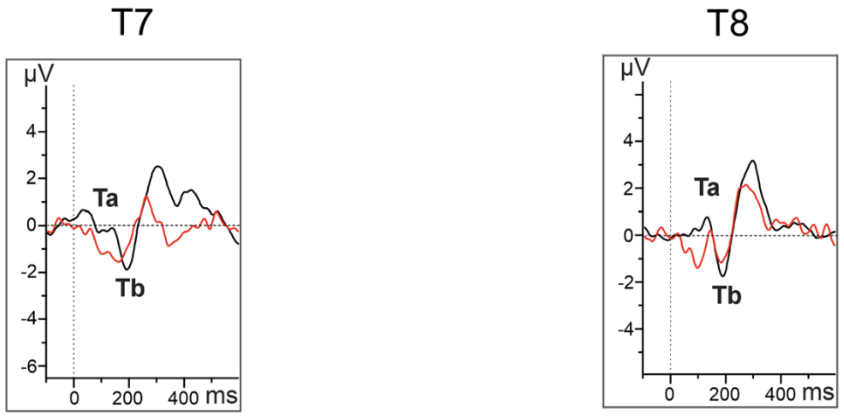

Figure 40. *The grand average of temporal auditory evoked potentials in response to 1kHz, in white noise, in children with APD (red curve) and TD children (black curve).*

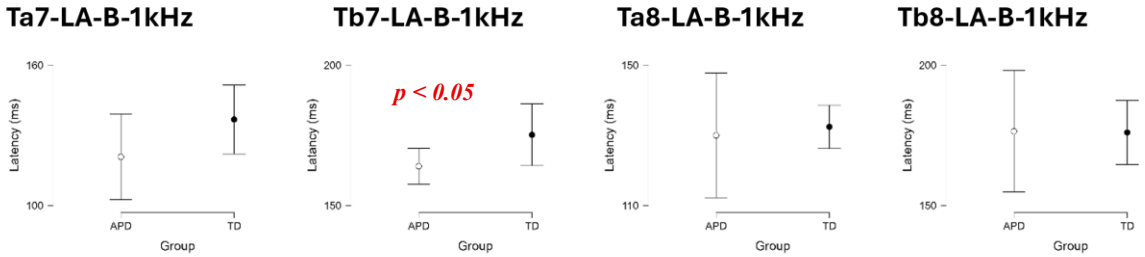

Figure 13*The latency of Ta and Tb in response to 1kHz in babble noise, recorder at T7 and T8, in children with APD and TD. Tb had a shorter latency in APD than TD.*

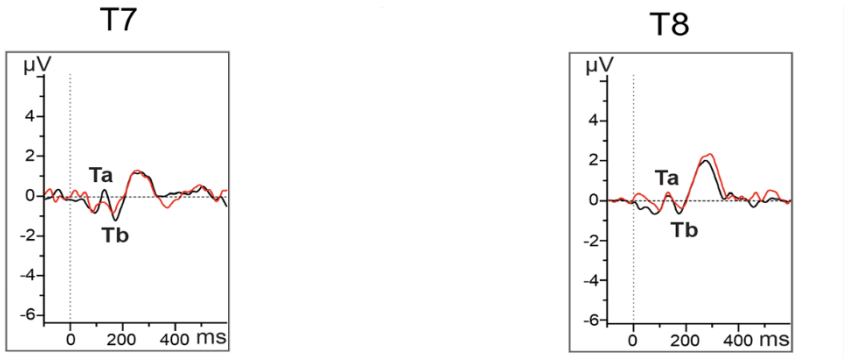

Figure 14*The latency of Ta and Tb in response to 1kHz in babble noise, recorder at T7 and T8, in children with APD and TD.*

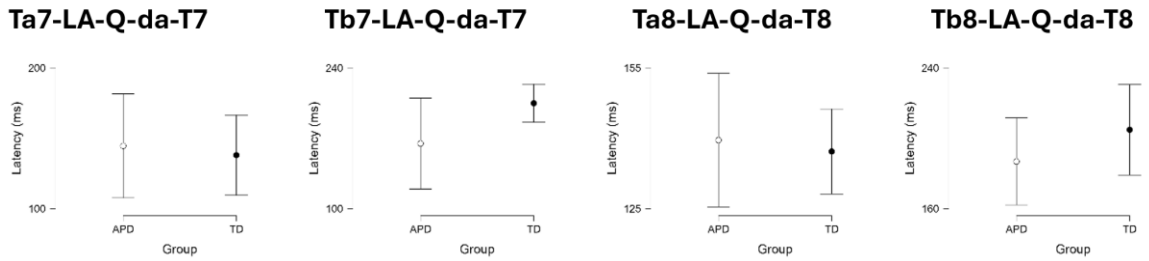

Figure 15 *The latency of Ta and Tb in response to /da/ in quiet, recorder at T7 and T8, in children with APD and TD children.*

Figure 44. *The grand average of temporal auditory evoked potentials in response to /da/, in quiet, in children with APD (red curve) and TD children (black curve).*

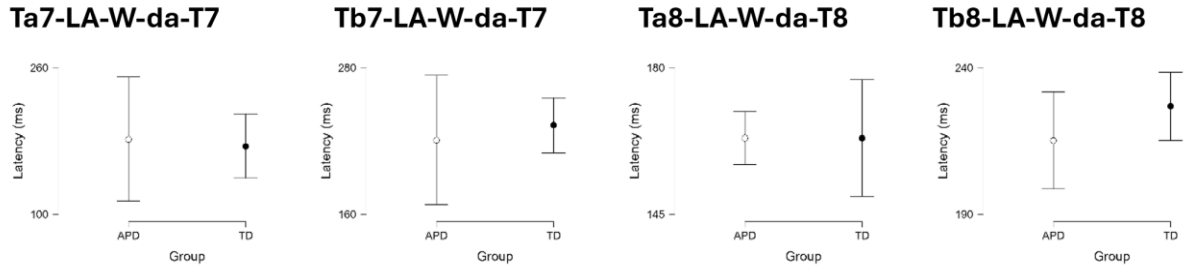

Figure 16 *The latency of Ta and Tb in response to /da/ in white noise, recorder at T7 and T8, in children with APD and TD children.*

Figure 46. *The grand average of temporal auditory evoked potentials in response to /da/, white noise, in children with APD (red curve) and TD children (black curve).*

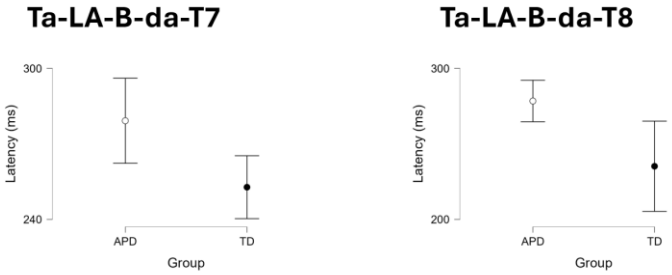

Figure 17 *The latency of Ta in response to /da/, in babble noise, recorder at T7 and T8, in children with APD and TD children.*

Figure 48. *The grand average of temporal auditory evoked potentials in response to /da/, in babble noise, in children with APD (red curve) and TD children (black curve).*

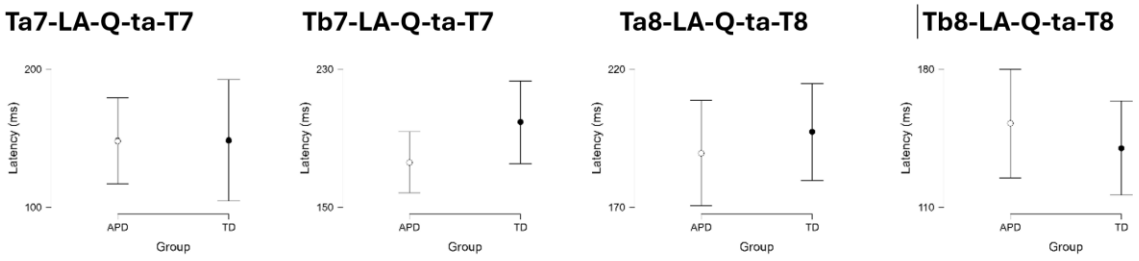

Figure 18 *The latency of Ta and Tb in response to /ta/, in quiet, recorder at T7 and T8, in children with APD and TD children.*

Figure 50. *The grand average of temporal auditory evoked potentials in response to /ta/, in quiet, in children with APD (red curve) and TD children (black curve).*

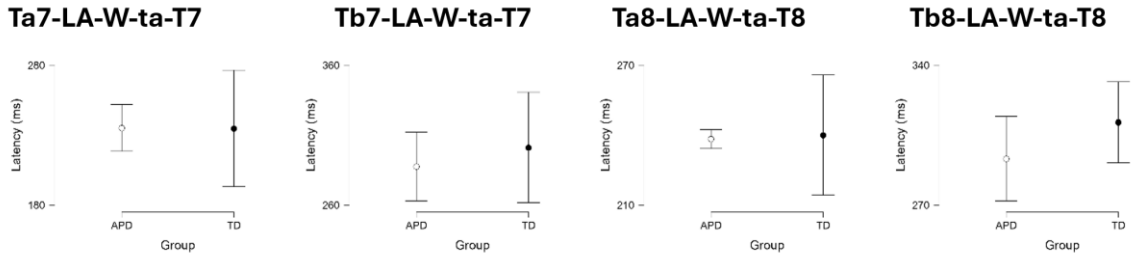

Figure 19 *The latency of Ta and Tb in response to /ta/, in white noise, recorder at T7 and T8, in children with APD and TD children.*

Figure 52. *The grand average of temporal auditory evoked potentials in response to /ta/, in white noise, in children with APD (red curve) and TD children (black curve).*

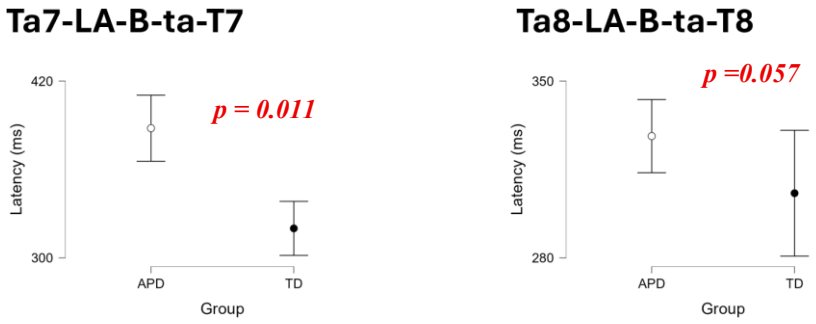

Figure 20 *The latency of Ta in response to /ta/, in babble noise, recorder at T7 and T8, in children with APD and TD children. Ta had a longer latency at T7 and T8 in APD than TD group.*

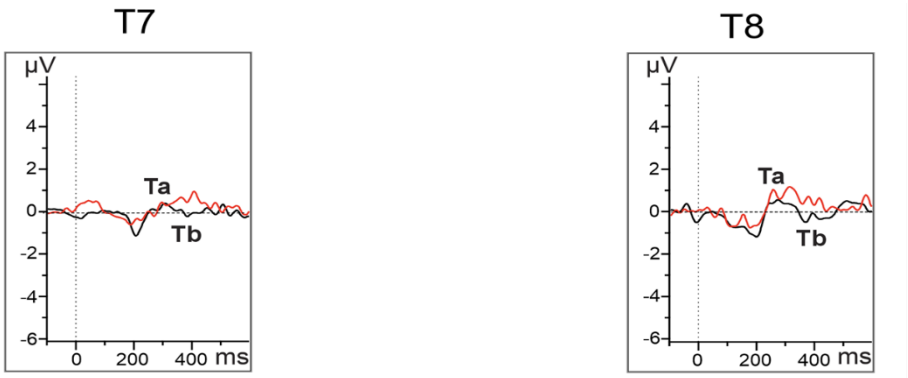

Figure 54. *The grand average of temporal auditory evoked potentials in response to /ta/, in babble noise, in children with APD (red curve) and TD children (black curve).*

6.3.4. Results of Latency, Frontocentral responses

Regarding the frontocentral responses, the significant difference was observed in latency of P1, N1, and N2. There was a longer latency of P1 and N1 in babble noise using /da/ ($p < 0.05$), and a shorted latency of N2 using 1kHz in the presence of white noise in APD versus TD group ($p < 0.05$) (Table 4 of appendix) (Figure 55-72).

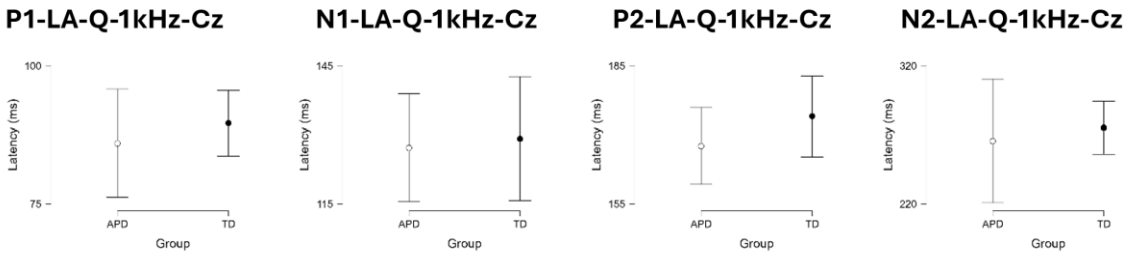

Figure 21 *The latency of frontocentral responses, in response to 1kHz, in quiet, recorder at Cz, in children with APD and TD children.*

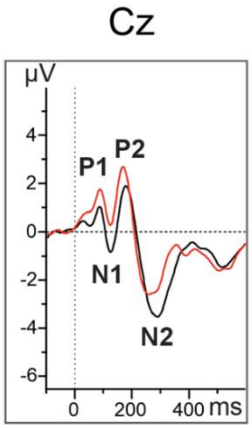

Figure 56. *The grand average of frontocentral auditory evoked potentials, in response to 1kHz, in quiet, in children with APD (red curve) and TD children (black curve).*

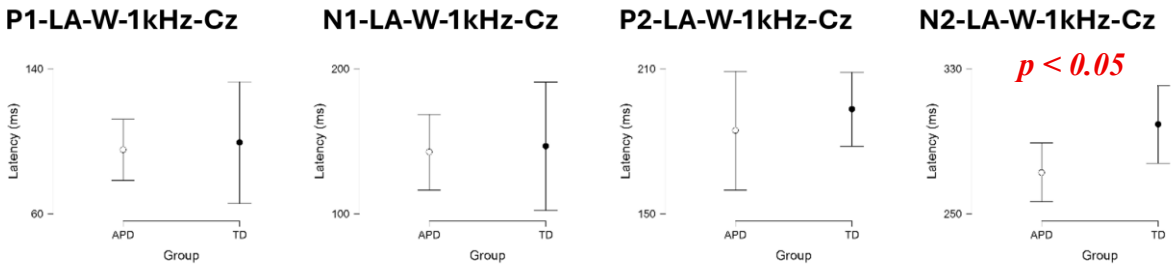

Figure 22 *The latency of frontocentral responses, in response to 1kHz, in white noise, recorder at Cz, in children with APD and TD children.*

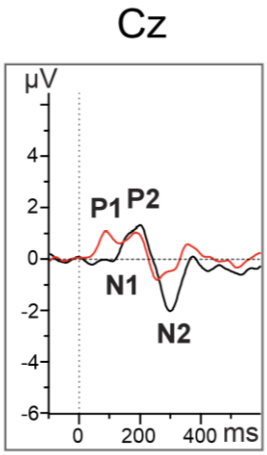

Figure 58. *The grand average of frontocentral auditory evoked potentials, in response to 1kHz, in white noise, in children with APD (red curve) and TD children (black curve).N2 has shorter latency in APD than TD.*

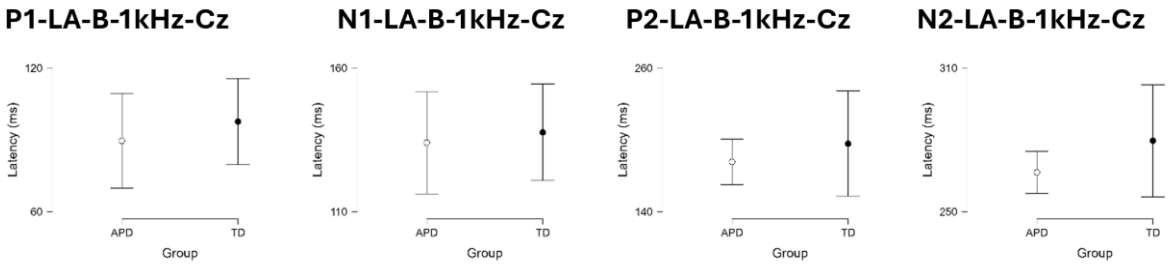

Figure 23 *The latency of frontocentral responses, in response to 1kHz, in babble noise, recorder at Cz, in children with APD and TD children.*

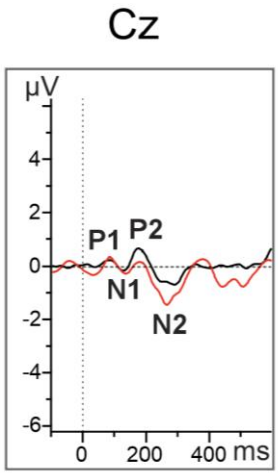

Figure 60. *The grand average of frontocentral auditory evoked potentials, in response to 1kHz, in babble noise, in children with APD (red curve) and TD children (black curve).*

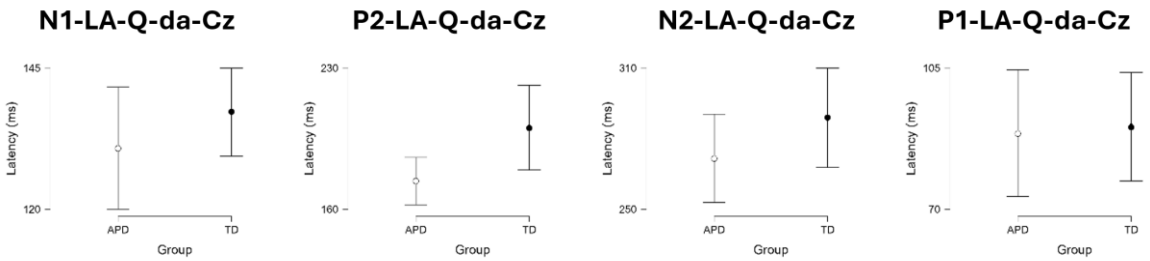

Figure 24 *The latency of frontocentral responses, in response to /da/, in quiet, recorder at Cz, in children with APD and TD children.*

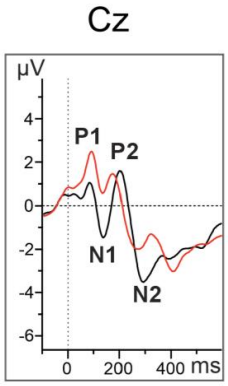

Figure 62. *The grand average of frontocentral auditory evoked potentials, in response to /da/, in quiet, in children with APD (red curve) and TD children (black curve).*

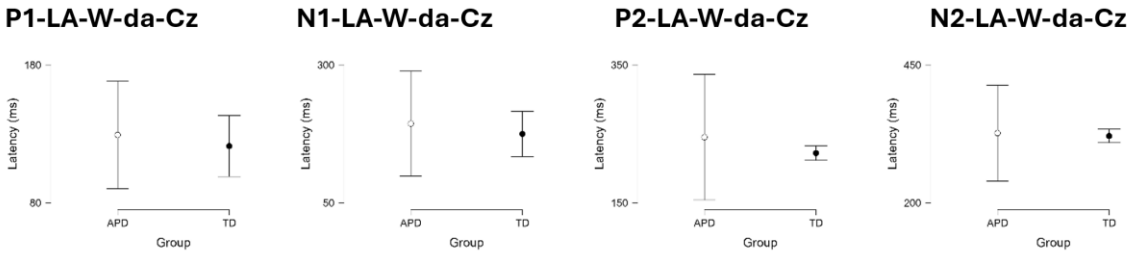

Figure 25 *The latency of frontocentral responses, in response to /da/, in white noise, recorder at Cz, in children with APD and TD children.*

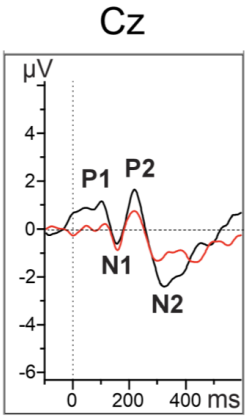

Figure 64. *The grand average of frontocentral auditory evoked potentials, in response to /da/, in white noise, in children with APD (red curve) and TD children (black curve).*

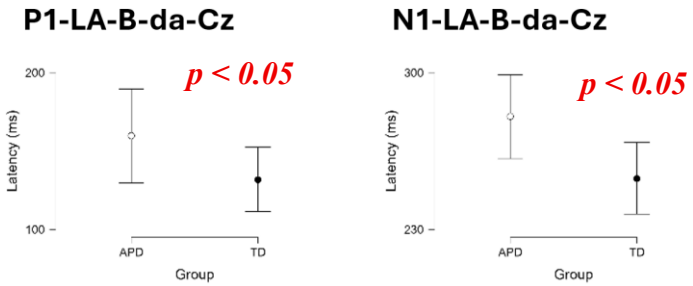

Figure 26 *The latency of frontocentral responses, in response to /da/, in babble noise, recorder at Cz, in children with APD and TD children.*

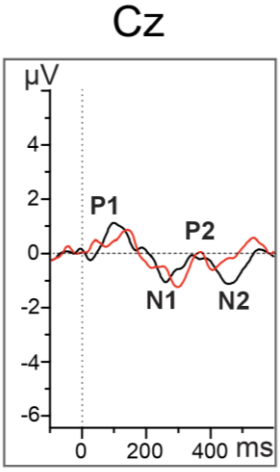

Figure 66. *The grand average of frontocentral auditory evoked potentials, in response to /da/, in babble noise, in children with APD (red curve) and TD children (black curve).*

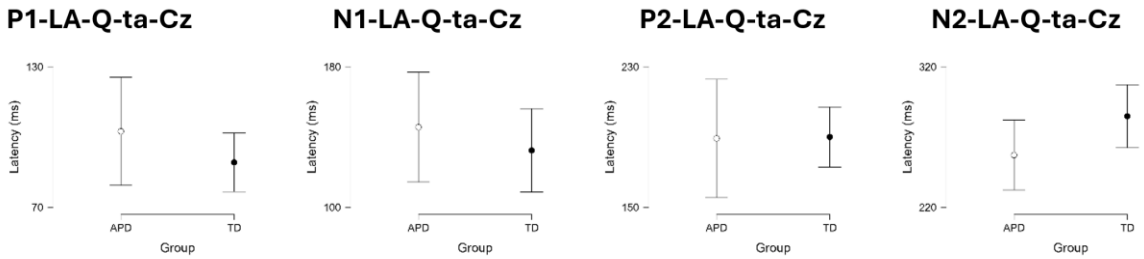

Figure 27 *The latency of frontocentral responses, in response to /ta/, in quiet, recorder at Cz, in children with APD and TD children.*

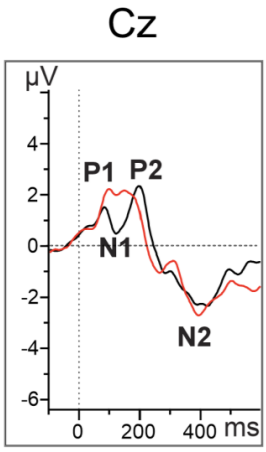

Figure 68. *The grand average of frontocentral auditory evoked potentials, in response to /ta/, in quiet, in children with APD (red curve) and TD children (black curve).*

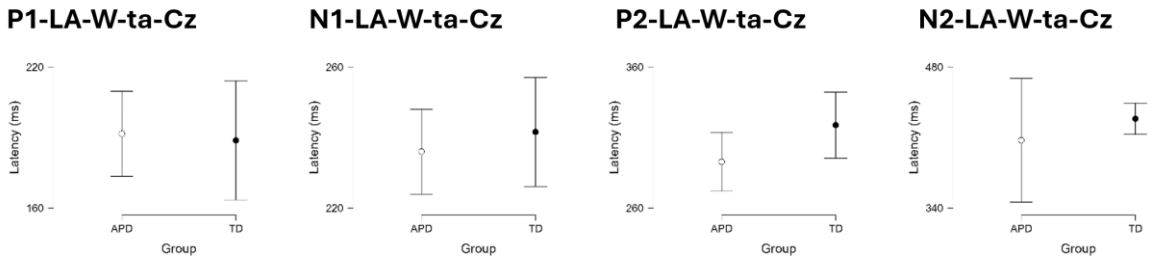

Figure 28 *The latency of frontocentral responses, in response to /ta/, in white noise, recorder at Cz, in children with APD and TD children.*

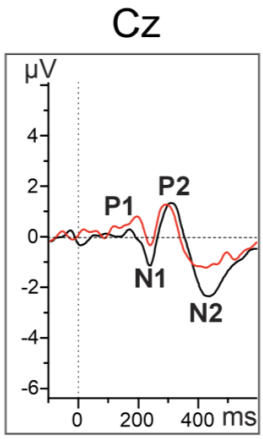

Figure 70. *The grand average of frontocentral auditory evoked potentials, in response to /ta/, in white noise, in children with APD (red curve) and TD children (black curve).*

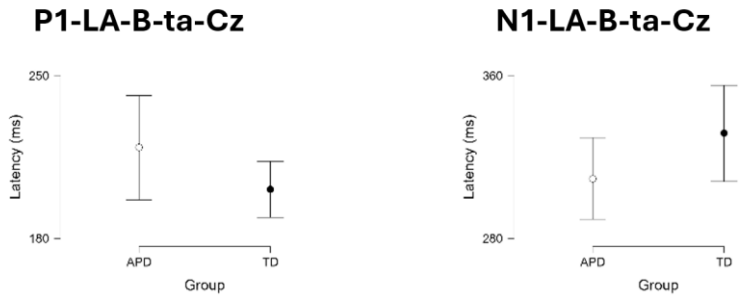

Figure 29 *The latency of frontocentral responses, in response to /ta/, in babble noise, recorder at Cz, in children with APD and TD children.*

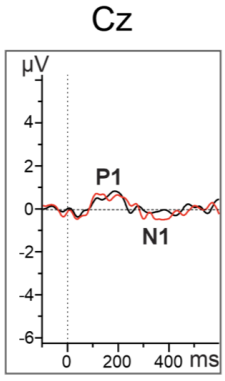

Figure 72. *The grand average of frontocentral auditory evoked potentials, in response to /ta/, in babble noise, in children with APD (red curve) and TD children (black curve).*

6.4. Results of the second objective (VOT experiment or stimuli effect)

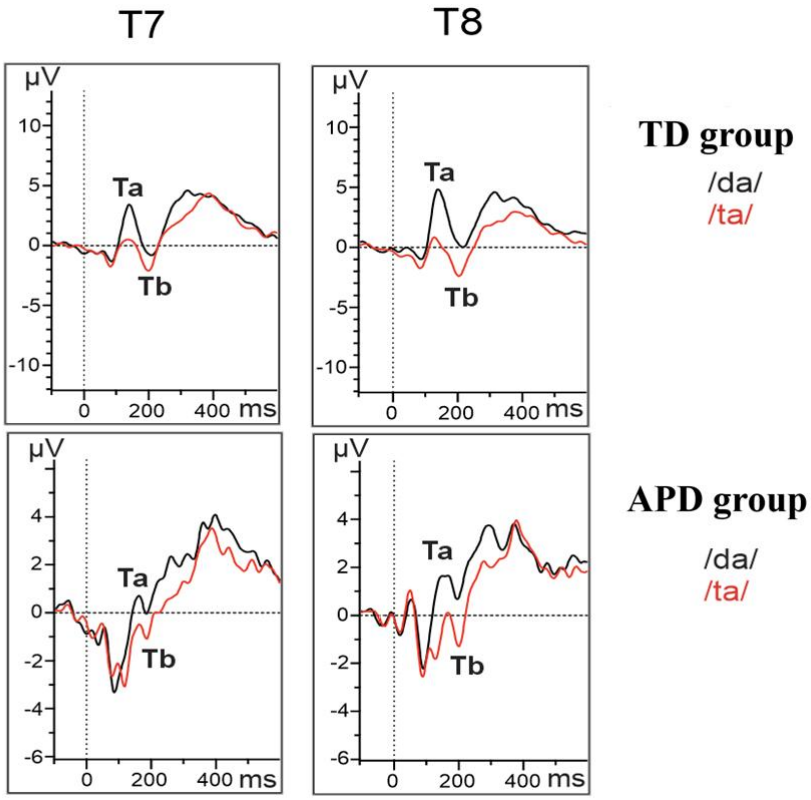

Figure 73. *The Ta and Tb responses to syllable /da/ and /ta/, Black curve is response to /da/ and red curve is response to /ta/, in quiet, in normal group (upper graph) and APD group (lower graph). Tb showed significantly larger response, at T8, to /ta/ than /da/ in TD versus APD group.*

6.4.1. Amplitude

In TD group, Wilcoxon test results showed marginally significant effect of Stimuli on amplitude of Ta and Tb. There was a trend (near to significant) of larger amplitude of Ta at electrodes T8 and T7 to speech sound /da/ than speech sound /ta/ ($p = 0.063$). Also, there was a marginally larger amplitude of Tb at electrode T8, to /ta/ versus /da/ ($p = 0.063$) (Figure 73 and 74, table 5 of appendix). Results in APD group showed a marginally significant effect of Stimuli only on amplitude of Ta (Table 8). There was a near to significant or a trend of larger amplitude of Ta to speech sound /da/ than speech sound /ta/ at electrode T8 ($p = 0.063$) (Figure 73 and 74, table 7 of appendix).

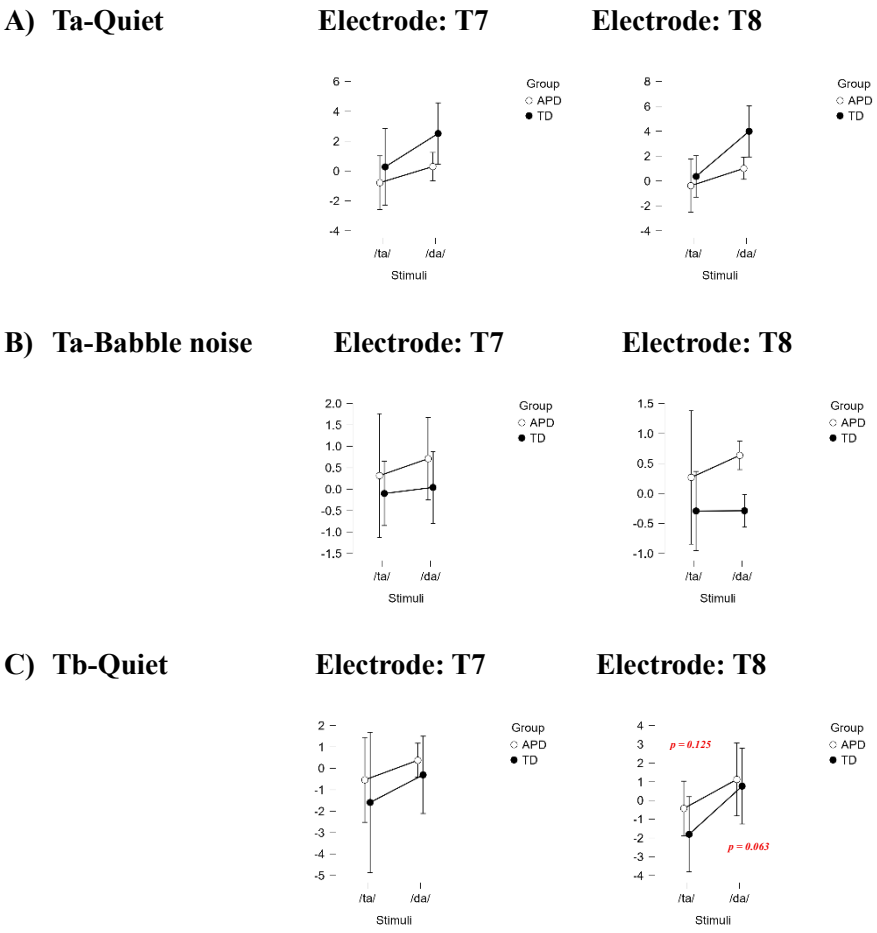

Figure 74. *The amplitude of Ta (A and B) and Tb (C), in response to /da/ and /ta/ in APD and TD group, in Quiet (Q) and Babble noise (B). There was a larger amplitude of Ta to speech sound /da/, than speech sound /ta/ at electrode T7 and T8. Tb has a larger amplitude to speech sound /ta/ than speech sound /da/ at electrode T8.*

6.4.2. Latency

In TD group, results showed a marginally significant effect of stimuli only in noisy condition and only for Ta component. There was a near to significant or a trend of longer latency of Ta in response to speech sound /ta/ than speech sound /da/, in babble noise (*p=0.063*) (Figure 76, table 6 of appendix). Results of APD group showed marginally significant effect of stimuli only in noisy condition. There was a near to significant or a trend of longer latency of Ta in response to speech sound /ta/ than speech sound /da/ (*p=0.063*) (Figure 76, Table 8 of appendix).

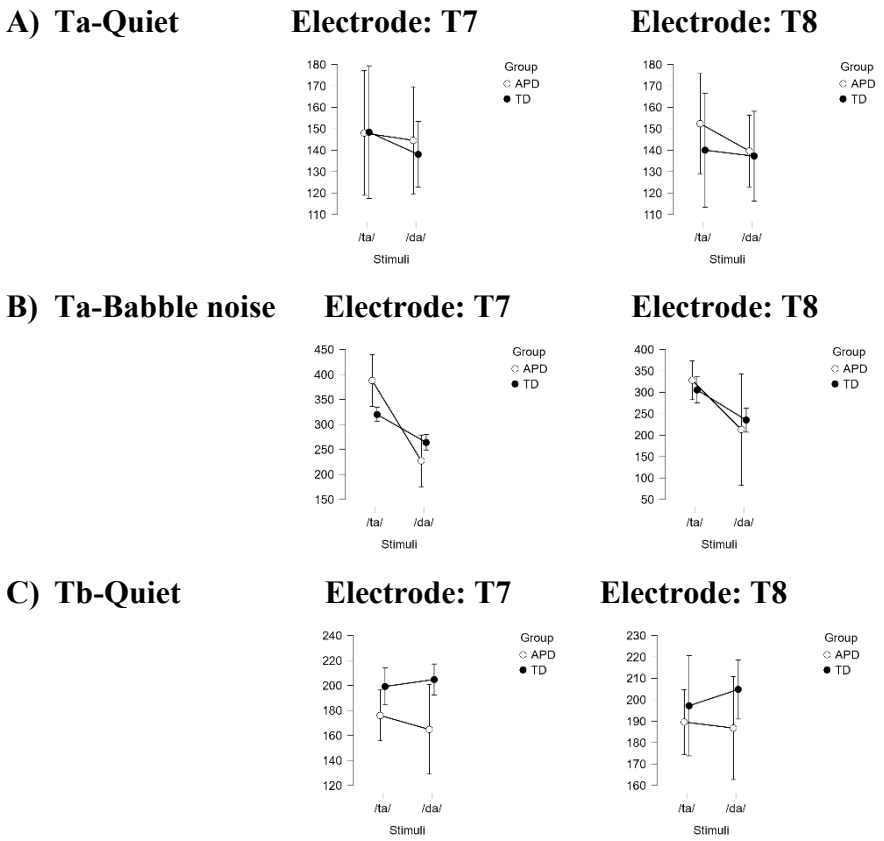

Figure 75. *The latency of Ta (A and B), and Tb (C), in response to /da/ and /ta/, in APD and TD groups. There was a longer latency of Ta to speech sound /ta/, than speech sound /da/ at electrode T7 and T8, in babble noise.*

6.5. Behavioral evaluation

The results of Mann-Whitney test showed no differences in accuracy (AC) and reaction time (RT) in quiet (Q) and in babble noise (B), between TD and APD groups (Table 4, Figure 18,19). Although there was a pattern of lower accuracy and shorter reaction time in children with APD versus TD children, results did not show significant differences between APD and TD groups in behavioral measurements.

Table 4. *The results of Independent Samples T-Test to investigate the differences in AC and RT between TD and APD groups in Quiet (Q) and Babble noise (B).*

	U	df	p
AC-da-Q	15.500		0.209
AC-ta-Q	15.000		0.600
AC-da-B	11.500		1.000
AC-da-B	14.000		1.000
RT-da-B	16.000		0.548
RT-da-Q	16.000		0.548
RT-ta-Q	15.000		0.690
RT-ta-B	20.000		0.151

Note. Mann-Whitney U test.

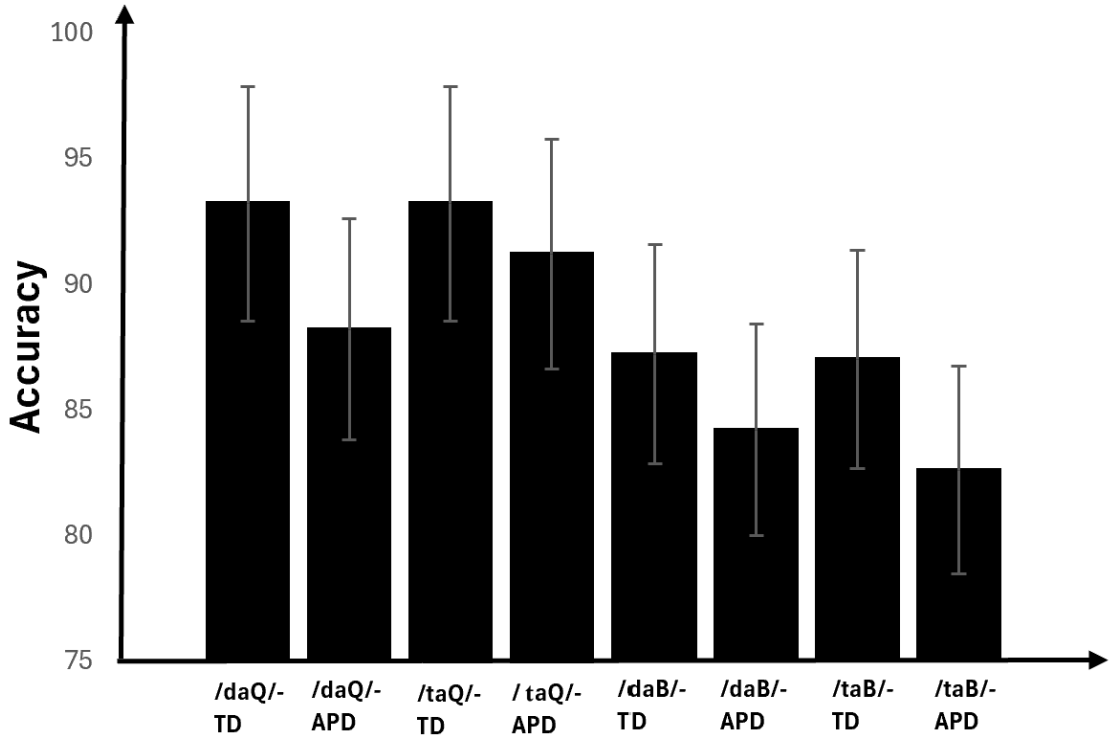

Figure 76. *The average of accuracy (ACC) to /da/ and /ta/, in quiet (Q) and babble noise (B), in children with APD and TD.*

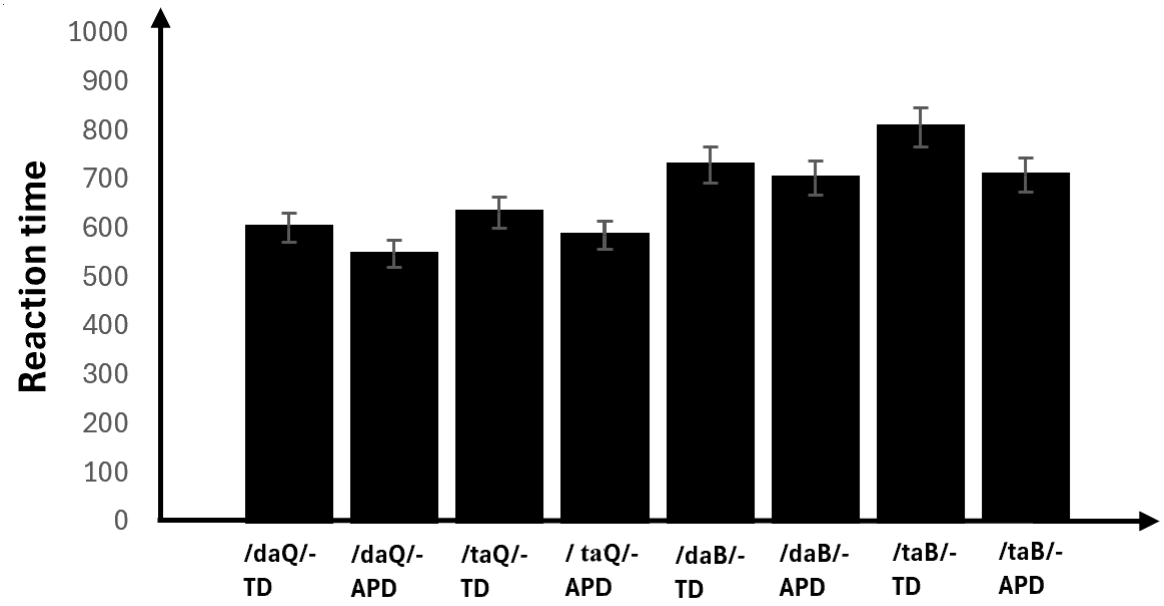

Figure 77.30 *The average of Reaction time (RT) to /da/ and /ta/, in quiet (Q) and babble noise (B), in children with APD and TD.*

6.6. Summary of findings

The summary of finding of noise and stimuli studies have been inserted in table 5.

Table 5. *The summary of finding collected from first and second objectives of the APD study.*

Goal			Results
Investigation, APD-Normal comparison in noise and quiet		**APD vs. TD**	With 1kHz, Ta at electrode T7, and there was shorter latency of Tb at electrode T7 in babble noise and N2 in white noise, in APD versus TD. P1 in white noise had a significantly larger amplitude in APD group than TD children, in white noise. Also, N2 had a near to significant larger amplitude in babble noise, and a smaller amplitude in white noise in APD group than TD children. With /da/, there were **no significant differences between APD and normal groups in amplitude of AEPs**. However, with the same stimuli, there was a longer latency of P1 and N1 in babble noise in APD than TD. With /ta/, Ta had longer latency at electrode T7 and shorter at electrode T8, using /ta/ in babble noise, in APD than TDs group. There was no significant difference in temporal response's amplitudes between APD and TD. Additionally, there was **no significant difference in amplitude and latency of frontocentral responses** between TD and APD.
Investigation, Effect of VOT patterns in APD and Normal	**AEP result**	**TD**	There was significantly larger Tb to speech sound /ta/ than speech sound /da/ at electrode T8, and there was a larger Ta to speech sound /da/ than speech sound /ta/ at electrodes T7 and T8. There was a longer latency of Ta to speech sound /ta/ than /da/ in noise. There was no significant difference in temporal response between speech sound /ta/ and speech sound /da/. However, there was only a lager Ta to speech sound /da/ than speech sound /ta/ at electrode T8.
		APD	There was longer latency of Ta to speech sound /ta/ than /da/ in noise.
	Behavioral results	**APD vs. TD**	There **were no significant differences between accuracy and reaction time between** TD and APD groups.

6.7. Discussion

The present investigation of cortical auditory evoked potentials in children with APD should be considered exploratory. This study builds upon patterns observed in our previous investigations in typically developing adults and children, which examined the effects of noise and stimulus characteristics on frontocentral and temporal auditory responses (Ahmadi et al., 2026a accepted, 2026b submitted, 2026c submitted and 2026d submitted). Given the challenges associated with recruiting children with clinically diagnosed APD and the strict diagnostic criteria required for inclusion, the current study was conducted with a limited sample size. Therefore, the findings should be interpreted as preliminary and hypothesis-generating, providing initial insights into potential differences in cortical auditory processing between children with APD and typically developing peers, and helping to inform directions for future research.

The current study compared the frontocentral and temporal auditory responses between children with APD and TD children under quiet and in noisy listening conditions using both speech and non-speech stimuli. In addition, the neural representation of VOTs, indexed by T-complex, was examined in children with APD and compared with that of TD children. Behavioral performance, including accuracy and reaction time during a speech discrimination task, was also compared between the two groups. In the following sections, the results related to the first objective (AEPs in noise) and second objective (VOT effect) are discussed.

6.7.1. AEPs in noise and quiet

Both amplitude and latency measures revealed differences between APD and TD groups, however, atypical patterns were not observed for all of the AEPs component like P2 (Figure 21-34). The absence of significant group differences in P2 are partially related to developmental factors. Previous research has showen that components such as N1 and P2 are not fully mature in children, which may result in less stable or efficient cortical responses potentially masking group differences (Ubiali et al., 2025). This interpretation is consistent with previous studies reporting that group differences in children are often more pronounced in developmentally sensitive componenets such as P1 and N2 (Hussain et al., 2022; Morlet et al., 2019). Nevertheless, these findings should be interpreted cautiously given the limited statistical power of the present study. Due to the small sample size, the study may have failed to detect subtle group differences. Therefore, further studies with larger samples are required to confirm these findings.

Contrary to our initial hypothesis that T-complex and frontocentral responses would show distinct patterns, both responses demonstrated atypical patterns in children with APD compared to TD children, including larger amplitudes and shorter or longer latencies. These differences were observed in at least one component within each response type, suggesting that atypical auditory processing in APD may affect multiple levels of cortical auditory processing rather than being restricted to a specific cortical response type. These findings also do not clearly support the idea that the T-complex is a more sensitive measure for identifying auditory processing impairments compared to frontocentral responses. However, given the limited sample size, further research is required to confirm this observation.

The results have been elaborated for amplitude and latency measurements, in more details in the following section.

6.7.2. Amplitude results

Interestingly, the strongest group differences in amplitude were observed for the 1 kHz stimulus rather than for speech stimuli. Response to pure tone versus speech stimuli, primarily reflect the more fundamental encoding mechanisms of auditory stimuli within the auditory cortex (Näätänen & Picton, 1987). If children with APD exhibit alterations in early auditory neural encoding, this effect may be more detectable with simple acoutic stimuli such as pure tone (Dillon & Cameron, 2021b). In contrast, speech stimuli contain multiple acoutical and linguistical cues that engage broader neural networks involved in speech perception (Hickok & Poeppel, 2007, 2015). Accordingly, it can scpeculate that recruiting additional cortical areas may allow children with APD to partially compensate for auditory processing difficulties, thereby reducing observables group differences. More studies using speech and non-speech stimuli is needed to confirm these findings.

Among the frontocentral responses, the P1 component showed a larger amplitude in the APD group compared with the TD group in the white noise condition when using 1 kHz stimulus. Similarly, the N2 component showed a smaller amplitude in white noise but a larger amplitude in babble noise in children with APD compared with TD children for the 1 kHz stimulus.

Larger amplitudes of AEPs in noisy conditions in children with APD compared to TD children have been observed previpousely (Hussain et al., 2022; Morlet et al., 2019). Morlet et el, (2019) reported larger peak-to-peak P2-N2 amplitudes in children with APD relative to TD children. Increased AEP amplitudes in noise in APD group have been interpreted as reflecting impairments in inhibitory processing mechanisms (Anderson, Chandrasekaran, et al., 2010). AEP amplitude is linked to the number of activated neurons or synapses and to the synchronicity of neurons contributing to the evoked responses (Eggermont, 1988). Top-down inhibition normally modulates sensory inputs by supressing irrelevant neural activity, thereby improving the efficiency of auditory processing (Allman et al., 1985; Dustman et al., 1981). In individuals with typical auditory processing, effective inhibition processing may reduce unnecessary inputs and enhance the efficiency of neural responses during speech perception in noise (Anderson, Chandrasekaran, et al., 2010; Morlet et al., 2019). In contrast, children with APD may have reduced inhibitory control over irrelevant auditory inputs (Anderson, Chandrasekaran, et al., 2010; Morlet et al., 2019). As a result, greater neural activation may occur when processing sounds in noisy environments, leading to larger cortical responses ampltiude. The observed increased neural activity may also reflect greater processing demands

placed on the auditory system of children with APD when attempting to segregate targer sounds from background noise (Hussain et al., 2022).

One of the significant findings of the present study is the different pattern of changes in N2 amplitude observed in white noise compared with babble noise. Specifically, N2 showed smaller amplitude in white noise but larger amplitude in babble noise in the APD group relative to the TD group. To our knowledge, this pattern has not been previously reported, as earlier studies investigating auditory evoked potentials in children with APD have not examined responses under different types of background noise. One possible explanation may be related to the fundamental differences between babble noise and white noise. White noise is a strong acoustical maksing as it contains a relatively flat spectrum across the frequencies, which can strongly interfere with the neural representation of auditory signals and suppress sensory responses (Kaplan-Neeman et al., 2006). In individuals with impaired auditory processing, such as children with APD, this strong acoustic masking may further challenge auditory encoding mechanisms, potentially leading to greater suppression of neural responses (Anderson, Chandrasekaran, et al., 2010). In contrast, multi-talker babble noise posses informational masking effect addtion to energetic masking (Brungart, 2001). Informational masking involves higher-level perceptual and cognitive processes, including attentional and inhibitory mechanisms (Brungart, 2001). Consequently, processing speech signals in babble noise requires greater engagement of top-down auditory processing mechanisms. In populations with reduced inhibitory control, such as children with APD, inefficient suppression of competing auditory information may lead to increased neural activity in response to background sounds. This increased neural activity may be reflected as larger AEP amplitudes in babble noise compared with white noise. Accordingly, the larger N2 amplitudes observed in babble noise may reflect greater neural effort associated with processing competing auditory inputs in children with APD.

With respect to temporal responses, the present study revealed smaller Ta amplitudes in response to the 1 kHz stimulus in white noise in the APD group compared with the TD group. Although no previous studies have specifically examined T-complex components in children with APD, similar explanations proposed for reducd frontocentral AEP amplitudes in noise may also apply to the Ta (Jenna Cunningham et al., 2001; J.-H. Han et al., 2020). It has been mentioned that more reduced amplitude of AEPs in noise in children with listening difficulties may show the less efficient of auditory processing leading to incomplete or ‘’blurred’’ representaiton of acoustic features of sppech in noise.

It is to be noted that significant amplitude differences between groups were observed only under noisy listening conditions. This pattern is consistent with previous AEP studies reporting that auditory processing deficits in children with APD are more evident in the presence of background noise (Anderson, Skoe, et al., 2010; Cunningham et al., 2002; Hussain et al., 2022; Russo et al., 2005). Noisy listening environments impose greater demands on auditory processing and require more precise neural synchronization to successfully encode and segregate target sounds from competing background signals. Consequently, auditory dysfunction in children with APD may become more apparent under these challenging listening conditions (Hussain et al., 2022). Collectively, these findings support the notion that auditory processing in children with APD is less efficient under noisy listening conditions compared with their typically developing peers (Anderson, Skoe, et al., 2010; Cunningham et al., 2002; Russo et al., 2005). Children with APD may require greater neural effort and longer processing time to encode acoustic stimuli and may have reduced access to the cognitive and neural resources necessary for efficient sound processing in noisy environments (Hussain, 2022; Liasis, 2003).

6.7.3. Latency results

Latency measures also revealed several differences between the APD and TD groups across noisy listening conditions. In response to the 1 kHz stimulus, children with APD showed shorter latencies of Tb in babble noise and N2 in white noise compared with TD children. In response to the /da/ stimulus, longer latencies of P1 and N1 were observed in babble noise in the APD group relative to the TD group. For the /ta/ stimulus presented in babble noise, the Ta component showed a longer latency at electrode T7 and T8 in children with APD compared with TD children.

Prolonged AEP latencies in children with APD under noisy conditions have been reported in previous studies (Hussain et al., 2022). Longer cortical response latencies may reflect slower neural conduction within auditory pathways, suggesting that encoding and representation of auditory stimuli require more processing time in children with APD (Anderson, Chandrasekaran, et al., 2010).

The shorter latencies observed for Tb and N2 in the present study have not been widely reported in previous investigations of cortical auditory responses in APD populations. As this is the first study examining the T-complex in children with APD, direct comparisons with previous research remain limited. Nevertheless, atypically short latencies of T-complex

components have been reported in other neurodevelopmental conditions such as dyslexia (Taylor et al., 2003). These findings have been interpreted as reflecting superficial or inefficient cortical processing, potentially leading to inaccurate sensory encoding and impaired representation of auditory stimuli. Accordingly, the shorter latencies of N2 observed in the present study may reflect atypical or inefficient neural processing of auditory stimuli in children with APD under noisy listening conditions. Another possible explanation for reduced latency in noise in the APD group may relate to differences in efferent auditory processing such as observed differences in non-musicians compared to musicians (Rabelo et al., 2015; Tomchik & Lu, 2006). Musicians typically exhibit better speech perception in noise relative to non-musicians (Anderson et al., 2013; Benjamin Rich Zendel et al., 2015). Previous studies examining auditory processing in musicians have demonstrated modulation of cortical responses through efferent mechanisms (Rabelo et al., 2015; Tomchik & Lu, 2006). Rabelo et al. (2015) recorded P300 responses in musicians and non-musicians and found that response latencies increased in noise for musicians but not for non-musicians. The authors suggested that activation of the efferent auditory system may reduce the firing rate of afferent neurons in the presence of background noise, thereby increasing response latency (Rabelo et al., 2015; Tomchik & Lu, 2006). By analogy, reduced or less effective efferent suppression in children with APD may limit the expected latency prolongation in noisy conditions, resulting in atypically shorter latencies compared with typically developing peers.

6.7.4. Stimuli effect

The results related to the second objective of the study indicated that both groups showed a marginal significant effect, or a trend toward a stimulus effect, on T-complex components (Figure 25). However, the pattern of stimulus-related modulation differed between the APD and TD groups. In the APD group, only the Ta component at electrode T8 showed a marginal difference or trend between the /ta/ and /da/ stimuli, whereas the TD group showed a trend of encoding of long and short VOTs through Ta responses in both hemispheres and Tb responses at electrode T8. These findings may support previous research showing the impairments of encoding short and long VOTs in children with APD (Hestvik et al., 2023). Hestvik et al. (2023) recorded the mismatch negativity to [dæ] and [tæ] syllables in groups of TD and APD children. The results showed reduced MMN amplitude in the APD group, suggesting impaired neural representation of phonetic contrasts. The authors interpreted these findings as supporting a bottom-up sensory deficit model of APD, in which auditory processing

impairments originate at early sensory stages rather than primarily reflecting higher-level cognitive dysfunction (Hestvik et al., 2023).

Moreover, the trend toward rightward lateralization of Tb responses to the /ta/ stimulus compared with the /da/ stimulus observed in the TD group is consistent with previous findings suggesting that the right hemisphere is more sensitive to slower temporal acoustic changes (Sandmann et al., 2007; Segalowitz & Cohen, 1989; Steinschneider et al., 1999). Hemispheric specialization for temporal processing has been shown to emerge relatively early during development (Clunies-Ross, 2020; K. L. Clunies-Ross et al., 2015; Clunies-Ross et al., 2018). The possible absence of rightward lateralization of VOT representation in children with APD may therefore reflect immaturity or atypical development of temporal processing mechanisms within higher-level auditory cortical regions, particularly within the superior temporal gyrus. Reduced hemispheric specialization for speech processing has also been reported in other neurodevelopmental conditions such as dyslexia (Giraud et al., 2005; Giraud et al., 2008). Considering the overlapping symptoms between dyslexia and APD (de Wit et al., 2018), it may be palusible to observe an absence of typical lateralization patterns in the present APD group. However, given that this is the first study reporting such findings in children with APD, further research will be necessary to confirm and better understand these patterns.

6.7.5. Behavioral results

Although visual inspection of the behavioral data may suggest that children with APD tend to show lower accuracy and shorter reaction time compared with TD children, these differences did not reach statistical significance. However, alongside the atypical neural findings, visually observed differences in behavioral performance between children with APD and TD children may indicate a trend toward deficits in VOT representation and auditory discrimination that were not fully captured by behavioral measures in the current sample. This may also reflect limitations of behavioral measures, which can be influenced by various non-auditory factors such as attention, motivation, and task strategies, potentially reducing their sensitivity to underlying neural processing differences (Alanazi, 2023; Dillon & Cameron, 2021a). Another possible explanation is the limited statistical power associated with the small sample size in the present study.

Given the exploratory nature of the study and the small sample size, additional research with larger participant groups will be necessary to clarify the relationship between neural encoding of speech cues and behavioral performance in children with APD.

6.8. Limitation

Several limitations of the present study should be acknowledged. First, the sample size was small (n = 5 per group), which substantially limited statistical power. This limited power likely contributed to the presence of near-significant findings and non-significant results despite observable trends and moderate effect sizes. With such a small sample, the probability of detecting true effects is reduced, increasing the risk of Type II error. Therefore, the lack of statistical significance in some comparisons should be interpreted cautiously and not necessarily as evidence of absence of group differences. Replication with larger samples is necessary to confirm the reliability and generalizability of these findings.

Finally, some auditory evoked potential components could not be reliably identified in all participants and conditions and were therefore excluded from the analyses. While this approach ensured that the analyses were based on clearly identifiable responses, it also reduced the number of observations available for statistical comparison. Moreover, the duration of the experimental session was relatively long, which may have increased fatigue, particularly in pediatric participants. Fatigue can increase neural response variability and reduce sustained engagement, potentially affecting the stability of cortical auditory measures.

6.9. Conclusion

The present study demonstrated distinct effects of background noise on cortical auditory evoked potentials in children with APD compared with typically developing peers. Differences between groups were observed in both amplitude and latency measures of several AEP components under noisy listening conditions. These findings suggest that auditory processing in children with APD may be less efficient in the presence of background noise.

In addition, the neural representation of voice onset time partially differed between the APD and TD groups. Children with APD did not exhibit strong typical lateralized processing of short and long VOT observed in TD children at the level of secondary auditory cortical responses. These findings may reflect atypical or immature temporal processing mechanisms within higher-level auditory cortical regions in children with APD.

Overall, the results provide preliminary evidence that both noise processing and temporal speech encoding may be altered in children with APD. Further studies with larger samples will be necessary to confirm these findings and to better understand the neural mechanisms underlying auditory processing difficulties in this population.

Appendix

Table 1. The results of Mann-Whitney, to investigate the differences between APD and TD children in Ta and Tb, recorded to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
Ta_T7_1kHz-Q	21.000		0.095	0.680	0.365
Tb_T7_1kHz-Q	15.000		0.690	0.200	0.365
Ta_T8_1kHz-Q	12.000		1.000	-0.040	0.365
Tb_T8_1kHz-Q	15.000		0.690	0.200	0.365
Ta_T7_1kHz-W	23.000		0.032	0.840	0.365
Tb_T7_1kHz-W	10.000		0.690	-0.200	0.365
Ta_T8_1kHz-W	19.000		0.222	0.520	0.365
Tb_T8_1kHz-W	15.000		0.690	0.200	0.365
Ta_T7_1kHz-B	14.000		0.841	0.120	0.365
Tb_T7_1kHz-B	10.000		0.690	-0.200	0.365
Ta_T8_1kHz-B	14.000		0.841	0.120	0.365
Tb_T8_1kHz-B	9.000		0.548	-0.280	0.365
Ta_T7_da-Q	17.000		0.421	0.360	0.365
Ta_T8_da-Q	20.000		0.151	0.600	0.365
Tb_T7_da-Q	10.000		0.690	-0.200	0.365
Tb_T8_da-Q	15.000		0.690	0.200	0.365
Ta_T7_da-W	19.000		0.222	0.520	0.365
Ta_T8_da-W	11.000		0.841	-0.120	0.365
Tb_T7_da-W	15.000		0.690	0.200	0.365
Tb_T8_da-W	13.000		1.000	0.040	0.365
Ta_T7_da-B	15.000		0.690	0.200	0.365
Ta_T8_da-B	9.000		0.548	-0.280	0.365
Ta_T7_ta-Q	20.000		0.151	0.600	0.365
Ta_T8_ta-Q	15.000		0.690	0.200	0.365
Tb_T7_ta-Q	9.000		0.548	-0.280	0.365
Tb_T8_ta-Q	10.000		0.690	-0.200	0.365
Ta_T7_ta-W	18.000		0.310	0.440	0.365
Ta_T8_ta-W	13.000		1.000	0.040	0.365
Tb_T7_ta-W	11.000		0.841	-0.120	0.365
Tb_T8_ta-W	15.000		0.690	0.200	0.365
Ta_T7_ta-B	11.000		0.841	-0.120	0.365
Ta_T8_ta-B	8.000		0.730	-0.200	0.387

Note. For the Mann-Whitney test, effect size is given by the rank biserial correlation.

Note. Mann-Whitney U test.

Table 2. The results of Mann-Whitney, to investigate the differences between APD and TD children in frontocentral responses, recorded at Cz to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
N1_Cz_1kHz-Q	3.000		0.056	-0.760	0.365
P1_Cz_1kHz-Q	6.000		0.222	-0.520	0.365
P2_Cz_1kHz-Q	10.000		0.690	-0.200	0.365
N2_Cz_1kHz-Q	6.000		0.222	-0.520	0.365
N1_Cz_1kHz-W	7.000		0.310	-0.440	0.365
P1_Cz_1kHz-W	1.000		0.016	-0.920	0.365
P2_Cz_1kHz-W	15.000		0.690	0.200	0.365
N2_Cz_1kHz-W	2.000		0.032	-0.840	0.365
N1_Cz_1kHz-B	13.000		1.000	0.040	0.365
P1_Cz_1kHz-B	11.000		0.841	-0.120	0.365
P2_Cz_1kHz-B	18.000		0.310	0.440	0.365
N2_Cz_1kHz-B	21.000		0.095	0.680	0.365
N1_Cz_da-Q	5.000		0.151	-0.600	0.365
P1_Cz_da-Q	8.000		0.421	-0.360	0.365
P2_Cz_da-Q	7.000		0.310	-0.440	0.365
N2_Cz_da-Q	7.000		0.310	-0.440	0.365
N1_Cz_da-W	14.000		0.841	0.120	0.365
P1_Cz_da-W	15.000		0.690	0.200	0.365
P2_Cz_da-W	15.000		0.690	0.200	0.365
N2_Cz_da-W	6.000		0.222	-0.520	0.365
N1_Cz_da-B	13.000		1.000	0.040	0.365
P1_Cz_da-B	13.000		1.000	0.040	0.365
P2_Cz_da-B	13.000		1.000	0.040	0.365
N1_Cz_ta-Q	7.000		0.310	-0.440	0.365
P1_Cz_ta-Q	6.000		0.222	-0.520	0.365
P2_Cz_ta-Q	14.000		0.841	0.120	0.365
N2_Cz_ta-Q	9.000		0.548	-0.280	0.365
N1_Cz_ta-W	7.000		0.310	-0.440	0.365
P1_Cz_ta-W	5.000		0.151	-0.600	0.365
P2_Cz_ta-W	15.000		0.690	0.200	0.365
N2_Cz_ta-W	7.000		0.310	-0.440	0.365
N1_Cz_ta-B	12.000		1.000	-0.040	0.365
P1_Cz_ta-B	17.000		0.421	0.360	0.365

Note. For the Mann-Whitney test, effect size is given by the rank biserial correlation.
Note. Mann-Whitney U test.

Table 3. The results of Mann-Whitney, to investigate the differences between APD and TD children in latency of Ta and Tb, recorded at T7 and T8, to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
Ta7-LA-Q-1kHz-T7	9.500		0.600	-0.240	0.365
Tb7-LA-Q-1kHz-T7	13.000		1.000	0.040	0.365
Ta8-LA-Q-1kHz-T8	13.000		1.000	0.040	0.365
Tb8-LA-Q-1kHz-T8	19.500		0.168	0.560	0.365
Ta7-LA-W-1kHz-T7	13.500		0.917	0.080	0.365
Tb7-LA-W-1kHz-T7	8.500		0.463	-0.320	0.365

Table 3. The results of Mann-Whitney, to investigate the differences between APD and TD children in latency of Ta and Tb, recorded at T7 and T8, to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
Ta8-LA-W-1kHz-T8	15.000		0.674	0.200	0.365
Tb8-LA-W-1kHz-T8	15.000		0.671	0.200	0.365
Ta7-LA-B-1kHz-T7	5.500		0.173	-0.560	0.365
Tb7-LA-B-1kHz-T7	2.000		0.036	-0.840	0.365
Ta8-LA-B-1kHz-T8	8.000		0.401	-0.360	0.365
Tb8-LA-B-1kHz-T8	13.500		0.917	0.080	0.365
Ta7-LA-Q-da-T7	14.500		0.752	0.160	0.365
Ta8-LA-Q-da-T8	13.000		1.000	0.040	0.365
Tb7-LA-Q-da-T7	4.000		0.095	-0.680	0.365
Tb8-LA-Q-da-T8	7.500		0.344	-0.400	0.365
Ta7-LA-W-da-T7	11.000		0.841	-0.120	0.365
Ta8-LA-W-da-T8	12.500		1.000	0.000	0.365
Tb7-LA-W-da-T7	7.000		0.310	-0.440	0.365
Tb8-LA-W-da-T8	5.500		0.168	-0.560	0.365
Ta7-LA-B-da-T7	1.000		0.020	-0.920	0.365
Ta8-LA-B-da-T8	20.000		0.139	0.600	0.365
Ta7-LA-Q-ta-T7	12.500		1.000	0.000	0.365
Ta8-LA-Q-ta-T8	19.000		0.209	0.520	0.365
Tb7-LA-Q-ta-T7	4.500		0.106	-0.640	0.365
Tb8-LA-Q-ta-T8	9.000		0.548	-0.280	0.365
Ta7-LA-W-ta-T7	14.500		0.753	0.160	0.365
Ta8-LA-W-ta-T8	14.500		0.753	0.160	0.365
Tb7-LA-W-ta-T7	9.500		0.600	-0.240	0.365
Tb8-LA-W-ta-T8	5.000		0.130	-0.600	0.365
Ta7-LA-B-ta-T7	0.000		0.011	-1.000	0.365
Ta8-LA-B-ta-T8	22.000		0.057	0.760	0.365

Note. For the Mann-Whitney test, effect size is given by the rank biserial correlation.

Note. Mann-Whitney U test.

Table 4. *The results of Mann-Whitney test, to investigate the differences between APD and TD children in latency of P1, N1, P2, N2, recorded at Cz to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).*

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
P1-LA-Q-1kHz-Cz	8.500		0.462	-0.320	0.365
N1-LA-Q-1kHz-Cz	11.000		0.841	-0.120	0.365
P2-LA-Q-1kHz-Cz	6.500		0.248	-0.480	0.365
N2-LA-Q-1kHz-Cz	7.500		0.346	-0.400	0.365
P1-LA-W-1kHz-Cz	11.500		0.917	-0.080	0.365
N1-LA-W-1kHz-Cz	13.000		1.000	0.040	0.365
P2-LA-W-1kHz-Cz	8.500		0.463	-0.320	0.365
N2-LA-W-1kHz-Cz	2.000		0.036	-0.840	0.365
P1-LA-B-1kHz-Cz	10.000		0.675	-0.200	0.365
N1-LA-B-1kHz-Cz	11.500		0.917	-0.080	0.365
P2-LA-B-1kHz-Cz	12.000		1.000	-0.040	0.365
N2-LA-B-1kHz-Cz	6.500		0.249	-0.480	0.365
N1-LA-Q-da-Cz	6.500		0.234	-0.480	0.365
P2-LA-Q-da-Cz	2.000		0.032	-0.840	0.365
N2-LA-Q-da-Cz	5.000		0.142	-0.600	0.365
P1-LA-W-da-Cz	14.000		0.841	0.120	0.365

Table 4. *The results of Mann-Whitney test, to investigate the differences between APD and TD children in latency of P1, N1, P2, N2, recorded at Cz to 1kHz, /ta/ and /da/, in quiet (Q), white noise (W) and babble noise (B).*

	U	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
N1-LA-W-da-Cz	12.000		1.000	-0.040	0.365
P2-LA-W-da-Cz	11.000		0.841	-0.120	0.365
N2-CzLA-W-da-Cz	6.500		0.249	-0.480	0.365
P1-LA-Q-ta-Cz	19.000		0.207	0.520	0.365
N1-LA-Q-ta-Cz	17.000		0.421	0.360	0.365
P2-LA-Q-ta-Cz	9.500		0.600	-0.240	0.365
N2-LA-Q-ta-Cz	3.000		0.690	-0.760	0.365
P1-LA-W-ta-Cz	13.000		1.000	0.040	0.365
N1-LA-W-ta-Cz	9.000		0.528	-0.280	0.365
P2-LA-W-ta-Cz	3.500		0.074	-0.720	0.365
N2-LA-W-ta-Cz	9.000		0.548	-0.280	0.365
P1-LA-B-ta-Cz	18.500		0.242	0.480	0.365
N1-LA-B-ta-Cz	4.500		0.112	-0.640	0.365
P1-LA-B-da-Cz	22.000		0.056	0.760	0.365
N1-LA-B-da-Cz	23.000		0.036	0.840	0.365
P1-LA-Q-da-Cz	12.000		1.000	-0.040	0.365

Note. For the Mann-Whitney test, effect size is given by the rank biserial correlation.
Note. Mann-Whitney U test.

Table 5. *The results of Wilcoxon Test to investigate the differences in T-complex in response to /ta/ and /da/, in quiet and in n noise recorded at T7 and T8, in TD children.*

Measure 1		Measure 2	W	z	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
Ta_T7_da-Q	-	Ta_T7_ta-Q	15.000	2.023		0.063	1.000	0.458
Tb_T7_da-Q	-	Tb_T7_ta-Q	11.000	0.944		0.438	0.467	0.458
Ta_T8_da-Q	-	Ta_T8_ta-Q	15.000	2.023		0.063	1.000	0.458
Tb_T8_da-Q	-	Tb_T8_ta-Q	15.000	2.023		0.063	1.000	0.458
Ta_T7_da-B	-	Ta_T7_ta-B	10.000	0.674		0.625	0.333	0.458
Ta_T8_da-B	-	Ta_T8_ta-B	14.000	1.753		0.125	0.867	0.458

Note. Wilcoxon signed-rank test.

Table 6. *The results of Wilcoxon Test to investigate the differences in latency T-complex in response to /ta/ and /da/, recorded at T7 and T8, in TD children.*

Measure 1		Measure 2	W	z	df	p
Ta7-LA-Q-da-T7	-	Ta7-LA-Q-ta-T7	1.000	-1.461		0.197
Ta8-LA-Q-da-T8	-	Ta8-LA-Q-ta-T8	7.000	-0.135		1.000
Ta7-LA-B-da-T7	-	Ta7-LA-B-ta-T7	0.000	-2.023		0.063
Ta8-LA-B-da-T8	-	Ta8-LA-B-ta-T8	0.000	-2.023		0.063
Tb7-LA-Q-da-T7	-	Tb7-LA-Q-ta-T7	10.000	1.826		0.098
Tb8-LA-Q-da-T8	-	Tb8-LA-Q-ta-T8	11.000	0.944		0.438

Table 6. *The results of Wilcoxon Test to investigate the differences in latency T-complex in response to /ta/ and /da/, recorded at T7 and T8, in TD children.*

Measure 1	Measure 2	W	z	df	p

Note. Wilcoxon signed-rank test.

Table 7. *The results of Wilcoxon Test to investigate the differences in T-complex in response to /ta/ and /da/, in quiet and in noise recorded at T7 and T8 in APD children*.

Measure 1		Measure 2	W	z	df	p	Rank-Biserial Correlation	SE Rank-Biserial Correlation
Ta_T7_da-Q	-	Ta_T7_ta-Q	14.000	1.753		0.125	0.867	0.458
Tb_T7_da-Q	-	Tb_T7_ta-Q	14.000	1.753		0.125	0.867	0.458
Ta_T8_da-Q	-	Ta_T8_ta-Q	15.000	2.023		0.163	1.000	0.458
Tb_T8_da-Q	-	Tb_T8_ta-Q	14.000	1.753		0.125	0.867	0.458
Ta_T7_da-B	-	Ta_T7_ta-B	11.000	0.944		0.438	0.467	0.458
Ta_T8_da-B	-	Tb_T8_ta-B	15.000	2.023		0.093	1.000	0.458

Note. Wilcoxon signed-rank test.

Table 8. *The results of Wilcoxon Test to investigate the differences in latency T-complex in response to /ta/ and /da/, recorded at T7 and T8, in APD children.*

Measure 1		Measure 2	W	z	df	p
Ta7-LA-Q-da-T7	-	Ta7-LA-Q-ta-T7	7.000	-0.135		1.000
Ta8-LA-Q-da-T8	-	Ta8-LA-Q-ta-T8	3.000	-1.214		0.313
Ta7-LA-B-da-T7	-	Ta7-LA-B-ta-T7	0.000	-2.023		0.063
Ta8-LA-B-da-T8	-	Ta8-LA-B-ta-T8	0.000	-2.023		0.063
Tb7-LA-Q-da-T7	-	Tb7-LA-Q-ta-T7	6.000	-0.405		0.813
Tb8-LA-Q-da-T8	-	Tb8-LA-Q-ta-T8	7.000	-0.135		1.000

Note. Wilcoxon signed-rank test.

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Chapter 5. General Discussion and Conclusion

General Discussion

The previous studies in current thesis were aimed to answer the existing literature gap among the AEP studies in the typical and atypical populations. The first study, Noise study, evaluated the modulation of auditory evoked potentials with different type of stimuli and noise in typically developing children and adults. Both frontocentral and temporal auditory responses showed noise-induced changes including smaller/ or larger amplitude and longer latency, compared to quiet conditions. Also, both stimulus types and noise types appear to affect the pattern of noise-induced changes in amplitude and latency of auditory evoked potentials, in both children and adult populations. The second study, Stimuli study, was aimed to evaluate the effect of long and short voice onset time in normal groups on T-complex. This study demonstrated the representation of temporal features like short and long voice onset time indexed by T-complex components including Ta and Tb in all participants. Interestingly, there was rightward lateralization of processing of short and long voice onset time indexed by Tb peak in both adult and children. The third study (APD study) investigated the differences in AEPs between TD and APD groups, using different stimuli and noise. Results showed a trend of atypical auditory responses in children with APD, in the presence of noise. Also, it seemed there was a trend of atypical representation of VOT in children with APD, compared to TD.

Building on the findings of previous studies this thesis was able to investigate how cortical auditory processing adapts to noise and temporal features across development, and how this adaptive system is altered in children with auditory processing disorder (APD). Across several studies conducted to form this dissertation, the findings converge to suggest that efficient speech-in-noise perception relies on dynamic cortical modulation, hemispheric specialization, and the maturation of top-down regulatory mechanisms. Moreover, our findings would provide novel evidence that these adaptive processes are disrupted in children with APD.

The results of this thesis suggest that cortical responses to noise should not be interpreted as isolated electrophysiological effects. Instead, they reflect a developmental process in which sensory encoding, cortical regulatory mechanisms, and hemispheric specialization interact to support auditory perception in challenging listening environments. To synthesize these findings, the discussion is organized into three main sections: 1. Developmental Effects on Noise-Induced Modulation in TD groups, 2. Hemispheric Specialization and Noise Adaptation in TD groups, 3. Representation of Voice Onset Time Across Development, 4. Cortical Processing in Children with APD.

7.1. Developmental effects on Noise-Induced Modulation

The Noise study demonstrated that both frontocentral (P1–N1–P2–N2) and temporal (T-complex, Ta-Tb) components are affected by background noise in both children and adults. However, the pattern and magnitude of these modulations differed across age groups (Figure 1, 2, 3 and 4, table 1).

In adults, noise elicited both suppressions and enhancement effects, including increased amplitude of N1 and T-complex components in response to auditory stimuli. These findings suggest adaptive cortical recruitment under degraded listening conditions (Alain et al., 2009; Guinan, 2006; Haider et al., 2013; Huffman & Henson, 1990; Scharf et al., 1997). Notably, enhancement effects were speech-specific, possibly indicating that top-down mechanisms likely contribute to maintaining speech representation in noise.

In contrast, children showed reduced evidence of such adaptive modulation. While latency prolongation in noise was observed in both groups, children did not exhibit the same degree of amplitude enhancement seen in adults (Table 1). This developmental difference could be consistent with immaturity of cortical regulatory mechanisms and incomplete development of top-down inhibitory control (Leibold & Buss, 2013; Wightman & Kistler, 2005; Leibold, 2017). Children are consistently reported to require more favorable signal-to-noise ratios (SNRs) than adults to achieve comparable levels of speech detection and recognition (Elliott, 1979; Nittrouer & Boothroyd, 1990; Leibold & Buss, 2013). These developmental differences have been attributed to immaturity in both sensory encoding and top-down cognitive processes, including auditory working memory, selective attention, and linguistic strategies (Leibold & Buss, 2013; Wightman & Kistler, 2005; Leibold, 2017). Additionally, the central auditory system continues to mature throughout childhood, contributing to less efficient processing of degraded signals (Hall et al., 2002). Only two studies have directly compared children and adults using cortical AEPs under noisy conditions (Gustafson et al., 2019; Duquette-Laplante et al., 2024). Gustafson et al. (2019) reported no significant group differences at +15 dB SNR, suggesting that mild noise levels may not sufficiently challenge the developing auditory system. In contrast, Duquette-Laplante et al. (2024) demonstrated fewer identifiable cortical components in children compared to adults across noise conditions, attributing this to immaturity of central auditory pathways.

***Table 1**. The differences and similarities in noise-induced changes of AEPs, between children and adults.*

Peak	Similarities in amplitude changes	Differences in amplitude changes	Similarities in latency changes	Differences in latency changes
P1	In both groups, amplitude of P1 was reduced in white noise.	Amplitude of P1 was reduced on adults, only with speech stimuli, but with both stimuli in children.	In both groups, there was longer latency in noise, it was significant only with speech stimuli.	There are no differences between adults and children.
N1	No similarities between children and adults.	Significant noise induced changes, including increasing and reducing amplitude in adults, but no significant changes in children.	In both groups, there was longer latency in noises with speech stimulus and in babble noise.	The longer latency of N1 in children was not significant in white noise with 1kHz.
P2	In both groups, amplitude of P2 was reduced in noise, both stimuli in babble noise, and only speech stimuli in white noise.	Same patterns were observed and there were no differences.	In both groups, there was longer latency in noise, significantly with speech stimulus.	There are no differences between adults and children.
Ta	In both groups, the smaller amplitude of Ta was seen in noise.	Increasing pattern of amplitude changes was not seen in children, also there were no noise-induced location differences between T7 and T8 in children's groups.	In both groups, there was longer latency in noise, significantly with speech stimulus.	Contrary to adults, children showed longer latency with 1kHz, but only in white noise.
Tb	No similarities between children and adults.	Significant noise induced changes, including increasing and reducing amplitude in adults, no significant changes in children.	In both groups, there was longer latency in noise, significantly with speech stimulus.	Contrary to adults, children showed longer latency with 1kHz, but only in white noise.

Taken together, processing speech in noise places increased demands on the auditory system because relevant acoustic cues must be extracted from competing signals. The present findings support the view that cortical adaptation to noise is not merely a sensory process, but a dynamically regulated mechanism that matures through adolescence. Adult listeners appear to recruit compensatory cortical resources to preserve speech encoding under adverse conditions. Children, however, exhibit a less flexible adaptive system, potentially explaining their increased vulnerability to background noise.

7.2. Hemispheric Specialization and Noise Adaptation

A novel contribution of this thesis is the demonstration of rightward lateralization of T-complex responses in adults during noisy speech processing. This pattern aligns with evidence suggesting that the right hemisphere plays a greater role in processing slower temporal modulations and spectral features, which become more salient in degraded listening conditions (Neophytou et al., 2022). In noise, cortical processing may shift toward reliance on slower temporal cues that are more resistant to masking (Ding & Simon, 2013). This lateralization suggests that hemispheric specialization may contribute to cortical adaptation when speech is processed under degraded listening conditions.

In summary, the enhanced activation of right temporal regions in adults is likely reflected in adaptive neural reweighting. This lateralization was not observed in children (Figure 1, Table 1). The absence of this lateralized adaptation in children may indicate ongoing maturation of incomplete hemispheric specialization under challenging listening conditions. Thus, developmental differences in speech-in-noise perception may stem not only from immature sensory encoding, but also from ongoing maturation of functional lateralization of auditory cortical networks.

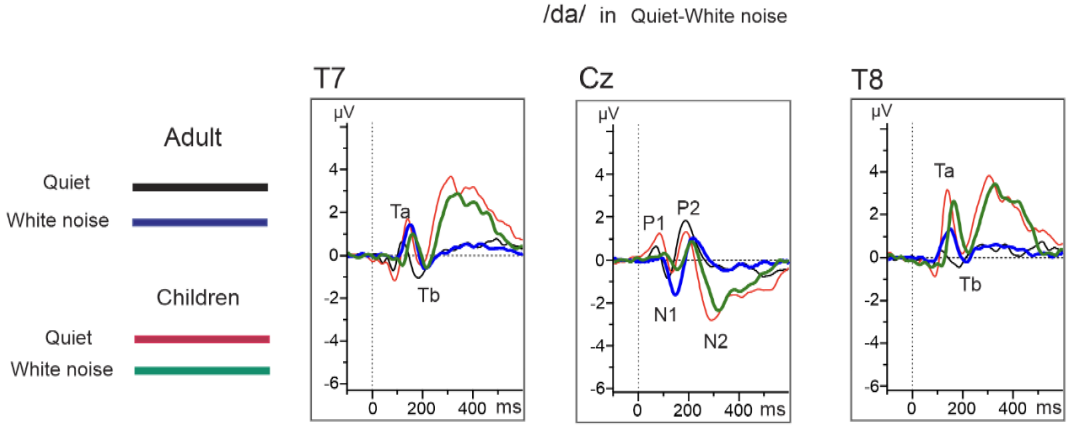

Figure 1. *The grand average of AEPs in children and adults, in response to 1kHz, in quiet and white noise.*

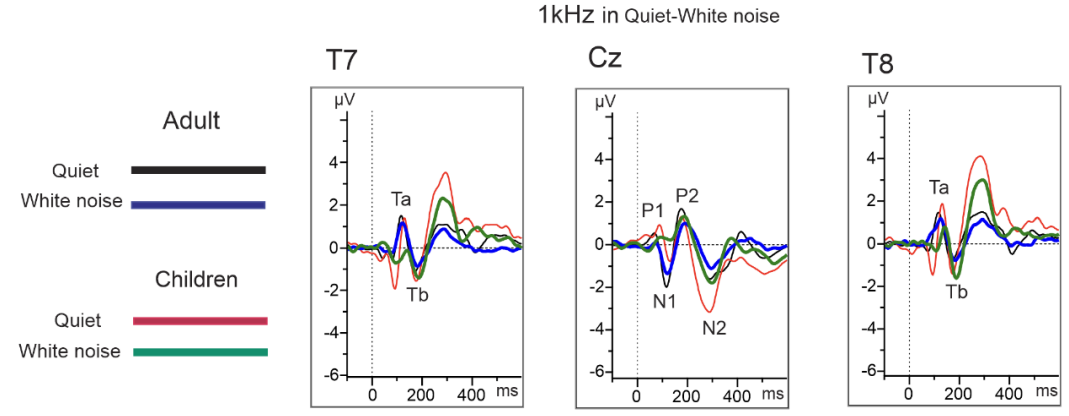

Figure 2. *The grand average of AEPs in children and adults, in response to /da/, in quiet and white noise.*

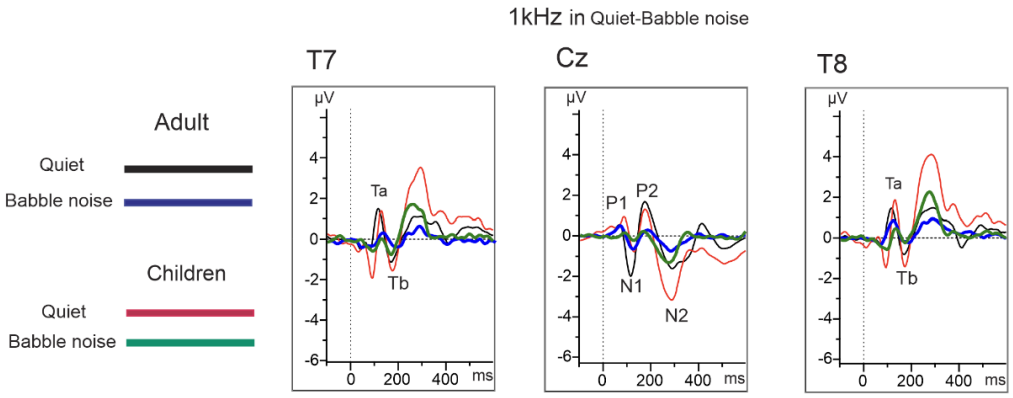

Figure 3.31 *The grand average of AEPs in children and adults, in response to 1kHz, in quiet and babble noise.*

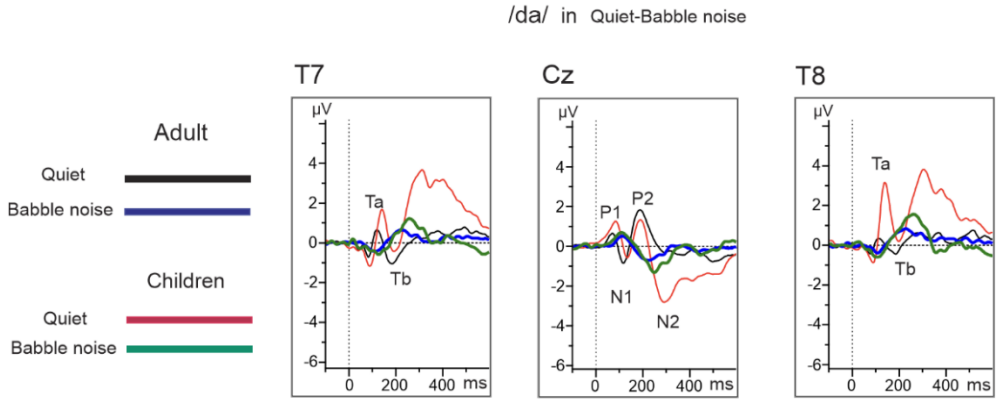

Figure 4. *The grand average of AEPs in children and adults, in response to /da/, in quiet and babble noise.*

7.3. Representation of Voice Onset Time (VOT) Across Development

Temporal processing is central to speech perception, particularly under adverse conditions (Cunningham et al., 2002; Schnupp et al., 2010; Shinn-Cunningham & Best, 2008). Previous research has shown developmental differences in frontocentral AEP morphology in response to VOT contrasts. Adults exhibit double N1 responses to long VOT stimuli (Sharma & Dorman, 1999), whereas children often lack this morphology (King et al., 2008; Almeqbel & McMahon, 2015). These differences have been attributed to prolonged neuronal refractory periods in immature cortical networks (Ceponiene et al., 1998; Gilley et al., 2005). The Stimuli study extended this developmental framework by examining cortical representation of VOT using the T-complex (Figure 5, Table 2). In contrast to frontocentral components observed in the literature, which exhibit marked developmental changes, present T-complex morphology showed similar patterns in children and adults (Figure 5). Both age groups demonstrated rightward lateralization of long VOT representation indexed by Tb amplitude. This suggests

that temporal asymmetry at secondary auditory cortical levels is established relatively early in development. Given prior evidence that the T-complex matures earlier than frontocentral responses (Bishop et al., 2011; Tonnquist-Uhlen et al., 2003), these findings indicate that certain aspects of temporal encoding are developmentally stable by middle childhood. Furthermore, the adult-children similarities in lateralized representation of long temporal changes versus fast temporal changes are in line with previous findings that report the existence of temporal processing asymmetries in young children (Thompson et al., 2016). Thompson et al., (2016) reported a lateralization of high-frequency (20–50 Hz) endogenous cortical oscillations, even in younger children aged 3–5 years. Therefore, the presence of hemispheric differences in VOT representation indexed by Tb in children aged 8–12 years is consistent with the expected maturation of secondary auditory cortical processing

Table 2. *The similarities and differences in Ta and Tb between results of Adult and Children in response to speech sound /da/ and /ta/.*

Peak	Quiet		Noise	
	Similarities	**Differences**	**Similarities**	**Differences**
Ta	Both groups had larger amplitude to /da/ than /ta/, symmetric at electrodes T7 and T8. No stimuli effect on latency.	No differences between adults and children	Both groups had longer latency to /ta/than /da/ at electrodes T7 and T8. No stimuli effect on amplitude.	No differences between adults and children
Tb	Both groups had larger amplitude to /ta/ than /da/, at electrode T8, in quiet. No stimuli effect on latency.	No differences between adults and children	Both groups had longer latency to /ta/ than /da/ at electrodes T7 and T8. No stimuli effect on amplitude.	No differences between adults and children

Importantly, these results position Tb as a robust marker of temporal speech processing across development. This suggests that secondary auditory cortex may provide a stable neural index of temporal speech encoding even when frontocentral cortical responses are still maturing.

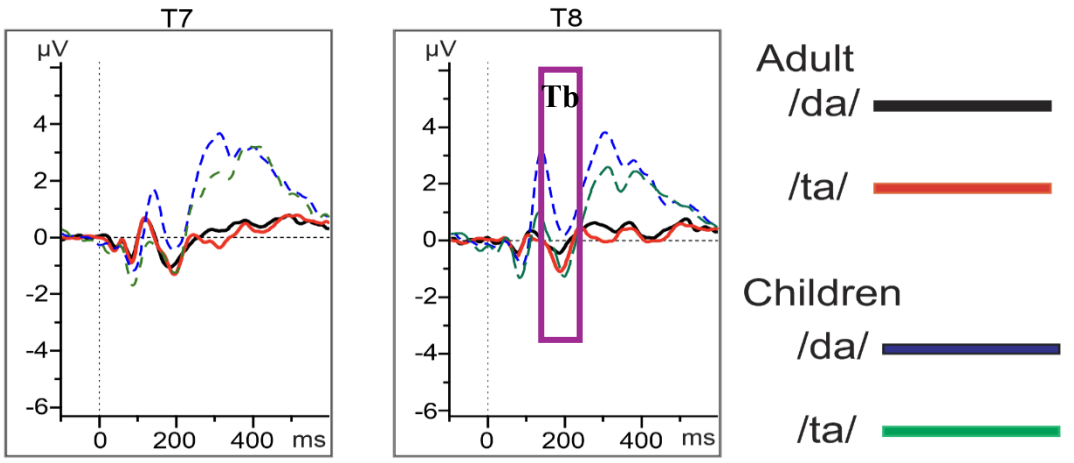

Figure 5. *The grand average of T-complex to /ta/ and /da/, in children and adults. In both groups, there was larger Ta to /da/ than /ta at T7 and T8, and larger Tb to /ta/ than /da/ at T8.*

7.4. Cortical Processing in Children with APD

The APD study provides critical insight into how this adaptive cortical system may be disrupted in children with APD. Children with APD demonstrated atypical cortical morphology primarily under noisy conditions. Significant group differences were observed in both frontocentral and temporal components, suggesting alterations in cortical auditory processing. Two principal abnormalities emerged: atypical modulation in noise, possibility of absence of lateralized VOT representation, indicating altered secondary auditory cortical specialization.

Collectively, these findings support the latest perspective regarding APD (BSA, 2018) considering a dual-mechanism interpretation of APD, involving both bottom-up sensory encoding differences and top-down regulatory dysfunction. The disrupted lateralization further implies that higher-order cortical organization may be altered in this population.

7.5. Behavioral Findings and Interpretation

In contrast to the electrophysiological findings, behavioral measures did not reach statistical significance between APD and typically developing children. However, an important pattern emerged: children with APD demonstrated a tendency toward shorter reaction times accompanied by lower response accuracy. Although these differences did not reach statistical significance, likely due to limited sample size, the observed pattern may be theoretically meaningful. Specifically, the combination of faster responses and reduced accuracy may reflect altered cognitive control processing, particularly inefficient inhibitory regulation or impulsive response strategies (Leotti & Wager, 2010; Schulz et al., 2023). In tasks requiring discrimination of temporally complex stimuli, optimal performance depends not only on

accurate sensory encoding but also on the ability to suppress premature or incorrect responses. The shorter reaction times observed in children with APD, coupled with reduced accuracy, may suggest compromised executive control mechanisms rather than purely auditory sensory deficits (Drosos, 2024; Schulz et al., 2023; Suarez et al., 2021).

7.6. Clinical Implications

The findings of this thesis provide new insights into the neurophysiological mechanisms underlying speech perception in noise and how these processes are modulated by stimulus characteristics, background noise, and population differences such as APD. The findings also contribute to a better understanding of how cortical auditory processing develops and adapts to challenging listening environments.

Given the absence of well-established objective cortical markers for auditory processing impairments, the present findings suggest that electrophysiological measures. Particularly the Tb component, may represent a promising candidate for investigating temporal processing deficits in children with APD. Considering the earlier maturation of the T-complex relative to frontocentral cortical responses, these findings may help inform future development of objective assessment approaches aimed at identifying atypical auditory cortical processing during development.

Regarding the frontocentral response, the current result of larger amplitude of N2 in noise in APD seems to be the most consistent finding of AEPs in APD with the literature. Out of four AEP studies in APDs (Sharma et al., 2014, Hassan 2015, Morlet 2019, Hussain 2022), three studies have recorded N2 and reported a larger amplitude in noise in APD than TD groups. Presumably, this pattern may be valuable to draw a consistent pattern of cortical AEPs in children with APD.

7.7. Limitations

Although the present findings should be interpreted cautiously due to the relatively small sample size, the results also demonstrated more atypical auditory processing patterns in children with APD under noisy listening conditions. This observation highlights the potential importance of incorporating background noise into future objective assessment paradigms for APD, particularly when evaluating speech-related auditory processing.

This thesis has several limitations that should be acknowledged. The sample size of APD study is one of the main limitations of current thesis. The existence of nonsignificant

results in APD study can be attributed to small sample size of participants. Further exploration with larger sample size is necessary to be utilized in APD population to clarify the results.

The rigorous inclusion criteria applied in the current thesis likely contributed to the small sample size. Participants were required to present impairments exclusively in the auditory domain, with no associated deficits in attention or cognition. Given the high comorbidity between APD and ADHD, identifying children with pure APD proved challenging. Recruitment took place from October 2024, following ethics approval, until August 2025; despite this extended recruitment period, only five children met the inclusion criteria.

Another contributing factor to the small sample size may have been the demanding nature of the experimental design. The study included long paradigms, with multiple stimuli that generated numerous blocks, resulting in lengthy testing sessions. Such long and cognitively demanding ERP paradigms are often difficult for children to tolerate compared to adults. Therefore, future research should consider developing shorter paradigms that are more suitable for pediatric populations. Additionally, while this thesis focused on children with APD, future investigations could extend this line of research to adults with APD, who are generally better able to tolerate longer testing sessions and may offer complementary insights into auditory processing mechanisms across the lifespan.

Although the results from adult populations might highlight the top-down effects and adaptation of auditory neural system to speech stimuli in noise versus nonspeech stimuli, this thesis reported only the results of passive paradigm which eliminate the effect of top-down and attentional processing.

7.8. Future Directions

Recording AEPs in noise, when participants are engaging in a task could help better understand the impacts of noise on auditory processing and the contribution of top-down processing in understanding speech in noise.

The behavioral pattern observed including shorter reaction time in the present study raises important questions regarding the role of cognitive control in APD. While the current experiments focused primarily on stimulus-locked cortical responses, future research should directly examine higher-order cognitive processes, particularly inhibitory control and error monitoring. Electrophysiological markers such as the N2 component and error-related negativity (ERN) provide objective indices of inhibitory processing and performance monitoring (Falkenstein et al., 2000; Gehring et al., 2018). These components have been shown

to be altered in neurodevelopmental conditions characterized by executive dysfunction, including ADHD and dyslexia (Balogh et al., 2017; Su et al., 2025). Given the emerging evidence of atypical cortical modulation in APD, investigation of response-locked and cognitive-control-related potential may clarify whether auditory difficulties are partly driven by impaired executive regulation. Importantly, incorporating cognitive control evaluation into auditory evoked potentials assessments in APD would allow differentiation between 1. primary sensory encoding deficits, 2. impaired top-down auditory modulation. 3. and broader executive dysfunction affecting task performance. Such differentiation has direct clinical implications. If inhibitory control deficits contribute to auditory processing difficulties, intervention strategies may need to extend beyond auditory training and incorporate executive function training approaches. Therefore, future research should investigate top-down processing more directly, including response-locked potentials such as ERN, to better characterize inhibitory and cognitive control mechanisms in APD.

Moreover, physiological evaluations like suppression OAE in addition to electrophysiological responses will bring more information regarding top-down processing and efferent system in children with APD.

7.9. Conclusion

This thesis advances understanding of cortical auditory processing by suggesting that speech-in-noise perception involves dynamic cortical modulation, hemispheric specialization, and developmental maturation of top-down mechanisms. Adults appeared to exhibit adaptive enhancement and lateralized recruitment of temporal cortical regions in noise, whereas children showed reduced adaptive flexibility. In the present study, children with APD exhibited atypical cortical modulation patterns and reduced hemispheric specialization compared to typically developing children.

These findings contribute to a developmental neurophysiological framework of speech-in-noise processing and may help inform future research toward more sensitive objective assessment approaches. Importantly, the findings suggest that the Tb component may represent a promising marker of temporal speech processing across age groups, potentially reflecting relatively stable temporal speech encoding within secondary auditory cortical regions while frontocentral cortical responses continue to mature.

Given the relatively small APD sample size, these findings should be interpreted cautiously and considered preliminary. Nevertheless, they highlight the potential value of electrophysiological measures in future investigations of early identification and rehabilitation approaches for APD.

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Appendix

1.Methodology

Participants characteristics

Three groups were involved in this thesis proposal: two groups as normal groups including 20 adults (18-30 years old), TD children, n=15 (8-12 years old) and one experimental group include children with auditory processing disorder, n=5 (8-12 years old).

Ethics approval

The University of Ottawa Research Ethics Board approved this study, Ethic file number=H-07-24-10451. Prior to testing, the purpose of the study was explained to each participant. All participants in adult group received and signed a copy of the written consent form. Also, an informed consent was obtained from children and a signed a copy of the written consent form their parents. (A copy of ethics approval documents and the consent forms will be added in appendix).

Recruitment

The study was started with adult population. The group of adults were selected among the students studying at the University of Ottawa. After finishing adult's data collection and analysis, the next phase including typically developing children and APD were started. The typically developing children were age-matched to the APD group and were recruited from the researchers' environment. Regarding APD group, participants with previously diagnosed APD were recruited from a private audiology clinic in Ottawa. They had already had a clinical diagnosis of APD before enrolling in the study. That diagnosis was carried out separately from the current study in the private audiology clinic.

Inclusion and exclusion criteria and process

All participants including adults and children from normal and APD groups had normal hearing detection threshold at 15 dB HL or less in octave frequencies from 500 Hz to 8 kHz, bilaterally, based on the American National Standards Institute (1996)'s protocol. Tympanograms were normal in all participants (admittance curve with a single peak between +50 to -100daPa using a 226-Hz probe tone). All groups were right-handed and it was determined according to an adapted protocol to assess laterality (De Agostini & Dellatolas, 1988). No history of otological or neurological disorders was reported by any of the normal participants. In addition, participants with musical training were not included, as previous

studies have shown that music practice can enhance speech-in-noise perception and increase the amplitude of AEPs in the presence of background noise compared to quiet (Anderson et al., 2013; Benjamin Rich Zendel et al., 2015). None of the children in APD group did not have musical training as well.

Auditory processing domain questionnaire (APDQ) was used to assess auditory processing, attention and language abilities of children in TD groups. APDQ is a screening tool with 70-85% accuracy and evaluate auditory processing, attention and language skill's of individual between 7-18 years old (O'Hara & Mealings, 2018). Two out of 17 children from normal group had poor performance in attention skills, so they were excluded from the study. Considering that the poor perception of speech in noise is the hallmark of APD, the ability to understanding speech in noise was also evaluated in normal group to make sure there is not listening difficulties among the included normal group. Bamford-Kowal-Bench Sentences in Noise (BKB-SIN) (BKB-SIN, 2005) was used for this purpose. The BKB-SIN test is designed to estimate the required listener's SNR to obtain 50% of key words correct. The test consists of 18 list pairs (e.g., lists 1a and 1b are to be used together) of 10 sentences each spoken by a male speaker and in the presence of four-talker babble. The participants needed to repeat the whole sentence. In each list, the sentences are presented at pre-recorded SNRs that decrease 3-dB steps from a +21 (for the first sentence) to a -6 dB SNR (for the last sentence). Each sentence is prefaced with ''ready'' and has three target words, except for the first sentence which has four target words. Each list of 10 sentences requires 95s to administer. The following is one example of sentences in the BKB-SIN: The doge made and angry noise. All the 15 children in normal group had normal performance in BKB-SIN test.

Children either in normal or APD groups also needed to have normal performance in cognitive ability test. In this regard, the auditory Continuous Performance Test (ACPT) was used, which is a cognitive task designed to measure a person's sustained attention and vigilance, particularly in an auditory context (Keith, 1994; Riccio et al., 1996).

Procedure

During the experiment, after familiarization with the testing equipment and procedure, the participants were seated in a sound-attenuated testing room subjects on a comfortable reclining chair, where they completed the experimental tasks. The experiments procedure included passive and active paradigms. Passive paradigm would bring information mainly on auditory processing with elimination of attention. Active paradigm was also done because it is

closer to real-life listening situations and compare its results to passive paradigm may bring information on the effect of attentional processing in auditory speech in noise. Considering effect of fatigue, especially in children, the testing was always started with active paradigm and then followed by passive paradigm.

For the passive conditions, subjects watched a silent, closed-captioned movie of their choice while stimuli were presented. During the recording, they were alert and calm. For the active condition, participants indicated which sound they heard between /da/ and /ta/ by pressing a labeled button.

Stimuli

Considering that this study was the first research evaluating T-complex noise, even in normal population, there was no previous result as reference, so both simple non-speech stimuli and complex stimuli like speech syllables were used to provide a comprehensive information and evaluation of auditory processing manifested by T-complex. Non-speech stimulus was a pure tone 1kHz with 250ms in duration and 10ms rise and fall time, made by Audacity software. The speech stimuli were two syllables /da/ and /ta/ (Dubno & Schaefer, 1992). These two speech stimuli have similar articulation place (Digeser et al., 2009) and contain similar formant frequencies (Figure 1and 2). The /da/ and /ta/ differ mainly in consonant voicing which is cued by voice onset time (VOT), /da/ has a fast temporal dynamic at the onset of the syllables (short voicing time, around 20ms) and /ta/ has a slow temporal dynamic at the onset of the syllables (long voicing time, around 100ms) (Figure 1 and 2). Two types on noise including White noise and Babble noise were used in this project. White noise was made and extracted from Audacity software and babble noise was generated in the same method as French babble noise was generated in study of Zendel et al, 2015, but in English (Benjamin Rich Zendel et al., 2015). The SNR was kept at +5dB. All three stimulus were presented through an insert-earphone (E-A-RTONE 3A), binaurally at 80-dB SPL. Interstimulus interval (ISI) was randomly ranging 1100-1300 second. Since hearing in noise is typically a binaural process, it is important to implement test measures that increase the ecological validity, or the extent to which the results can generalize to real-life listening situations. The stimuli were delivered in a non-oddball, homogenous paradigm, because Oddball paradigms introduce cognitive variables such as attention and stimulus context effects, which complicate the interpretation of earlier CAEP components (Billings et al., 2011).

Passive paradigm included non-speech and speech experiments. It was started with non-speech experiments, including three blocks, one block for each listening condition including quiet, white noise and babble noise. Each block contained 300 1kHz, so a total of 900 presentations was presented by E-prime software in the non-speech part. Non-speech experiment was followed by speech experiment with three blocks for three listening conditions. Each blocks contains 300 /da/ and 300 /ta/, they were ordered randomly in each block. Active paradigm contained only speech experiment and conducted in two listening conditions including in quiet and in babble noise. There were two blocks for each listening condition, with 80 /da/ and 80 /ta/ in each block. The order of the listening conditions was random in both passive and active paradigm.

To minimize the fatigue's effect and to maintain the motivation, 1-5 minutes of break was considered after each block. The whole test lasted about 90 minutes for the passive mode and about 20 minutes for the active test.

The current thesis reported findings derived from the passive paradigm and the behavioral measures of active paradigm, while the EEG results of the active paradigm will be disseminated in future publications.

The passive paradigm results were structured into two main research articles, each addressing a specific thesis objective. Each article was divided into two papers tailored separately for adult and children population.

- The first article examines the effect of noise on T-complex and frontocentral cortical responses, using results from 1 kHz tones and /da/ stimuli presented in quiet, white noise, and babble noise.
- The second article investigates the representation of short and long voice onset time (VOT) by the T-complex, based on responses to /da/ and /ta/ stimuli in quiet and babble noise conditions. Also, the behavioral responses to /da/ and /ta/ have been evaluated.

Electrophysiological recording

The electrical responses were recorded from 32 active silver/silver chloride electrodes attached to an electrode cap (Brain Products GmbH, Munich, Germany). The electrodes were placed over frontal, central, parietal, temporal, and occipital areas of the scalp, using 10-20 standard system. A ground electrode was placed on the forehead. Vertical eye movements and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left

eye. The tip of the nose served as an online reference for all channels, including the electro-oculogram (EOG). Interelectrode impedances were kept between 25 and 50kΩ. The EEG signals were digitized continuously at a sampling rate of 500 Hz and stored on a hard disk for later analyses.

Electrophysiological data analysis

The data were analysed using Brain Products' Analyzer2 software. The signals were digitally filtered using a low-pass filter set at 20 Hz (24 dB/octave roll-off). A common average reference was calculated as off-line reference (Clunies-Ross et al., 2018). Eye movements and blink artefacts were corrected in the EEG using Independent Component Analysis (ICA) (Chaumon et al., 2015; Makeig et al., 1996). To achieve this, vertical and horizontal EOG activity was computed. Vertical EOG was derived by subtracting the inferior orbital channel from FP2, while horizontal eye movements were obtained by subtracting FT9 from FT10 (Duda-Milloy et al., 2019). The ICA model assumes that the observed electrophysiological signals represent a linear mixture of neural sources and artifacts that are mutually statistically independent. Using blind source separation, the algorithm was trained to identify each participant's ocular artifact signature, which was then removed from the EEG traces. Epochs were excluded if they had amplitudes exceeding ±100μV at any site. Less than 10% of trials were excluded. Moreover, the EEG was visually inspected to ensure adequate artifact rejection. Epochs were segmented from 100ms pre-stimulus onset to 600ms post-stimulus onset. Segmented waveforms were baseline corrected around the 100ms pre-stimulus interval and averaged based on condition. Although both Cz and Fz were selected for the detection of frontocentral responses (P1-N1-P2), only the responses at Cz are reported as there were more robust responses at Cz compared to Fz (Näätänen & Picton, 1987). Additionally, the amplitude of the N1 at Cz has been considered the strongest predictor of individual speech-in-noise perception thresholds (Billings et al., 2013). The T-complex was extracted from the temporal regions, T7 and T8 (Wolpaw & Penry, 1975). Tonnquist-Uhlen and colleagues (2003) also suggest that T7 and T8 are optimal for measuring the T-complex since they reflect little activity from the sources underlying the P1, N1 and P2. Regarding T-complex components, as Na was not detected clearly in most of the participants, only Ta and Tb are reported in the results.

A peak detection procedure was performed for each participant to measure peak latency for each component. Regarding amplitude measurements a mean amplitude method was applied. Mean amplitude rather than peak amplitude is less affected by noise, leading to more

efficient responses (Clayson et al., 2013; Luck, 2012). The mean amplitudes were calculated based on an interval between two points (25ms) on either side of the peak (i.e., a 50ms window) for each peak. This time window was based on the grand averaged ERPs at sites Cz, T7 and T8 (Anderson, Chandrasekaran, et al., 2010; J. A. Hämäläinen et al., 2011).

2.Scoping review

Temporal and Fronto-Central Auditory Evoked Responses in Children with Neurodevelopmental Disorders: A Scoping Review

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Abstract: At the cortical level, the central auditory neural system (CANS) includes primary and secondary areas. So far, much research has focused on recording front-to-central auditory evoked potentials/responses (P1-N1-P2), originating mainly from the primary auditory areas, to explore the neural processing in the auditory cortex. However, less is known about the secondary auditory areas. This review aimed to investigate and compare fronto-central and T-complex responses in populations at risk of auditory dysfunction, such as individuals with neurodevelopmental disorders. After searching the electronic databases (PubMed, Web of Science, Scopus, and Ovid), ten studies encompassing six neurodevelopmental disorders were included for the analysis. All experimental populations had atypical T-complexes, manifesting as an absence of evoked responses, shorter latency, and/or smaller amplitude. Moreover, in two experimental groups, dyslexia and attention deficit/hyperactivity disorder (ADHD), abnormal T-complex responses were observed despite the presence of normal fronto-central responses. The presence of abnormal T-complex responses in combination with normal fronto-central responses in the same population, using the same experiment, may highlight the advantage of the T-complex for indexing deficits in distinct auditory processes or regions, which the fronto-central response may not track.

Keywords: auditory evoked potentials; T-complex; primary auditory area; secondary auditory area

1. Introduction

Listening difficulties are commonly reported in children with neurodevelopmental disorders. In many cases, these challenges are not fully explained by disordered transduction in the cochlea or abnormal early processing along the auditory brainstem pathway. It is therefore likely that these listening challenges are associated with how auditory information is processed in the cortex. Auditory information enters the cortex through primary auditory areas and then projects to secondary and tertiary auditory areas [1,2]. The primary auditory cortex (PAC) is the first cortical region involved in acoustical processing and receives inputs from the thalamus and the medial geniculate body (MGB) [3,4]. The PAC can be segmented into three regions; from anterior to posterior, these regions consist of the planum polare (PP), Heschl's gyrus (HG), and the planum temporale (PT). HG is located on the supratemporal plane, extending diagonally from the superior temporal gyrus (STG) [5,6]. The STG encompasses the secondary auditory areas and anatomically interfaces with the lower-level auditory areas and higher-level structures contributing to language, music, and other forms of auditory processing [7]. The STG plays a special role in the neural analysis of speech sounds and phonological processing [8,9].

Impairments in the central auditory system may lead to poor listening abilities, mostly labeled as auditory processing disorder (APD) [10]. This

disorder affects around 5–7% of children [11–14]. APD and other neurodevelopmental disorders such as language impairment (LI), attention deficit/hyperactivity disorder (ADHD), and autism share a common characteristic: developmental delays, which can impair personal, social, academic, or occupational functioning [15]. Moreover, various developmental disorders may overlap with each other [16]. Multiple studies have shown that LI, ADHD, and autism are often comorbid with APD [16–19]. Hence, evaluating auditory processing, along with language, attention, and other cognitive abilities, in these populations is highly recommended [20]. Over the years, auditory evoked potentials (AEPs) have been utilized to explore impairments in the auditory cortex in these populations [21–25].

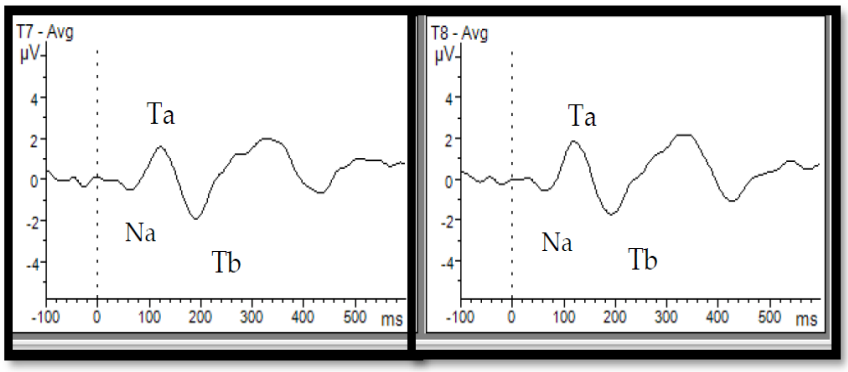

Figure 1. Auditory evoked potentials from the temporal lobe and T-complex. T-complex waveforms—consisting of successive Na, Ta, and Tb peaks— were recorded at the left (T7) and right (T8) temporal sites in response to a 1 kHz tone. This figure was derived from data collected at our Electrophysiology Lab, University of Ottawa, and has not been published before.

AEPs are series of changes in electrical brain activity triggered by acoustic stimulation. At the cortical level, there are two categories of AEPs: fronto-central and temporal responses [26,27]. Fronto-central responses include P1-N1-P2-N2 and are measured over fronto-central sites but arise mainly from primary auditory areas and index the acoustical processing of sound [27,28]. Numerous studies recording P1-N1-P2-N2 have indicated deficits in cortical auditory processing among individuals with neurodevelopmental disorders [22,29–32]. For instance, in a group of children with language impairments, smaller amplitudes and/or longer latencies of P1, N1, and P2 were observed compared to those of responses obtained from typically developing (TD) groups [29,33].

The T-complex, or temporal auditory response, is another AEP that corresponds to fronto-central responses in terms of timing but is recorded over temporal sites and consists of the Na-Ta-Tb sequence (Figure 1) [26,34,35]. Both fronto-central and temporal responses are sensitive to physical modulations of stimuli such as changes in intensity, interstimulus interval (ISI), and stimulus onset asynchrony (SOA) in children and adults [36–39]. For instance, consistent with fronto-central responses, the T-complex's amplitude increases with a longer SOA [40]. Despite sharing similar time windows, there are some differences between T-complex and fronto-central responses. In fact, P1-N1-P2 has a frontal topography, represents activity mainly in the primary auditory areas [41–43], and features a tangential dipole orientation [26,41,42]. In contrast, the T-complex has a temporal topography and reflects the activity of lateral temporal structures within the posterior lateral superior temporal gyrus, with a radial dipole orientation [26,35,41]. Maturational studies also suggest that the T-complex matures earlier than the fronto-central response (about 4–5 years old vs. 8–9 years old) and remains stable with increasing age [35,44,45]. Considering the developmental trajectory, it is thought that the T-complex relates to the more complex processing of sound and better correlates with speech and language processing as compared to fronto-central responses [40,44,46].

Multiple studies have demonstrated the effectiveness and reliability of the T-complex in assessing auditory processing at the cortical level [21,45,47]. The evaluation of fronto-central and T-complex waveforms in individuals with

auditory dysfunction is promising, as it can yield information regarding the origin of the deficit, specifically, whether it arises from within the superior temporal plane or the posterior lateral superior temporal gyrus [47].

To the best of our knowledge, no review has investigated and compared the properties of the T-complex and front-to-central responses of different populations with auditory dysfunctions. Therefore, this review aims to compile studies measuring T-complex and front-o-central responses in populations with neurodevelopmental disorders. Specifically, the first objective is to report and document the properties of T-complex responses in populations with neurodevelopmental disorders. The second objective is to explore the similarities and differences between the T-complex and fronto-central responses.

2. Methods

A scoping review was carried out in order to incorporate multiple types of studies with disparate methodologies [48]. Two reviewers (ZA and AK) were involved in the review process [49]. The second reviewer (AK) analyzed only articles included by the first reviewer, using a liberal accelerated approach. If a disagreement arose, a third reviewer was tasked with its resolution (FDL) [49].

In order to avoid missing any related articles, key words were selected in a manner designed to provide as many as studies as possible on the T-complex, with no preference for age or population characteristics. Then, the studies matched with the target population (neurodevelopmental disorder) were identified during the screening steps based on the inclusion criteria. The search terms relating to the T-complex included auditory or speech-evoked potential or response, electroencephalography (EEG) assessment, electrophysiological indices, neural encoding, cortical processing, neural activity, event-related potential (ERP) response, neural response, mismatch negativity, speech signal, oscillatory EEG response, CAEP, auditory neural integrity, event-related potential, auditory measure, electrophysiological measure, and electroencephalography.

Databases, including PubMed, the Web of Science, Scopus, and Ovid, were searched separately, and results were compiled in the Covidence database, where search strategies were dated and organized. Conference papers, master's dissertations, and doctoral theses (gray literature) were searched through ProQuest Dissertations and Theses Global, Google Scholar, and Theses Canada.

This review included literature published until 2023. The method for screening and finding related studies followed the PRISMA process [50]. In the initial screening, reviewer 1 (ZA) retained or rejected articles based only on title analysis. Both reviewers (ZA and AK) completed the second and third screenings; in these steps, the eligibility was based on abstract and full-text analyses, respectively. The exclusion and inclusion criteria are described in Appendix A. According to the goals of this review, only studies with participants with neurodevelopmental disorders were included. Also, because using a translator was not feasible for this study, only articles in English were selected.

3. Results

From the databases, a total of 164 articles were identified. Following the initial screening step and removal of duplicates (n = 91), 73 articles remained. Among the 73 articles, 23 articles were included based on title evaluation (Bishop et al., 2012; Bruneau et al., 2003; Bruneau et al., 1999; Bruneau et al., 2015; Cacace et al., 1988; Carrillo-de-la-Peña, 1999; Čeponien et al., 1998; Clunies-Ross et al., 2015; Clunies-Ross et al., 2018; Gomes et al., 2012; Groen et al., 2008; J. A. Hämäläinen et al., 2011; Ponton et al., 2002; Shafer et al., 2011; Silva et al., 2020; Rinker et al., 2021; Taylor et al., 2003; Tonnquist-Uhlén, 1996; Tonnquist-Uhlen et al., 2003; Wolpaw & Penry, 1975, 1977; Wagner et al., 2016; Woldorff & Hillyard, 1991).[18,21,23,26,28,29,35–37,39,40,45,46,51–59]. As a result of abstract screening, 13 studies were excluded, as they did not include abnormal populations among their participants (Bruneau et al., 2015; Cacace et al., 1988; Carrillo-de-la-Peña, 1999; Čeponien et al., 1998; Clunies-Ross et al., 2015; Clunies-Ross et al., 2018; Ponton et al., 2002; Silva et al., 2020; Tonnquist-Uhlen et al., 2003; Wolpaw & Penry, 1975, 1977; Wagner et al., 2016; Woldorff & Hillyard, 1991).[26,35,37,39,40,45,46,56–60]. The remaining ten articles

underwent thorough full-text screening, and all of them met the eligibility criteria and were consequently included in this study. The PRISMA flow diagram is presented in Figure 2. The data, encompassing information about experimental and control groups, event-related potentials (ERPs) of interest, and the results, have been succinctly summarized in Table 1.

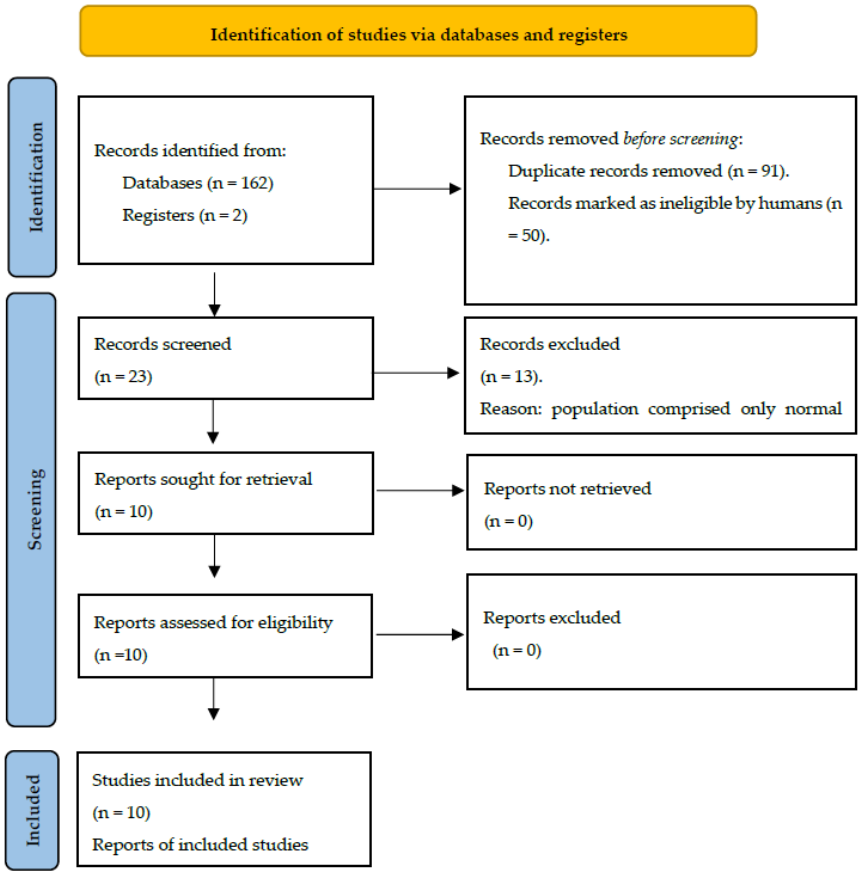

Figure 2. Flow chart for the scoping review process [50].

Table 1. (**A**) Summary of included studies. (**B**) Summary of results.

A					
Authors	**Year**	**Experimental Group**	**Control group**	**Size and Age**	**AEPs**
Bishop, Hardiman, and Barry [29]	2012	Specific language impairment group	Normal children and teenagers	16 children (7–11) and 16 teenagers (12–16)	Ta
Bruneau, Roux, Adrien, and Barthélémy [21]	1999	Autism group and intellectual disability group	Normal children	16 autistic children (4–8) 16 intellectually disabled children (4–8) (4–8)	N1b, N1c (Tb)
Bruneau, Bonnet-Brilhault, Gomot, Adrien, and Barthélémy [18]	2003	Autism group	Normal children	Autism: 16 children (4–8) Normal: 26 children (4–8)	N1c (Tb)
Gomes, Duff, Ramos, Molholm, Foxe, and Halperin [51]	2012	ADHD group	Normal adults and children	ADHD: 15 children (7.3–12.5) Normal: 16 adults (18–45) and 15 children (8 7.5–13.5)	P1, Ta
Groen, Alku, and Bishop [52]	2008	Down syndrome group	Normal children	DS: 19 children (10–12). Normal: 19 children (10–12)	Ta-Tb
Hämäläinen, Fosker, Szücs, and Goswami [36]	2011	Dyslexia group	Normal adults	11 adults (20–54)	N1-P2, Na-Ta-Tb
Rinker [53]	2021	Developmental language disorder, monolingual German children, and bilingual Turkish German children	Normal monolingual and bilingual children	14 monolingual German children with DLD (6 49–73 months) 16 monolingual German children (4.2–6) 12 Turkish German children (55–81 months)	Na-Ta-Tb
Shafer [54]	2011	Specific language impairment	Normal children	22 SLI children 32 normal children	Na-Ta-Tb
Taylor [55]	2003	Dyslexia group	Normal children	DS: 11 children (average age = 10.08) Normal: 11 children (average age = 10.29±)	N1a (Na)-N1b-N1c (Tb)
Tonnquist-Uhlen [23]	1996	Severe language impairment group	Normal children	Normal: 20 children (9–15) SLI: 20 children (9–15)	Ta-Tb, N1

B					
			Results		
Author	**Year**	**Reported Measurements**	**Results Related to the T-Complex (Na-Ta-Tb), Amplitude**	**Results Related to the T-Complex (Na-Ta-Tb), Latency**	**Results Related to the Fronto-Central Response (P1-N1-P2)**
Bishop, Hardiman, and Barry [29]	2012	Amplitude of Ta	Ta in SLI was smaller ($p < 0.001$) than Ta in TD. This difference was not significant with tone stimuli ($p = 0.1$) but highly significant with speech stimuli ($p = 0.002$).	-	Not measured
Bruneau, Roux, Adrien, and Barthélémy [21]	1999	Amplitude and latency of N1a, N1b, and N1c	The amplitude of N1c was smaller for AUT than for RET and normal ($p < 0.0001$). N1b and N1c showed longer and smaller amplitudes among AUT compared to normal children ($p < 0.0001$).	The longest latency of N1c was seen for AUT. RET and normal did not differ in N1c latency. N1b and N1c showed longer latency among AUT compared to normal children ($p < 0.0001$).	The smallest and longest N1b was observed in the RET group ($p < 0.02$). There was a smaller N1b in AUT than in the normal group ($p < 0.0001$). The latency of N1b was not different between normal and AUT groups.
Bruneau, Bonnet-Brilhault, Gomot, Adrien, and Barthélémy [18]	2003	Amplitude and latency of Tb	A smaller amplitude of Tb was seen in AUT than in normal ($p < 0.0005$ and $p < 0.02$, respectively).	A longer latency of Tb was seen in AUT than in normal ($p < 0.0005$ and $p < 0.02$, respectively).	
Gomes, Duff, Ramos, Molholm, Foxe, and Halperin [51]	2012	Amplitude of Ta and P1	Ta had a smaller amplitude ($p < 0.005$) in ADHD for attended and unattended conditions.	-	There was no difference in the P1 peak between ADHD and normal groups.
Groen, Alku, and Bishop [52]	2008	Amplitude and latency of Ta and Tb	A larger contralateral Tb was seen in typical children ($p = 0.01$), not in DS ($p = 0.5$).	A shorter contralateral Ta was seen at T7 in typical children ($p = 0.00$) but not in DS children ($p = 0.5$). Tb was 12 ms delayed in the DS group compared to the normal group ($p = 0.001$).	Not measured
Hämäläinen, Fosker, Szücs, and Goswami [36]	2011	Amplitude and latency of Na, amplitude of Ta and Tb, amplitude and latency of N1 and P2	No group difference was seen based on the Na peak. The amplitude of Na did not show the effect of rise time (RT) changes. Only the T-complex showed a group difference as a function of sound rise time. Participants with dyslexia did not show a similar reduction pattern in Tb amplitude to the normal group with increasing rise times ($p < 0.005$).	-	N1 and P2 showed typical morphology in adults with and without dyslexia. The changes in N1 and P2 as a function of rise time were typical.
Rinker [53]	2021	Latency of Na and Tb, amplitude of Ta	The difference between DLD and TD was significant only with vowel stimuli.	Regarding Na, bilingual had a longer latency than TD and DLD ($p = 0.01$). Tb had a shorter latency ($p < 0.001$)	Not measured

			The group effect was dependent on the stimuli type for Ta; for vowel stimuli, Ta was smaller for DLD and bilingual than TD ($p = 0.04$). In tone condition, Ta was smaller ($p < 0.05$) for bilingual than for DLD and TD (DLD and TD did not differ).	for DLD than TD and bilingual (bilingual and TD did not differ).	
Shafer [54]	2011	Amplitude of Ta and peak-to-peak amplitude of Na-Ta and Ta-Tb	Ta was smaller in the LI group than in the normal group ($p < 0.05$). The peak-to-peak amplitudes, including Na-Ta and Ta-Tb, were also smaller in the LI group than in the normal group ($p < 0.05$).	-	Not measured
Taylor [55]	2003	Amplitude and latency of N1a, N1b, and N1c	N1a was larger in dyslexia than in the normal group. The N1c amplitude was larger for mistuned than tuned stimuli ($p < 0.025$), with no group effects or interactions.	There was an interaction with stimuli: When the stimuli were tuned ($p < 0.018$), N1a was shorter for dyslexia than normal, and vice versa for mistuned stimuli ($p < 0.02$). N1c was shorter for the dyslexic than control children ($p < 0.029$) (with no interaction with stimuli), but only over the right lateral temporal electrode.	N1b did not show a group difference between normal and dyslexia groups. This response was larger for mistuned stimuli.
Tonnquist-Uhlen [23]	1996	Amplitude and latency of Tb, amplitude of Ta	A smaller amplitude of Tb was reported in LI children, but the results did not reach significance.	Ta was longer in LI children ($p < 0.001$). Tb was longer in the LI group than normal ($p < 0.0005$–0.001). In both groups, there was a shorter Tb at T7, regardless of the ear.	N1 was prolonged in the LI group. However, the difference om N1 latency between the normal and experimental groups was not as high as the difference in Tb latency. The difference for Tb was almost 20 ms, and the difference for N1 was 5–10 ms

AEP: auditory event-related potential, ADHD: attention disorder/hyperactivity, AUT: autism with intellectual disability, DLD: developmental language disorder, DS: Down syndrome, LI: language impairment, RET: intellectual disability without autism, SLI: specific language impairment.

3.1. Study Characteristics

3.1.1. Type of Disorder

Four studies were found to assess LI using the T-complex [23,29,53,54]. A further study assessed ADHD [51]; two assessed dyslexia [36,55]; one assessed Down syndrome (DS) [52]; and two assessed autisms [18,21]. These neurodevelopmental disorders often have comorbidity with APD, potentially sharing symptoms related to auditory dysfunction [16,56]. Notably, there was no study investigating the T-complex in children diagnosed with auditory processing disorder.

3.1.2. Age

Only one study examined the T-complex in adults, while the remaining studies focused on child participants. One study included both children (aged 7–12 years) and teenagers (aged 13–16 years) [29]. The age range varied from the youngest participant at 4 years old to the oldest teenager at 16 years old [29]. However, among the included studies, one study was conducted on adults aged between 18 and 45 years old (average age was 25 years old).

3.1.3. Stimuli

Different stimuli were used in the included studies. Four studies used simple tone stimuli [18,21,33,51]; two studies used speech and tone stimuli [29,52]; two studies used speech stimuli [44,53]; one study used simple tones with varying rise times [36]; and another study used a complex tone, which was developed by combining 12 pure tones [55]

3.1.4. Recorded ERPs

Among the studies, only two investigated all three T-complex components (Na-Ta-Tb) [36,53]. Ta and Tb were investigated in two studies [23,52]. In other investigations, only one peak was of interest. For instance, Ta was the sole component examined in two studies [29,51], while some studies reported only the pattern of Tb [18,21]. Additionally, Na was explored in three studies [36,53,55].

Responses recorded over fronto-central sites were reported in five studies. One reported on P1 [51]; four studies reported on N1 (also called N1b) [21,23,36,55]; and one reported on P2 [36].

3.1.5. Findings

Overall, the review of findings revealed abnormal T-complex responses in children with neurodevelopmental disorders. This pattern signifies neural auditory dysfunction localized in the temporal lobe across multiple neurodevelopmental disorders. Most critically, compared to the T-complex, only two studies reported atypical fronto-central responses in children with neurodevelopment disorders [21,23]. Smaller amplitudes of N1 and longer latencies of N1 were observed in children with autism [21] and LI [23] compared to those in TD, respectively. The details of the results of each study are presented in Table 1.

Regarding the T-complex, it seems that both the latency (longer and/or shorter) [21,23,52,53,55] and amplitude (smaller and/or larger) [18,29,51,53–55] of the T-complex showed atypical patterns. Interestingly, specific deviations were observed in various components of the T-complex across different disorders. For instance, an atypical Tb was observed in the dyslexia group, despite a normal Na and Ta [36]. Na and Tb were abnormal in another study on dyslexia [55]. The Ta component was most affected in populations with language impairment [29,53,54] and ADHD [51]. Studies on autism and DS indicated atypical Tb components [21,52]. Furthermore, findings indicated discrepancies between T-complex and fronto-central responses in studies investigating dyslexia [36,55], language impairment [23], ADHD [51], and autism [21]. There were abnormal patterns of the T-complex, despite normal fronto-central responses [36,55].

4. Discussion

This study reviewed the evidence of changes in T-complex and fronto-central responses in neurodevelopmental disorders. Subsequently, the findings on Na, Ta, and Tb patterns were elaborated on, as well as the different patterns observed between the T-complex and the fronto-central responses.

4.1. Na Pattern

Na represents the early stage of the acoustical processing of sounds [28,40,47]. In children with dyslexia, Na was larger but occurred at a similar latency compared to that in controls. This heightened response in the dyslexic population may suggest that individuals with dyslexia require greater brain activation and effort for the same early perceptual processing of auditory stimuli compared to the control group [55]. Alternatively, enhanced auditory evoked responses could be related to a failure of inhibition. Older adults normally have larger auditory evoked potentials compared to younger adults [57,58]. This age-related increase in the amplitude of the AEPs has been interpreted as a decline in the inhibitory system [59,60]. Top-down inhibition exerts a modulatory effect on sensory processing by suppressing irrelevant information from further processing [61,62]. A decreased capacity for inhibition may lead to reduced listening abilities [58,63]. This explanation may justify the larger auditory responses in children with dyslexia, as studies consistently have documented inhibitory impairments in this clinical group [64,65].

While Taylor et al. (2003) reported an effect of dyslexia on Na amplitude [55], other studies have not replicated this result [36]. Differences in stimuli complexity and experimental design may contribute to variations in the observed results across these studies. Taylor's study employed an active paradigm involving complex stimuli composed of 12 pure tones, while Hämäläinen's study utilized a passive paradigm featuring a single pure tone (500 Hz) with varying linear rise times [36,55]. It is therefore possible that the impact of dyslexia on Na is related to attending to complex stimuli in the study of Taylor et al., 2003. Moreover, in the study of Hämäläinen et al., 2011, despite no group effect on Na recorded in the passive condition, there was a significant effect of dyslexia on the behavioral task of discriminating stimuli with high and low rise times. In other words, the dyslexia group performed worse than the normal group when required to attend to the sound [36]. These results may suggest that the deficits lie in top-down effects, underlying neural pathways from higher centers to the auditory areas of the brain. More research and more specific study designs are warranted to differentiate between bottom-up and top-down processing in these populations.

4.2. Ta Pattern

The Ta component represents acoustical discrimination [28,47]. The Ta component had a smaller amplitude and longer latency in individuals with LI compared to the healthy control group [23]. A smaller Ta was consistent with three subsequent studies focusing on the same disorder [29,53,54]. Furthermore, Shafer et al. (2011) reported that the peak-to-peak amplitudes, including Na-Ta and Ta-Tb, were smaller in individuals with LI compared to healthy controls [54]. This suggests auditory discrimination impairments in people with LI. This deficit could lead to phonological processing disorders in this population [23,29]. Interestingly, the atypical pattern in people with LI was observed only with speech stimuli and not with tones, highlighting the impaired verbal processing in children with language disorders [29,53]. Similar to LI, the ADHD population exhibited a reduced Ta [51]. The atypical appearance of Ta, which indicates impaired auditory processing in the CANS, may suggest that auditory processing disorder can co-exist with ADHD [16,51,66,67].

Accordingly, reduced Ta amplitudes could reflect auditory dysfunction in ADHD and LI populations and suggest a robust association between language impairment and altered T-complex responses, particularly in the Ta component.

4.3. Tb Pattern

Tb is the third component of the T-complex and indexes auditory processing in secondary auditory areas [26,28,40]. Taylor et al. (2003) reported that the dyslexia group had a shorter latency of Tb compared to healthy controls [55]. This faster brain response may reflect the superficial and inappropriate processing of sound in the dyslexic population [55]. Moreover, this abnormally short latency of Tb was observed only in the right hemisphere [55]. Such findings align with neuroimaging studies reporting greater right- than left-hemisphere activity in dyslexia, which leads to faster but less accurate processing [68,69].

In children with autism, Tb was smaller and delayed compared to that in healthy controls, and the differences in Tb indexed the magnitude of the auditory impairments [18,23]. The prolonged latency of Tb may reflect slower transmission in synaptic connections of the secondary auditory areas, which may be in line with studies showing reduced blood flow in the temporal areas of this population [70,71]. Additionally, children with autism exhibited atypical inter-hemispheric differences as the intensity was modulated. Specifically, the amplitude of Tb increased more prominently in the right hemisphere than in the left hemisphere, indicating rightward lateralization. This is in contradistinction to the healthy controls, who exhibited a leftward dominance with increasing intensity [21]. This difference may reflect dysfunction in the left hemisphere and might imply the reorganization and retuning of the left and right hemispheres for amplitude processing in autism, indicating a right-hemisphere compensation for left-hemisphere dysfunction [21]. These results align with behavioral and electroencephalography (EEG) studies demonstrating a rightward lateralization of auditory processing in the autistic population [72,73].

People with Down syndrome (DS) also exhibit atypical the lateralization of auditory neural processing [52]. Specifically, Tb is delayed in people with DS. This delay is attributed to myelination deficits in this population [52]. The results also highlighted the absence of a contralateral effect on Tb. Typically, the contralateral neural pathway leads to a shorter or larger Tb response, particularly when sound is presented to the right ear, and neural activity is recorded over the left hemisphere (right ear–left hemisphere advantage of Tb) [26,39,40]. In DS, the benefit of contralateral over ipsilateral recording was not observed for Tb. These results suggest a more distributed lateralization of neural function in people with Down syndrome [52].

Lateralization is a key feature of auditory processing. Multiple studies using different techniques, including behavioral observations, functional MRI, and magnetoencephalography (MEG), have documented hemispheric lateralization at higher and lower levels of the cortex [74–77]. In other words, the functional lateralization of the brain is not limited to high-level cognitive processes like language; it starts from the lower-level neural processing of acoustical features (e.g., frequency, duration, interval, and intensity) [77–79]. Moreover, converging evidence illustrates that the left and right auditory cortices are asymmetrically linked to temporal and spectral processing, with the right hemisphere more sensitive to spectral features and the left to temporal features [80–82]. Tb may reflect the asymmetric lateralized auditory function at the level of secondary areas. Moreover, atypical development may lead to the abnormal lateralization of temporal and/or spectral processing generated in the secondary auditory cortex, which has been observed in people with autism and DS.

4.4. Discrepancy Between Patterns of T-Complex and Fronto-Central Responses

The second goal of this review was to compare the T-complex and fronto-central responses in children with developmental disorders. Three articles reported abnormal T-complex responses despite normal fronto-central responses.[36,51,55]. Contrary to typically reduced amplitudes of P1 and N1 with a longer latency rise time, dyslexia did not show the latency effect on Tb amplitude [36]. Another study also demonstrated group effects on the amplitude of Na and the latency of Tb; the fronto-central response (N1b, a subcomponent of N1) did not show differences between TD and dyslexia groups [55]. In the case of ADHD, abnormal Ta responses were reported

despite the normal P1 response [51]. The typical results of fronto-central responses have been similarly documented in individuals with APD [83,84]. However, in the aforementioned studies, only fronto-central responses are reported, so there is no such comparison between T-complex and fronto-central responses to confirm the current findings.

Among the included studies, two other studies indicated that the T-complex showed more sensitivity in tracking auditory dysfunction than the fronto-central response [21,23]. In LI, although similar to N1, the Tb latency was longer, and the latency difference recorded for Tb was higher than that for N1 (the difference for Tb was almost 20 ms; the difference for N1 was 5–10 ms) [23]. Moreover, in people with autism, N1b was smaller than in the standard group, but Tb was both smaller and more delayed compared to that in healthy controls.[21]. Accordingly, Tb may be more sensitive to auditory impairments than the fronto-central response in LI and autism groups [21,23].

The divergent patterns observed between T-complex and fronto-central responses may serve to differentiate the origins of auditory deficits. The predominant generation of the T-complex occurs in the secondary auditory areas in the posterior and lateral parts of the superior temporal gyrus (Brodman 42 and 22) [35,42], while P1-N1-P2-N2 originates from the primary auditory areas in Heschl's gyrus (Brodman 41) and the associated areas [35,85,86]. Therefore, an abnormal T-complex signifies the involvement of the secondary and associated auditory areas in a specific disorder. Additionally, different origins and patterns may suggest that the T-complex manifests different auditory processes that are distinct from those reflected in fronto-central responses. For instance, Tb could index abnormal processing related to varying rise time and intensity, even in the presence of normal fronto-central responses, as well as reflecting the hemispheric lateralization of auditory processing at secondary auditory areas.

4.5. Limitations and Future Direction

Although T-complex differences were related to auditory dysfunction in all experimental groups, the patterns of all components of the T-complex were not reported. Some studies investigated only one component. It is unclear if other components would show the same pattern. For instance, the study of Shafer in 2011 investigated only Ta. Na and Tb were the only components of interest in the studies of Hämäläinen et al., 2011, and Bruneau et al., 1999, respectively. More research investigating all T-complex components is warranted to confirm the results.

Also, fewer than half of the studies reported and compared both fronto-central and temporal responses. It is not clear whether the conflicting results between these two AEPs could be confirmed in other disorders. Moreover, no study has investigated and compared these responses in APD. Similar to the results of fronto-central responses in dyslexia and ADHD discussed in this review, the literature has indicated that AEPs recorded at fronto-central sites did not consistently track auditory impairments in APD groups. One reason may lie in the fact that the related studies on APD have not assessed the T-complex. Hence, researching the effect of APD on the T-complex and comparing it with the fronto-central response using different methodologies may advance the knowledge about the origins of the dysfunction in APD and introduce a consistent biomarker for APD.

5. Conclusions

This study revealed that the T-complex can index auditory processing impairments at the cortical level in various neurodevelopmental disorders, even when there is a normal pattern of the fronto-central response. These opposite patterns may emphasize the different generators between T-complex and fronto-central responses.

Also, these findings may suggest that the neural generators of the T-complex contribute to distinct auditory processes that differ from those indexed by the fronto-central response and neural generators. Atypical T-complex patterns in all investigated experimental groups may also highlight the potential of the T-complex

as a sensitive biomarker for identifying auditory processing abnormalities across a range of neurodevelopmental conditions.

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Abbreviations

Attention deficit/hyperactivity disorder (ADHD), auditory evoked potentials (AEPs), auditory processing disorder (APD), autism with intellectual disability (AUT), central auditory neural system (CANS), developmental language disorder (DLD), Down syndrome (DS), Heschl's gyrus (HG), interstimulus interval (ISI), language impairment (LI), medial geniculate body (MGB), primary auditory cortex (PAC), planum polare (PP), planum temporale (PT), intellectual disability without autism (RET), specific language impairment (SLI), stimulus onset asynchrony (SOA), superior temporal gyrus (STG), typically developing (TD).

Table A1. Inclusion and exclusion criteria.

	Inclusion Criteria	Exclusion Criteria
Population (age and condition)	Children or adults, normal and neurodevelopmental disorders such as attention deficit/hyperactivity disorder, language impairment, and autism	The participants were in general health and the study did not include clinical groups
Evaluation	T-complex	Subcortical responses (ABR), ASSR, and other late auditory responses such as MMN, P300, and N400
Publication type	Peer-reviewed journals and gray literature published after 1950 and only in English	Any unscientific papers, magazines, editorials, and manuals or text in a language other than English
Outcome	Peak and latency of Na-Ta-Tb or Ta-Tb	No results related to T-complex components

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Table A2. The stimuli and recording features of included studies.

	Authors	Year	Title	Type (Single or Paired, Tone, Speech, or Complex Tone (Composed of Multiple Tones))	Intensity	Transducer	Delivery and Recording	ISI or SOA	Reference	Sample Rate	Filter
1	Bishop, Hardiman, and Barry	2012	Auditory Deficit as a Consequence Rather than Endophenotype of Specific Language Impairment: Electrophysiological Evidence	Single, 1000 Hz and /bah/	86.5 dB SPL	Headphone	Monaural; the study did not mention the mode of stimulation	SOA, 1 s	Mastoid right or left	500 Hz	0.1–70 Hz
2	Bruneau, Roux, Adrien, and Barthélémy	1999	Auditory associative cortex dysfunction in children with autism: evidence from late auditory evoked potentials (N1 wave ± T-complex)	Single, 750 Hz tone burst	50, 60, 70, and 80 dB SPL	Two speakers	Binaural	ISI, from 3 to 5 s	Linked earlobes	250 Hz	NMA
3	Bruneau, Bonnet-Brilhault, Gomot, Adrien, and Barthélémy	2003	Cortical auditory processing and communication in children with autism: electrophysiological/behavioral relations	Single, 750 Hz tone	50, 60, 70, and 80 dB SPL	Two speakers	Binaural	ISI, 3 to 5 s	Linked earlobes	250 Hz	NMA
4	Gomes, Duff, Ramos, Molholm, Foxe, and Halperin	2012	Auditory selective attention and processing in children with attention deficit/hyperactivity disorder.	Single, four types of two standards [1000 Hz] (low channel) and two [2000 Hz]	82 dB SPL	Insert earphones	Monaural; the study did not mention the mode of stimulation	SOA, 850 to 1150 ms	Tip of the nose	0.5–70 Hz	500 Hz

5	Groen, Alku, and Bishop	2008	Lateralisation of auditory processing in Down syndrome: A study of T-complex peaks Ta and Tb.	(high channel) Single, vowel /a/, 576 Hz for the simple tone; the complex tone was composed of four tones of 576, 1055, 2589, and 3163 Hz	50 dB SPL	Insert earphones	Monaural, ipsi and contra conditions	ISI, 1550 ms	left mastoid	1000 Hz	0.1–70 Hz.
6	Hämäläinen, Fosker, Szücs, and Goswami	2011	N1, P2 and T-complex of the auditory brain event-related potentials to tones with varying rise times in adults with and without dyslexia.	Single, 500 Hz with varying linear rise times: 10, 30, 60, 90, and 120 ms	75 dB SPL	Insert headphones	Binaural	ISI, 2.5 – 3.5 s	Vertex	500 Hz	0.1–200 Hz
7	Rinker	2021	Language Learning Under Varied Conditions: Neural Indices of Speech Perception in Bilingual Turkish-German Children and in Monolingual Children With Developmental Language Disorder (DLD)	Single, vowel [ε]	90 dB SPL	Headphones	Binaural	ISI, 650 ms	Left earlobe	500 Hz	0.1–70 Hz
8	Shafer	2011	Evidence of deficient central speech processing in children with specific language impairment: The T-complex	Various stimuli: “the” repetition standard /E/ standard /E/ first word in a pair of words.	65 dB SPL, 86.5 dB SPL, 86.5 dB SPL, 70 dB SPL	Insert phone	NM	ISI, 625 ms, 550 ms, 350 ms, 2000 ms	Nose	512 Hz	0.05 – 100 Hz
9	Taylor	2003	Neurophysiological Measures and Developmental Dyslexia: Auditory Segregation Analysis	Five complex sounds were obtained by combining 12 pure	70 dB SPL	Headphones	Binaural	ISI, 1.1 s	Cz	500 Hz	0.1–30 Hz

				tones. Four of the stimuli were mistuned, and their third harmonic was increased by 2, 3, 8, or 16% of original values.								
10	Tonnquist-Uhlen	1996	Topography of Auditory Evoked Long-Latency Potentials in Children with language impairment	Single, 500 Hz	75 dB HL	Insert phone	Monaural, ipsi and contra conditions	ISI, 1.0 s	Chin	500 Hz	0.1–60 Hz

dB = decibel, SL = sound level, nHL = normal hearing level, SPL = sound pressure level, Hz = Hertz, ipsi = ipsilateral, contra = contralateral, NM = not mentioned, N = number of articles, ISI = interstimulus interval, SOA = stimulus onset asynchrony, s = second, ms = millisecond.

3.Behavioral tests for APD evaluations

Categories of behavioral central auditory tests, processes assessed, and selected examples of each.

Test category	Processed assessed	Examples
Dichotic speech tests	Binaural separation, Binaural integration, Binaural fusion	Dichotic Digits, Dichotic Consonant-Vowels, Staggered Spondaic Word test (SSW), Competing Sentences, Dichotic Rhyme
Temporal processing tests	Temporal ordering/sequencing, Frequency discrimination, Duration discrimination, Temporal resolution	Duration Patterns, Frequency Patterns, Gaps-In-Noise (GIN), Random Gap detection
Monaural low-redundancy speech tests	Auditory closure	Low-Pass Filtered Speech, Time Compressed Speech with and without Reverberation
Binaural interaction tests	Binaural interaction, Localization/lateralization	Binaural Fusion tests, Masking Level Difference (MLD), Listening in Spatialized Noise-Sentence Test (LISN-S)
Auditory discrimination tests	Auditory discrimination	Frequency and Duration just noticeable differences (JNDs), Speech-sound discrimination

4.Consent form

uOttawa

Evaluation of frontocentral and temporal auditory evoked potential in children with auditory processing disorder

Consent form

Université d'Ottawa
Faculté des sciences de la santé
École des sciences de la réadaptation
University of Ottawa
Faculty of Health Sciences
School of Rehabilitation Sciences

I ……. am invited to participate in the research project described in this form. The data will be provided by the private practice; APD Ottawa, 1090 Ambleside Drive K2B 8G7.

I am being asked to participate in the study as titled above. I have been chosen as a participant because I have Auditory Processing Disorder (APD). The objective of the study is to analyze results of peripheral and central auditory tests performed on APD population. The results of this study aim to identify a reliable biomarker of APD of the auditory function, and therefore, provide guidance for better diagnosis and future rehabilitation possibilities.

I understand that the researchers of this study will have access to my personal health information related to the auditory processing tests. So, I understand that my test results regarding peripheral and central auditory tests performed on my medical files will be released to the researchers conducting the study. In order to ensure data confidentiality, only the researchers mentioned below will have access to the collected data.

I understand that my identity will not be revealed and that the number assigned to my file will ensure this anonymity unless I choose otherwise, in which case, I consent to having my identity revealed.

I also understand that there are no risks associated with participation in the study.

At any point during the study, I can choose to withdraw my participation without any consequences, in which case, my test results and data will immediately be destroyed.

In order to ensure data confidentiality, only the researchers mentioned below will have access to the data. I understand that under no circumstances will my identity be revealed and that a code assigned to my file will ensure this anonymity.

Finally, I am aware that the collected data will be locked away under lock and key in the hearing science laboratory located in room 280, at the Lees campus, and that they will be destroyed five years after the publication of results.

For additional information or questions regarding this research, I may contact any of the researchers involved in this study; Amineh Koravand, Zohreh Ahmadi. Their contact information is found below.

☎ 613-562-5436
613-562-5428
200Lees Avenue, Ottawa, ON K1N 6N5
www.uOttawa.ca

What will happen during the appointment?

Firstly, we will evaluate your audition capacities, which will take approximately 5 to 10 minutes. This step consists of two parts:

1) Filling out a questionnaire

A questionnaire (APDQ) about problems involving language development and language proficiency, attention deficits, audition problems and over all general development, will be filled in by parents.

2) Audiological screening

This screening includes measures that will allow us to determine the level of hearing ability. It includes these steps:

Otoscopy. With the help of a special lamp, we will look inside the ears in order to make sure nothing prevents us from doing the audition tests, like the presence of wax, redness, etc. You have nothing to do while we do this verification.

Screening audiometry. We will also verify how well you hears by presenting very low intensity sounds using headphones. You will be asked to respond to the sound by either pressing a button or playing with a toy.

Secondly, the event-related potential test will be done. The procedure has been inserted in the following.

Active part: Lay relaxed in a chair while responding to the sounds. Child needs to press right button when they hear ta and right button when they hear da. A cap with several electrodes will be placed on the head and gel will be inserted through the cap.

Passive part: Lay relaxed in a chair while watching a NetFlix film of their choice while a recording of the brain is made. A cap with several electrodes will be placed on the head and gel will be inserted through the cap.

The sounds used while doing the screening are safe and will not damage the ears. The analysis of the results will be done by an Audiologist or students in Audiology who will be under the supervision of an Audiologist.

For all information, requests or complaints regarding the ethical conduct of this study, enquiries can be addressed to the Protocol officer for ethics in research at the following contact information: Protocol officer for ethics in research, University of Ottawa, Tabaret Hall, 550 Cumberland, room 154, Ottawa, Ontario K1N 6N5, phone number: (613) 562-5387. e-mail: ethics@uottawa.ca. There are two copies of the consent form, one of which I may keep.

I, ……, have carefully read the content of this letter and understand that I will be giving access to my tests results on peripheral and central

auditory processing.

I ……. hereby accept to give access to my information for the research project as described in this form.

Access to Health Information

I hereby give researchers of this study, my permission, to obtain information pertinent to auditory processing and hearing from the APD Ottawa clinic: □Yes □ No

I consent to giving access to my information in this research project:

Researcher's signature: _____Date: _____

Participant's signature: … … … . Date: _____

☐ I do not consent to being recognizable in any published paper that will come from the data analyzed.

☐ I consent to be recognizable in any published paper that will come from the analyzed data.

I would like to receive a summary of the results of this research.

Please send it to the following address:

_____

_____

_____

☐ I do not want to receive a summary of the results of this research.

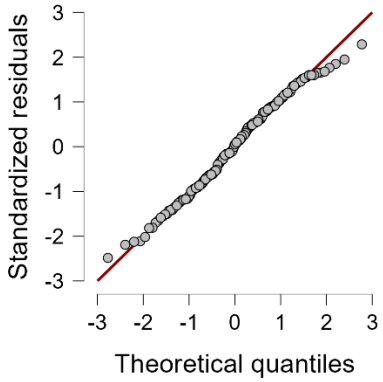
☐ I authorize the researchers to solicit my participation in future studies.

Names of researchers: Amineh Koravand, Ph.D., Zohreh Ahmadi, M.Sc., Ph.D. (S). University of Ottawa, Faculty of Health Sciences, School of Rehabilitation Sciences, 200Lees Avenue, Ottawa, ON K1N 6N5.

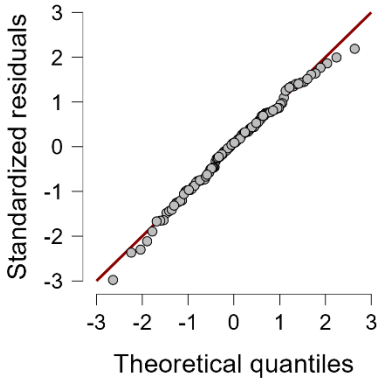
5. Assessment of the normality assumption in children's studies

A.Q-Q Plot Results for Assessment of Normality

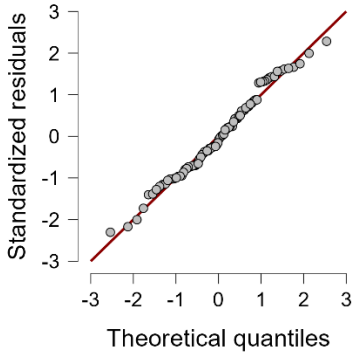
Q-Q Plot-Ta



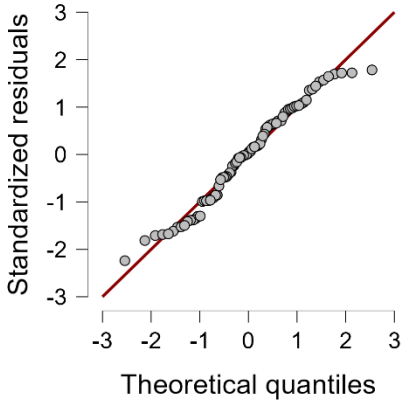
Q-Q Plot-Tb



Q-Q Plot-P1

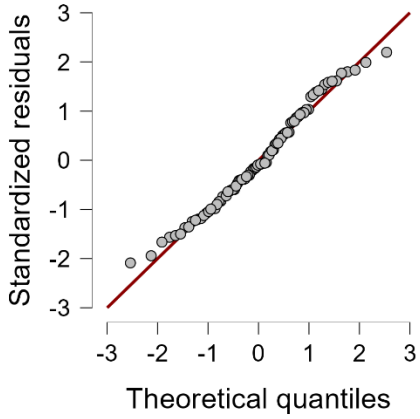


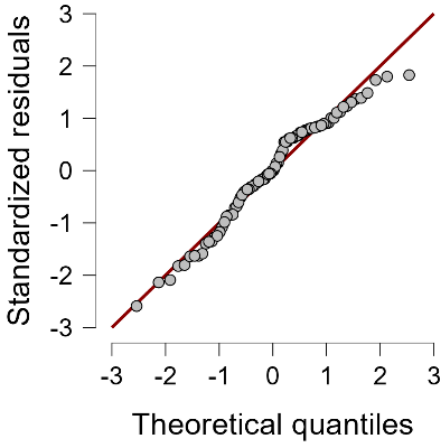
Q-Q Plot-N1



Q-Q Plot-P2

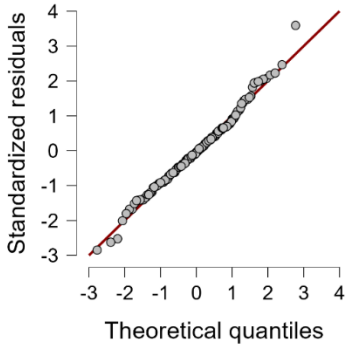
Q-Q Plot-N2



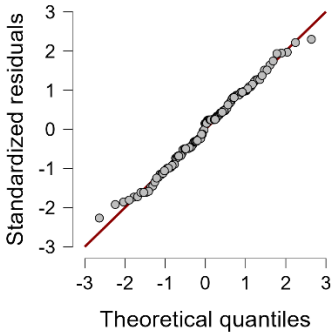


Assumption check, Latency data

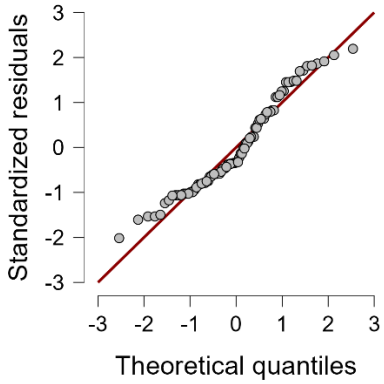
Q-Q Plot-Ta



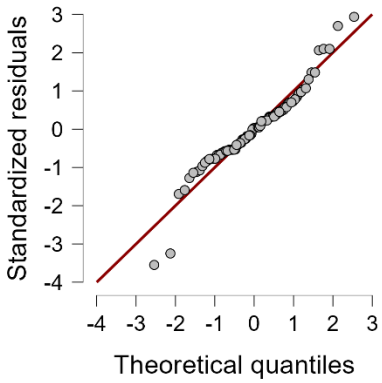
Q-Q Plot-Tb



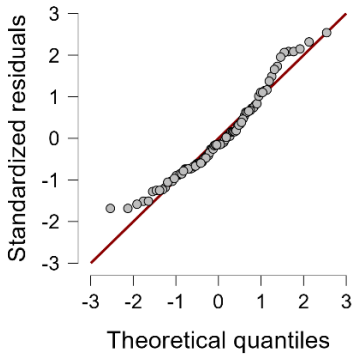
Q-Q Plot-P1



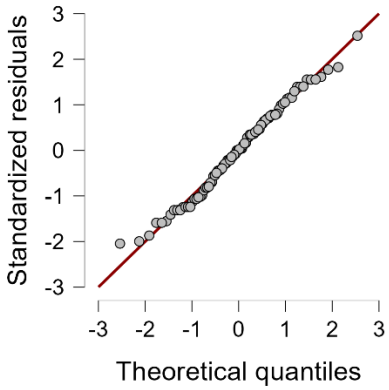
Q-Q Plot-N1



Q-Q Plot-P2



Q-Q Plot-N2



Quantile–Quantile (Q–Q) plots for the amplitude and latency measures of the auditory cortical responses (Ta, Tb, P1, N1, P2, and N2). Visual inspection of the Q–Q plots indicated that the majority of the variables were approximately normally distributed.

B. Shapiro–Wilk Test Results for Assessment of Normality

B1. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response amplitudes.

	1k Hz -Q- P1- Cz	1k Hz -Q- N1 -Cz	1k Hz -Q- P2- Cz	1k Hz -Q- N2 -Cz	1k Hz - W- P1- Cz	1k Hz - W- N1 -Cz	1k Hz - W- P2- Cz	1k Hz - W- N2 -Cz	1k Hz -B- P1- Cz	1k Hz -B- N1 -Cz	1k Hz -B- P2- Cz	1k Hz -B- N2 -Cz
Valid	15	15	15	15	15	15	15	15	15	15	15	15

B1. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response amplitudes.

	1kHz-Q-P1-Cz	1kHz-Q-N1-Cz	1kHz-Q-P2-Cz	1kHz-Q-N2-Cz	1kHz-W-P1-Cz	1kHz-W-N1-Cz	1kHz-W-P2-Cz	1kHz-W-N2-Cz	1kHz-B-P1-Cz	1kHz-B-N1-Cz	1kHz-B-P2-Cz	1kHz-B-N2-Cz
Missing	0	0	0	0	0	0	0	0	0	0	0	0
Mean	0.699	-0.320	1.028	-2.912	0.370	0.343	1.096	-1.656	0.102	-0.014	0.081	-1.376
Std. Deviation	0.712	0.974	1.338	1.418	0.668	0.683	0.907	1.106	0.470	0.615	0.622	0.980
Shapiro-Wilk	0.935	0.925	0.925	0.976	0.917	0.965	0.976	0.933	0.967	0.954	0.941	0.850
P-value of Shapiro-Wilk	0.326	0.229	0.233	0.932	0.170	0.780	0.938	0.299	0.804	0.584	0.392	0.617
Minimum	-0.266	-1.763	-0.996	-5.840	-0.747	-0.852	-0.362	-3.605	-0.944	-1.016	-0.839	-3.829
Maximum	2.072	1.275	3.353	-0.450	1.376	1.519	3.020	0.294	0.893	0.964	1.033	-0.313

B1. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response amplitudes.

	da-B-P1-Cz	da-B-N1-Cz	da-B-P2-Cz	da-B-N2-Cz	da-Q-P1-Cz	da-Q-N1-Cz	da-Q-P2-Cz	da-Q-N2-Cz	da-W-P1-Cz	da-W-N1-Cz	da-W-P2-Cz	da-W-N2-Cz
Valid	15	15	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0	0	0
Mean	0.591	-1.346	-0.056	-0.287	1.142	0.068	1.137	-2.341	0.295	-0.033	0.885	-1.929
Std. Deviation	0.879	0.940	1.232	1.361	0.845	1.215	1.233	1.323	0.809	1.042	1.170	1.208
Shapiro-Wilk	0.966	0.962	0.944	0.937	0.975	0.913	0.928	0.945	0.914	0.951	0.965	0.957
P-value of Shapiro-Wilk	0.801	0.726	0.429	0.343	0.920	0.152	0.251	0.443	0.154	0.543	0.773	0.634
Minimum	-1.109	-3.378	-2.541	-2.443	-0.631	-1.940	-0.498	-5.015	-0.705	-1.568	-1.311	-3.781
Maximum	1.977	-0.035	1.855	1.664	2.461	1.912	3.320	-0.566	2.079	1.700	3.134	0.091

B2. Descriptive statistics and Shapiro–Wilk normality test results for temporal response amplitudes.

	1k Hz -Q-Ta-T7	1k Hz -Q-Tb-T7	1k Hz -Q-Ta-T8	1k Hz -Q-Tb-T8	1k Hz -B-Ta-T7	1k Hz -B-Tb-T7	1k Hz -B-Ta-T8	1k Hz -B-Tb-T8	1k Hz - W-Ta-T7	1k Hz - W-Tb-T7	1k Hz - W-Ta-T8	1k Hz - W-Tb-T8
Valid	15	15	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0	0	0
Mean	0.377	-0.956	0.774	-0.808	-0.233	-0.516	0.110	-0.102	-0.536	-1.267	0.216	-1.176
Std. Deviation	1.334	1.224	1.046	1.578	0.683	0.718	0.776	0.676	1.114	0.999	0.971	1.370
Shapiro-Wilk	0.957	0.930	0.969	0.797	0.958	0.934	0.956	0.913	0.966	0.966	0.949	0.938
P-value of Shapiro-Wilk	0.644	0.272	0.846	0.303	0.662	0.312	0.619	0.150	0.791	0.798	0.502	0.357
Minimum	-1.446	-2.441	-1.361	-5.347	-1.678	-2.327	-1.248	-1.000	-2.153	-2.854	-1.778	-3.974
Maximum	3.323	1.245	2.309	0.859	0.726	0.596	1.261	0.938	1.559	0.657	1.758	0.956

B2. Descriptive statistics and Shapiro–Wilk normality test results for temporal response amplitudes.

	da-Q-Ta-T7	da-Q-Ta-T8	da-Q-Tb-T7	da-Q-Tb-T8	da-W-Ta-T7	da-W-Ta-T8	da-W-Tb-T7	da-W-Tb-T8	da-B-Ta-T7	da-B-Ta-T8
Valid	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0
Mean	0.588	1.658	-0.119	0.311	0.268	1.300	-0.326	-0.249	1.028	1.555
Std. Deviation	1.640	1.688	1.521	1.144	1.571	1.821	1.502	1.688	0.787	0.939
Shapiro-Wilk	0.975	0.964	0.977	0.959	0.966	0.882	0.964	0.936	0.947	0.945
P-value of Shapiro-Wilk	0.920	0.763	0.941	0.671	0.792	0.550	0.762	0.337	0.479	0.448
Minimum	-2.611	-1.035	-3.598	-2.232	-2.316	-3.072	-2.726	-2.972	-0.179	-0.096
Maximum	3.212	4.673	2.473	2.121	2.944	4.116	2.847	2.219	2.305	3.005

B3. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response latencies.

	P1-AM-Q-1kHz-Cz	N1-AM-Q-1kHz-Cz	P2-AM-Q-1kHz-Cz	N2-AM-Q-1kHz-Cz	P1-AM-W-1kHz-Cz	N1-AM-W-1kHz-Cz	P2-AM-W-1kHz-Cz	N2-CzAM-W-1kHz-Cz	P1-AM-B-1kHz-Cz	N1-AM-B-1kHz-Cz	P2-AM-B-1kHz-Cz	N2-AM-B-1kHz-Cz
Valid	15	15	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0	0	0
Mean	88.400	127.733	181.333	276.000	99.733	143.467	197.467	293.333	98.400	141.067	194.933	281.467
Std. Deviation	7.018	7.166	13.238	14.658	17.318	20.639	16.326	16.830	17.956	20.197	29.431	19.574
Shapiro-Wilk	0.949	0.938	0.875	0.950	0.939	0.867	0.858	0.879	0.883	0.884	0.898	0.918
P-value of Shapiro-Wilk	0.509	0.361	0.340	0.524	0.375	0.131	0.122	0.146	0.153	0.255	0.288	0.182

B3. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response latencies.

	P1-AM-Q-1kHz-Cz	N1-AM-Q-1kHz-Cz	P2-AM-Q-1kHz-Cz	N2-AM-Q-1kHz-Cz	P1-AM-W-1kHz-Cz	N1-AM-W-1kHz-Cz	P2-AM-W-1kHz-Cz	N2-CzAM-W-1kHz-Cz	P1-AM-B-1kHz-Cz	N1-AM-B-1kHz-Cz	P2-AM-B-1kHz-Cz	N2-AM-B-1kHz-Cz
Minimum	78.000	116.000	162.000	254.000	66.000	118.000	178.000	272.000	78.000	110.000	152.000	258.000
Maximum	100.000	142.000	208.000	298.000	130.000	202.000	234.000	316.000	134.000	182.000	256.000	316.000

B3. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response latencies.

	P1-AM-Q-da-Cz	N1-AM-Q-da-Cz	P2-AM-Q-da-Cz	N2-AM-Q-da-Cz	P1-AM-W-da-Cz	N1-AM-W-da-Cz	P2-AM-W-da-Cz	N2-CzAM-W-da-Cz	P1-AM-B-da-Cz	N1-AM-B-da-Cz	P2-AM-B-da-Cz	N2-AM-B-da-Cz
Valid	15	15	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0	0	0

B3. Descriptive statistics and Shapiro–Wilk normality test results for frontocentral response latencies.

	P1-AM-Q-da-Cz	N1-AM-Q-da-Cz	P2-AM-Q-da-Cz	N2-AM-Q-da-Cz	P1-AM-W-da-Cz	N1-AM-W-da-Cz	P2-AM-W-da-Cz	N2-CzAM-W-da-Cz	P1-AM-B-da-Cz	N1-AM-B-da-Cz	P2-AM-B-da-Cz	N2-AM-B-da-Cz
Mean	89.733	126.394	192.800	289.800	116.667	164.133	218.667	323.200	138.133	250.667	365.467	451.800
Std. Deviation	10.138	36.308	21.697	18.104	14.787	23.742	18.741	25.023	19.467	16.779	23.021	41.373
Shapiro-Wilk	0.940	0.589	0.919	0.937	0.873	0.856	0.946	0.883	0.897	0.748	0.923	0.930
P-value of Shapiro-Wilk	0.379	0.201	0.183	0.351	0.137	0.221	0.466	0.152	0.187	0.201	0.211	0.270
Minimum	74.000	1.910	166.000	262.000	102.000	138.000	190.000	278.000	110.000	198.000	328.000	370.000
Maximum	108.000	156.000	246.000	318.000	148.000	226.000	254.000	384.000	170.000	268.000	396.000	514.000

B4. Descriptive statistics and Shapiro–Wilk normality test results for temporal response latencies.

	Ta 7-AM-Q-1k Hz-T7	Ta 8-AM-Q-1k Hz-T8	Tb 7-AM-Q-1k Hz-T7	Tb 8-AM-Q-1k Hz-T8	Ta 7-AM-W-1k Hz-T7	Ta 8-AM-W-1k Hz-T8	Tb 7-AM-W-1k Hz-T7	Tb 8-AM-W-1k Hz-T8	Ta 7-AM-B-1k Hz-T7	Tb 7-AM-B-1k Hz-T7	Ta 8-AM-B-1k Hz-T8	Tb 8-AM-B-1k Hz-T8
Valid	15	15	15	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0	0	0	0
Mean	132.133	129.600	174.400	174.000	136.667	140.200	185.267	189.400	138.467	175.267	137.667	181.267
Std. Deviation	8.927	5.914	11.141	8.418	12.737	10.449	10.382	11.375	11.874	10.532	16.808	16.981
Shapiro-Wilk	0.948	0.939	0.962	0.896	0.947	0.959	0.916	0.960	0.918	0.913	0.553	0.841
P-value of Shapiro-	0.500	0.365	0.728	0.082	0.483	0.680	0.170	0.699	0.179	0.150	0.101	0.113

B4. Descriptive statistics and Shapiro–Wilk normality test results for temporal response latencies.

	Ta7-AM-Q-1kHz-T7	Ta8-AM-Q-1kHz-T8	Tb7-AM-Q-1kHz-T7	Tb8-AM-Q-1kHz-T8	Ta7-AM-W-1kHz-T7	Ta8-AM-W-1kHz-T8	Tb7-AM-W-1kHz-T7	Tb8-AM-W-1kHz-T8	Ta7-AM-B-1kHz-T7	Tb7-AM-B-1kHz-T8	Ta8-AM-B-1kHz-T8	Tb8-AM-B-1kHz-T8
Wilk												
Minimum	120.000	120.000	150.000	162.000	118.000	122.000	166.000	169.000	124.000	158.000	124.000	162.000
Maximum	150.000	142.000	192.000	192.000	167.000	156.000	198.000	206.000	164.000	192.000	196.000	220.000

B4. Descriptive statistics and Shapiro–Wilk normality test results for temporal response latencies.

	Ta7-AM-Q-da-T7	Tb7-AM-Q-da-T7	Ta8-AM-Q-da-T8	Tb8-AM-Q-da-T8	Ta7-AM-W-da-T7	Ta8-AM-W-da-T8	Tb8-AM-W-da-T8	Ta7-AM-B-da-T7	Ta8-AM-B-da-T8
Valid	15	15	15	15	15	15	15	15	15
Missing	0	0	0	0	0	0	0	0	0
Mean	137.867	194.267	138.933	194.267	166.933	162.800	219.333	257.867	247.533

B4. Descriptive statistics and Shapiro–Wilk normality test results for temporal response latencies.

	Ta7-AM-Q-da-T7	Tb7-AM-Q-da-T7	Ta8-AM-Q-da-T8	Tb8-AM-Q-da-T8	Ta7-AM-W-da-T7	Ta8-AM-W-da-T8	Tb8-AM-W-da-T8	Ta7-AM-B-da-T7	Ta8-AM-B-da-T8
Std. Deviation	15.519	16.884	7.959	18.805	21.232	10.712	11.075	22.469	45.158
Shapiro-Wilk	0.945	0.984	0.975	0.948	0.926	0.968	0.978	0.881	0.923
P-value of Shapiro-Wilk	0.448	0.990	0.921	0.487	0.237	0.835	0.951	0.149	0.213
Minimum	100.000	166.000	126.000	168.000	138.000	142.000	200.000	196.000	133.000
Maximum	160.000	226.000	154.000	236.000	208.000	184.000	240.000	300.000	334.000

The majority of the variables met the assumption of normality according to the Shapiro–Wilk test (Tables B1–B4).