

**Investigating Temporal Resolution in Children with Listening Difficulties
Using a Novel Multi-Deviant Paradigm**

Shaghayegh Omidvar

Thesis submitted to the University of Ottawa
in partial Fulfillment of the requirements for the degree of
Doctor of Philosophy in Rehabilitation Sciences

School of Rehabilitation Science
Faculty of Health Sciences
University of Ottawa

© Shaghayegh Omidvar, Ottawa, Canada, 2026

Abstract

Auditory temporal resolution is the ability to detect rapid changes in sound. It is often measured using gap-detection tasks, in which a listener detects a brief silent interval within a continuous or long-duration sound. Gap detection is important for speech perception because many speech cues depend on fine temporal information. However, behavioural gap-detection tasks require attention, motivation, and active responses. These factors are especially important when testing children, including those with auditory processing disorder (APD), because poor behavioural performance may not always reflect a pure auditory temporal-processing deficit.

The present thesis examined automatic auditory gap detection using event-related potentials (ERPs). The main focus was the deviant-related negativity (DRN), an electrophysiological response elicited by a gap deviant. The thesis investigated whether the temporal position of a gap affects the DRN and whether a time-efficient multi-deviant paradigm can be used to study multiple gap onset times within a single recording sequence. Behavioural and electrophysiological gap detection were examined in adults, typically developing (TD) children, and children with APD.

Participants completed passive electroencephalography (EEG) recordings while watching a silent subtitled video and ignoring the auditory stimuli. The standard stimulus was a 500-ms broadband-noise burst. Deviants were created by inserting a silent gap of either 10 or 30 ms at different onset times, ranging from 15 to 400 ms after stimulus onset. In a separate behavioural task, participants actively detected the occurrence of the gap.

There was no significant difference in DRN amplitude between the multi- and single-deviant paradigms. Across groups, DRN amplitude was larger for 30-ms than for 10-ms gaps and decreased when the gap occurred later in the stimulus. In adults, the DRN was reduced for gaps occurring at 300 and 400 ms. In TD children, this temporal limitation was stronger, with reliable DRNs mainly observed for early-occurring gaps. Behavioural performance was generally high, but children performed more poorly than adults. Children with APD also showed poorer behavioural gap detection than TD children. However, their overall DRN amplitude was not consistently reduced, although less stable responses were observed in some conditions.

Overall, this thesis showed that automatic neural detection of gaps is affected by both gap duration and gap onset time. The findings support the idea that auditory deviance detection is

limited by a short temporal integration window. This limitation appears stronger in children than in adults. In children with APD, poorer behavioural performance may partly reflect task demands or attention rather than a consistent deficit in automatic auditory change detection. The multi-deviant gap paradigm may therefore be considered in future work as a time-efficient approach for studying auditory temporal processing in adults, children, and clinical populations.

Keywords: auditory temporal resolution, gap detection, mismatch negativity, deviant-related negativity, temporal integration, auditory processing disorder, children, event-related potentials.

Preface

This thesis is the original work of Shaghayegh Omidvar, the doctoral candidate, and was conducted under the supervision of Dr. Amineh Koravand. The research was completed with the guidance and support of the supervisory committee and collaborators. The candidate was involved in the study design, data collection, data analysis, interpretation of the findings, and manuscript preparation. Contributions from co-authors and collaborators are acknowledged in the relevant chapters and manuscripts.

This research project was conducted with the approval of the University of Ottawa Research Ethics Board (file number # H-07-24-10412). The studies presented in this thesis involved adult participants, typically developing children, and children with auditory processing disorder. All participants and the parents or guardians of child participants provided consent or assent in accordance with the approved ethics protocols. Chapters 3, 4, and 5 are presented as manuscripts and are formatted according to the submission or publication requirements of the journals to which they have been submitted or are intended to be submitted.

Acknowledgements

This thesis was not completed alone. It was shaped by the support, patience, and guidance of many people who helped me throughout this long journey. I am deeply grateful to everyone who contributed to this work, both directly and indirectly.

First, I would like to thank my supervisor, Dr. Amineh Koravand, for her continuous support, guidance, and encouragement throughout my PhD. Thank you for believing in this work and for helping me grow as a researcher. Your advice and feedback helped me improve the quality of this thesis and gave me strength during the difficult stages of the process. I am deeply grateful for your patience, kindness, and trust in me throughout this journey. Your support helped me continue even during the moments when this path felt overwhelming.

I would like to express my deepest gratitude to Dr. Kenneth Campbell. Ken, your thoughtful ideas, careful guidance, and constant support shaped this thesis in many important ways. You helped me more than I can fully express. Your knowledge, your attention to detail, and your ability to see the meaning behind the data helped me understand my work more deeply. I am truly grateful for the time, patience, and care you gave to this research. This thesis would not have been the same without your support.

I would also like to thank Dr. Claude Alain and Dr. Steven Aiken for their valuable contributions, thoughtful comments, and support. Your expertise helped improve the manuscripts and strengthen the interpretation of the findings. I am grateful for the time you devoted to reading this work and for the constructive suggestions you provided throughout this process. Your guidance helped me view this research from different perspectives and improved the final quality of this thesis.

I am also grateful to Dr. Caryn Bursch for her help in recruiting participants for the APD study. Your support was very important in completing the APD study. I would like to thank Félix Harvey, Olivia Chanel Zupicic-Grsic, William Khawam, Olivia, and Felix for their assistance with data collection. I am also deeply thankful to all children, parents, and adult participants who took part in this research. This thesis would not have been possible without their time, patience, and willingness to participate.

To my husband, Hamid, thank you for being beside me through every step of this journey. You saw the difficult days, the long nights, the stress, and the moments when I felt I could not continue. You carried so much with patience and love, often quietly, so that I could keep going. Thank you for listening to me, encouraging me, calming me, and believing in me even when I doubted myself. Your support gave me strength in the moments when I needed it most. This thesis carries not only my work, but also your patience, sacrifices, and love. I could not have reached this point without you.

To my son, Ryan, you are the light of my life. Your smile, your voice, and your love gave meaning to this journey in a way nothing else could. There were many moments when I was tired, overwhelmed, or uncertain, but seeing you reminded me why I needed to keep going. You gave me hope without even knowing it. You taught me to be stronger, more patient, and more grateful. I hope one day, when you read these words, you know that you were with me in every step of this thesis. You were my biggest motivation and my sweetest reason to continue.

To my mother and father, thank you for your unconditional love, sacrifices, and endless support. You believed in me from the beginning and encouraged me through every stage of my life. Your love gave me the foundation to stand on, and your prayers and support helped me continue when the path was difficult. Everything I have achieved is rooted in your love, patience, and trust in me.

Finally, I would like to thank my friends and colleagues who supported me in different ways during this process. Your encouragement, kindness, and understanding meant a lot to me. This thesis represents not only years of work, but also the support of the people who helped me reach this point.

Table of Contents

Abstract	ii
Preface.....	iv
Acknowledgements.....	v
Table of Contents.....	vii
List of Tables	xii
List of Figures	xiii
List of Abbreviations and Acronyms.....	xvi
Chapter 1: Introduction	1
1.1 Introduction.....	1
1.2 Objective of the thesis.....	10
1.3 Overview of the thesis	11
Chapter 2: Literature Review.....	13
2.1 Auditory processing disorder (APD)	13
2.2 Auditory Temporal Processing	15
2.3 Temporal resolution	15
2.3.1 Behavioural assessment of temporal resolution in typically developing children..	17
2.3.2 Behavioural assessment of temporal resolution in children with APD	19
2.3.3 The effect of gap position on the behavioural gap detection threshold	21
2.4 Event-related potentials and mismatch negativity	23
2.4.1 Effect of gap duration on automatic deviance detection.....	25
2.4.2 Temporal distance effect in automatic deviance detection	27
2.4.3 Development of MMN to gap-inserted stimuli in typically developing children...	33
2.4.4 MMN responses in children with APD: evidence from gap and speech paradigms	

2.5 Summary and Research Gap	35
Chapter 3: The effect of the temporal position of a gap on its automatic detection	37
Abstract	38
3.1 Introduction.....	39
3.2 Methods.....	45
3.2.1 Participants.....	45
3.2.2 Procedure and stimuli	45
3.2.3 Active behavioural detection task.....	47
3.2.4 Electrophysiological recording and analyses.....	47
3.2.5 Quantification and statistical analyses	48
3.3 Results.....	50
3.3.1 Electrophysiological data.....	50
3.3.2 Behavioural data	54
3.4 Discussion	56
3.5 Limitations	60
3.6 Conclusions.....	60
Declaration of conflict of interest	61
Acknowledgements.....	61
References.....	61
Chapter 4: Effects of gap onset time and gap duration on the deviant-related negativity in typically developing school-age children	67
Abstract	68
4.1 Introduction.....	69
4.2 Methods.....	75
4.2.1 Participants.....	75

4.2.2	Procedure and stimuli	75
4.2.3	Electrophysiological recording and analyses.....	76
4.2.4	Behavioural detection	77
4.2.5	ERP Quantification and Analysis	78
4.3	Results.....	80
4.3.1	Electrophysiological data.....	80
4.3.2	Behavioural data	86
4.4	Discussion	87
4.5	Conclusion	92
	Acknowledgements.....	92
	References.....	93
Chapter 5: Neural Correlates of Auditory Temporal Resolution in Children with Auditory Processing Disorder Using a Multi-Deviant Gap Detection Paradigm: A Pilot Study.....		
	Abstract.....	102
5.1	Introduction.....	103
5.2	Methods.....	110
5.2.1	Participants.....	110
5.2.2	Procedure and stimuli	113
5.2.3	Electrophysiological recording and analyses.....	114
5.2.4	Behavioural detection	115
5.2.5	ERP Quantification and Analysis	115
5.3	Results.....	119
5.3.1	Behavioural data	119
5.3.2	Electrophysiological data.....	120
5.3.3	Power analysis	123

5.4 Discussion	124
5.5 Conclusions.....	128
Conflict of interest	129
Acknowledgements.....	129
References.....	130
Chapter 6: General discussion	137
6.1 Overview of the main findings	137
6.2 Behavioural measures	137
6.2.1 Effect of gap duration	138
6.2.2 Effect of gap onset time	139
6.3 Electrophysiological measures.....	140
6.3.1 Effect of gap duration	140
6.3.2 Effect of gap onset time	141
6.4 Comparing electrophysiological with behavioural measures	143
6.5 Limitations	145
6.6 Future directions	149
6.7 General Conclusions	150
6.7.1 Novel findings of the thesis	151
References (excluding articles).....	152
Appendices.....	168
Appendix 1. Ethics approval.....	168
Appendix 2. Consent form: Adults.	170
Appendix 2. Consent form: Typically Developing Children.....	175
Appendix 3. Consent form: Children with Auditory Processing Disorders.	180
Appendix 4. Recruitment Poster for Adults.....	185

Appendix 5. Recruitment Poster for Typically Developing Children.	186
Appendix 6. Compensation form.	187
Appendix 7. Demographic and Music Experience Questionnaire.....	188
Appendix 8. Demographic and Music Experience Questionnaire: Child’s version.	194
Appendix 9. Auditory Processing Questionnaire (APDQ)	199

List of Tables

Table 5.1 Results of participants with Auditory Processing Disorder (APD) on auditory-processing and attention screening tests.	112
Table 5.2 Latency windows used to compute DRN amplitude for each condition in children with APD (n = 6) and TD children (n = 6).	117
Table 5.3 Total and per-group estimated sample sizes for each of the 12 conditions to detect a statistically significant difference (power = 0.80, α = 0.05) based on the means and standard deviations for each group.	124

List of Figures

Figure 3.1 Multi-deviant and single-deviant sequences. A: In the multi-deviant sequence, the standard and deviant stimuli alternated. Three gap onset times are shown. In the single-deviant sequence, a gap was presented at either 100 or 300 ms, depending on the condition. B: Timing of stimulus presentation. For both the multi- and single-deviant sequences, the duration of all stimuli was 500 ms. The ISI was 400 ms.....	46
Figure 3.2. The grand average ERP waveforms following the presentation of the standard stimulus. A. The standard ERP is illustrated for the 10- and 30-ms gap duration conditions. The amplitude of N1 was very small because of the rapid rate of stimulus presentation. It was followed by a positivity, P2. Their amplitudes did not significantly differ between the 10- and 30-ms duration gap conditions. B. The standard ERP in the single- and multi-deviant conditions. Again, the amplitudes of N1 and P2 following the presentation of the standard did not differ significantly between the two paradigms.....	50
Figure 3.3 Grand averaged ERP deviant-standard difference waves in the multi-deviant condition as a function of gap duration and gap onset time. The onset of the deviant stimulus containing the gap is indicated by the solid vertical black line at 0 ms. The onset of the actual gap is indicated by the red dashed line. A DRN is apparent approximately 100–150 ms after gap onset. The DRN was significantly reduced when the gap duration was 10 ms. The DRN was much reduced when the gap occurred at 300 or 400 ms.....	51
Figure 3.4 Mean amplitude of the DRN for the 10- and 30-ms duration gaps as a function of gap onset time. Error bars represent the standard error (SE) of the mean.	52
Figure 3.5 The effect of single- and multi-deviant paradigms. The 10-ms duration gap deviant was presented in both paradigms, occurring at 100 or 300 ms after stimulus onset. The amplitude of the DRN was significantly reduced in both paradigms when the gap occurred at 300 ms. The amplitude of the DRN was also reduced in the multi-deviant paradigm, but the difference was not significant. Note, however, that random differences were apparent before the onset of the gap. The DRN was therefore also measured peak-to-peak with respect to the subsequent positivity.	54
Figure 3.6 Behavioural performance data. Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time. Error bars represent SE.	55
Figure 4.1 “Raw” standard (left column), deviant (middle column), and the deviant-standard difference wave (right column) in children. The standard and deviant were physically identical	

until the time of onset of the gap (indicated by the red vertical lines). In the left portion of the Figure, the ERPs to the standard stimulus are illustrated. The onset of the standard elicited a large positivity in the children. The middle portion illustrates the ERPs elicited by the different deviant stimuli. The deviant onset also elicited a large positivity. The right portion of the Figure illustrates the deviant-standard difference wave. Processing common to the standard and deviant should thus have been removed in the subtraction process. Thus, the large positive wave common to both the standard and deviant is removed in the difference wave, leaving processing unique to the gap deviant. The overall morphology of the difference wave was relatively similar in children and adults. 79

Figure 4.2 Standard ERP waveforms for the 10-ms and 30-ms gap duration conditions in children and adults. The amplitudes of the negative and positive deflections did not differ significantly within each condition. 81

Figure 4.3 Deviant–standard difference waves for children and adults at Fz and Cz, as a function of gap duration and gap onset time. The red dashed line indicates the time of onset of the gap. For children, a clear DRN is visible around 100–150 ms following gaps occurring 15 to 100 ms after the onset of the deviant, although CI testing confirmed a significant DRN only for the 30-ms gaps occurring at 15 and 50 ms. In adults, a clear DRN is visible for gaps occurring between 15 and 300 ms after the onset of the deviant. A subsequent positivity, P2, was larger in children than in adults. The DRN was significantly reduced when the gap duration was 10 ms. 82

Figure 4.4 Mean baseline-to-peak DRN amplitude in children and adults for the 10- and 30-ms duration gaps as a function of gap onset time at the frontal and central ROIs. Error bars represent the standard error (SE) of the mean. 83

Figure 4.5 Mean peak-to-peak DRN amplitude in children and adults for the 10- and 30-ms duration gaps as a function of gap onset time at the frontal and central ROIs. Error bars represent the standard error (SE) of the mean. 86

Figure 4.6 Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time in children and adults. Error bars represent the SE of the mean. As may be observed, d' was quite high overall regardless of gap duration and the onset time of the gap. Performance was nevertheless significantly poorer in children. 87

Figure 5.1 “Raw” standard (left column), deviant (middle column), and the deviant-standard difference wave (right column) in TD children (A), and children with APD (B) for the gap onset

times of 15, 100, and 300 ms at Fz. The standard and deviant were physically identical until the time of onset of the gap (indicated by the red vertical lines). In the left portion of the Figure, the ERPs to the standard stimulus are illustrated. The onset of the standard elicited a large positivity. The middle portion illustrates the ERPs elicited by the different deviant stimuli. The onset of the deviant also elicited a large positivity. The right portion of the Figure illustrates the deviant-standard difference wave. Processing common to the standard and deviant should thus have been removed during the subtraction process. Thus, the large positive wave common to both the standard and deviant is removed in the difference wave, leaving processing unique to the gap deviant..... 116

Figure 5.2 Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time in TD children and children with APD. Error bars represent the 95% SE of the mean. As may be observed, d' was quite high overall, regardless of gap duration and the onset time of the gap. Performance, however, was significantly poorer in children with APD..... 119

Figure 5.3 Standard ERP waveforms for the 10-ms and 30-ms gap duration conditions in TD and APD children at Fz. The amplitudes of the negative and positive deflections did not differ significantly within each condition..... 120

Figure 5.4 Deviant–standard ERPs in the multi-deviant condition for APD and TD children at Fz as a function of gap duration and gap onset time. The red dashed line indicates the onset of the gap. A clear DRN (green shaded regions) is visible around 100–150 ms following gaps occurring 15 to 100 ms after the onset of the deviant. For TD children, a clear DRN is visible for gaps occurring from 15 to 100 ms after the onset of the deviant. The DRN was not as clear as in TD children but appeared to be present at 15- and 50-ms onset times for the 30-ms gap duration.. 121

Figure 5.5 Mean peak-to-peak DRN amplitude in TD and APD children for the 10- and 30-ms duration gaps as a function of gap onset time at Fz. Error bars represent the standard error (SE) of the mean. 122

List of Abbreviations and Acronyms

ACPT	Auditory Continuous Performance Test
ANSI	American National Standards Institute
ANOVA	Analysis of Variance
APD	Auditory Processing Disorder
APDQ	Auditory Processing Domains Questionnaire
ASHA	American Speech-Language-Hearing Association
ATTR	Adaptive Test of Temporal Resolution
BC	Between-channel
CI	Confidence interval
DRN	Deviant-related negativity
EEG	Electroencephalography
EOG	Electrooculography
ERP	Event-related potential
GDT	Gap detection threshold
GIN	Gap-in-noise
ICA	Independent component analysis
ISI	Inter-stimulus interval
LiD	Listening difficulties
MMN	Mismatch negativity
MMR	Mismatch response
NH	Normal hearing
ROI	Region of interest
RGDT	Random Gap Detection Test

SD	Standard deviation
SE	Standard error
TD	Typically developing
TWI	Temporal window of integration
VOT	Voice onset time
WC	Within-channel

Chapter 1: Introduction

1.1 Introduction

Listening difficulties (LiDs) encompass challenges in auditory comprehension, particularly in noisy environments. These difficulties are not exclusive to individuals with hearing impairments but also affect those with cognitive deficits, language impairments, and auditory processing disorders (Dillon & Cameron, 2021). Auditory processing disorder (APD) is an umbrella term describing specific deficits that result in poorer-than-normal perception of auditory stimuli (Dillon & Cameron, 2021). Reported prevalence estimates for APD in school-age children vary considerably because of differences in diagnostic criteria, test batteries, and sampling methods; for example, Nagao et al. (2016) estimated a prevalence of 2.08 per 1,000 children (0.2%) in a Mid-Atlantic school-based cohort, whereas Shreyas and Jain (2024) reported a two-year clinical prevalence of 0.7% among children aged 6–18 years seen in an audiology outpatient service (Nagao et al., 2016; Shreyas & Jain, 2024). Despite this variability, APD remains clinically important because auditory processing deficits can interfere with speech understanding, especially in noise, and may contribute to broader listening, communication, and academic difficulties in children (Alanazi, 2023; American Speech-Language-Hearing Association [ASHA], n.d.; Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology, 2012; Dillon & Cameron, 2021).

The underlying mechanisms of APD remain debated. One hypothesis posits that APD reflects a bottom-up deficit in neural processing within the central auditory nervous system, despite normal peripheral hearing sensitivity (ASHA, 2005). In this view, APD is thought to result from impaired transmission of auditory information from lower to higher levels of the auditory system. A second hypothesis emphasizes that disruptions in top-down, concept-driven processes play an important role in APDs. This perspective holds that APD primarily arises from impairments in higher-order cognitive processes, such as attention, working memory, and central executive control. Such processes involve multimodal networks in frontal, parietal, and anterior temporal regions (Dhamani et al., 2013; Ferguson et al., 2011; Moore et al., 2010; Moore & Hunter, 2013). Reflecting both views, the British Society of Audiology (Moore et al., 2018) defines APD as

involving deficits in bottom-up auditory processing and top-down mechanisms, particularly auditory attention.

Children with APD often have difficulty understanding speech in background noise, following oral instructions, and processing rapid or complex speech, which can negatively affect listening, learning, and academic performance (ASHA, n.d.; Alanazi, 2023; Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology, 2012). Such everyday difficulties have directed attention toward possible deficits in central auditory processes. One process of particular interest is temporal processing, which refers to the analysis of timing information in sound (ASHA, n.d.). Among the different aspects of temporal processing, temporal resolution refers to the auditory system's ability to detect rapid temporal changes in acoustic signals, including brief silent intervals or gaps within or between sounds (Lister et al., 2006; Musiek et al., 2005). Temporal resolution offers valuable insights into an individual's capacity to distinguish rapidly changing sounds, such as speech sounds (Iliadou et al., 2014). The ability to perceive and react to changes in speech sounds is a fundamental aspect of language learning; therefore, temporal resolution plays a significant role in phonological representation and the perception of fine temporal cues in speech (Cabrera & Lau, 2022; Drosos et al., 2024; Hautus et al., 2003; Phillips, 1999; Tallal et al., 1993; Walker et al., 2002).

One commonly used method for evaluating auditory temporal resolution involves gap detection (Keith, 2000; Musiek et al., 2005). A silent interval (the “gap”) is inserted either into a continuous (Musiek et al., 2005) or a brief-duration sound (Keith, 2000). The shortest detectable gap duration defines the gap detection threshold (GDT). The ability to detect such gaps is critical for speech comprehension because the continuous flow of normal speech is interrupted by moments of silence or reduced sound intensity. Several studies have shown higher GDTs in children with APDs compared to their typically developing (TD) peers (Cameron et al., 2005; Dias et al., 2012; Jain et al., 2025; McWeeny & Norton, 2023; Omidvar et al., 2023; Phillips et al., 2010), although this difference diminishes with increasing age (Dias et al., 2012). For example, Dias et al. (2012) used the Random Gap Detection Test (RGDT) in a large cohort of participants aged 5 to 25 years, with and without CAPD, and found poorer performance in the CAPD group, especially in younger children. They argued that age-related improvement likely reflects auditory maturation and noted that behavioural gap-detection results in young children may also be influenced by non-auditory factors. However, these findings are not consistent across all gap-

detection paradigms. Phillips et al. (2010) found no significant group difference for within-channel gap detection, although children with APD showed poorer performance in the between-channel condition. Similarly, Jain et al. (2025) found no significant differences between children with APD and TD children for broadband-noise or within-channel gap-detection measures, with a significant group difference observed only in the across-channel condition. These findings suggest that poorer gap-detection performance is observed in many children with APD, but the presence and magnitude of group differences may depend on the specific task and stimulus conditions. Gap detection is closely related to speech perception (Buss et al., 2017; Gay et al., 2020). For example, the silent interval between the release of a stop consonant and the onset of voicing of the following speech sound is known as voice onset time (VOT). Adequate gap-detection ability can support the discrimination of phonemes with different VOTs, such as /ba/ and /pa/ (Elangovan & Stuart, 2008). However, while these speech-related silent intervals occur over very short time scales, the placement of gaps in clinical behavioural gap-detection tests is often much longer (Phillips, 1998). In the Behavioural Gap in Noise (GIN) test, for example, the time between stimulus onset and gap onset is at least 500 ms (Musiek et al., 2005), while in the RGDT the silent interval consistently occurs in the middle of stimulus pairs (Keith, 2000). Detecting a gap may be even more difficult when the gap occurs close to stimulus onset or offset rather than near the middle of the sound (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999).

Another important limitation of behavioural gap-detection tests is that they require active participation. The child must maintain attention throughout a relatively long testing session and make repeated perceptual decisions. Performance can therefore be influenced not only by auditory sensitivity, but also by non-sensory factors such as motivation, attention, and memory for task procedures. This is particularly important in normal-hearing children and particularly in children with APD, who may also experience attentional difficulties (Dillon & Cameron, 2021). As a result, an elevated GDT may reflect a true bottom-up temporal processing disorder, but it may also reflect a top-down listening problem, such as difficulty sustaining attention during the task.

Scalp recordings of event-related potentials (ERPs) can provide a non-invasive neurophysiological method for passively measuring temporal resolution, without requiring participants to actively engage in the gap detection task. An ERP component labelled as the mismatch negativity (MMN) is often used in gap-detection studies (Bertoli et al., 2001; Bertoli et al., 2002; Desjardins et al., 1999; Duda-Milloy et al., 2019; Duda et al., 2025; Duda et al., 2020;

Heinrich et al., 2004; Tamakoshi et al., 2016; Todd et al., 2011; Uther et al., 2003; Yabe et al., 2005). The MMN is usually studied using a so-called oddball paradigm. Participants are presented with a frequently occurring "standard" stimulus. At rare (or "odd") and unpredictable times, a feature of the standard stimulus is changed to form a "deviant." All auditory stimuli, including both standards and deviants, elicit a series of obligatory positive ("P") and negative ("N")-going ERPs, typically labelled as P1, N1, P2, and N2. In adults, P1 peaks at about 50 ms, followed by N1 at around 80-100 ms, and subsequently P2 at approximately 180-250ms after stimulus onset, and a subsequent N2 response between 200 and 300 ms (Sabri et al., 2000). In addition to these obligatory responses, the deviant stimulus elicits the MMN, a negative deflection that peaks approximately 100 to 200 ms after the onset of the deviance. It is maximal in amplitude over frontocentral scalp regions and inverts in amplitude (it is seen as a negative deflection) at regions inferior to the auditory cortex (Näätänen et al., 1978).

Auditory ERPs in young children differ from the mature P1–N1–P2–N2 waveform typically observed in adults. In children, the waveform is mainly characterized by the P1 and N2 components. The P1 is particularly prominent and has a longer latency than in adults, usually occurring between 50 and 100 ms. The N2 is the dominant negative component in children, whereas the N1 is the dominant negative peak in adults. The emergence of the N1 in children depends on factors such as the inter-stimulus interval (ISI), and it is generally observed from about 9 years of age when the ISI exceeds 1 s (Čeponien et al., 2002).

In gap-detection studies, a short-duration gap is inserted some time after the onset of a long-duration (200-800 ms) deviant. Thus, the deviant is physically identical to the standard stimulus until the occurrence of the gap. Although many studies classify the negativity elicited by an infrequent gap deviant as an MMN, this response may not be a "pure" MMN (Campbell & Macdonald, 2011; He et al., 2013; Lister & Roberts, 2005; Palmer & Musiek, 2013; Pratt et al., 2007). The gap onset also elicits an N1 component, which is generated by abrupt transients such as stimulus onset (Näätänen & Picton, 1987). Because the onset period of the standard and deviant stimuli is physically identical, they elicit the same N1. The onset of the gap will also elicit an N1. The occurrence of the gap deviant, however, signals an acoustic change from the standard and will thus also elicit an MMN. Therefore, the gap elicits a composite negativity, the N1+MMN (Duda-Milloy et al., 2019). This composite negativity has been labelled as a deviant-related negativity (DRN) in adults (Woldorff et al., 1998).

In adults, studies have shown that a supra-threshold gap elicits a DRN occurring about 100 ms after the gap onset, with its amplitude increasing with gap duration (Alain et al., 2004; Bertoli et al., 2001; Desjardins et al., 1999; Duda-Milloy et al., 2019; Duda et al., 2025; Duda et al., 2020; Tamakoshi et al., 2016; Todd et al., 2011; Yabe et al., 2005). For example, Duda-Milloy et al. (2019) used a 200-ms white-noise burst as the standard stimulus, with gap deviants created by inserting a silent interval in the centre of the standard. Six deviants were presented, with gap durations ranging from 2 to 40 ms. As expected, a DRN was difficult to observe following the presentation of the sub-threshold 2-ms gap. It was, however, apparent for the near-threshold 5-ms gap. The DRN amplitude was larger for the longer-duration gaps. Follow-up studies have replicated these findings (Duda et al., 2020; Duda et al., 2025).

Only a limited number of studies have examined ERPs to gap deviants in infants and children. Most of these studies have used oddball-like paradigms (Putkinen et al., 2014; Trainor et al., 2001; Trainor et al., 2003). For example, Trainor et al. (2003) presented a deviant containing a 16-ms silent gap within a frequently occurring standard that lacked a gap. They showed that infants as young as 4 to 6 months old exhibited MMN-like responses to the silent gaps, suggesting that temporal sensitivity emerges very early in development. Other studies have used multi-feature paradigms (Partanen et al., 2013; Petermann et al., 2009; Rapaport et al., 2023), in which a gap deviant was presented among several other deviants. In these studies, gap deviants reliably elicited an MMN in children. Partanen et al. (2013), for example, examined 30 children aged 4 to 12 years using a gap deviant consisting of a 100-ms silent interval embedded in the middle syllable of a pseudoword (/tatata/). The gap deviant elicited a frontocentral MMN in all children, and MMN amplitude did not differ between preschool and school-aged children. Similarly, Petermann et al. (2009) studied children aged 5 to 7 years using a multi-deviant paradigm that included a 16-ms silent gap inserted into the centre of a 100-ms standard stimulus, and again found an MMN response. In a more recent multi-deviant magnetoencephalography study, Rapaport et al. (2023) examined children aged 3 to 7 years and included a 7-ms silent gap as one of the deviants. They reported larger mismatch field amplitudes in the older children (5–7 years). However, because the response to the gap deviant was not analyzed separately from the responses to the other deviant types, conclusions specific to gap processing are limited.

Although several studies have used MMN to investigate auditory deficits in children with APD, they have generally used speech or pure-tone deviants rather than gap deviants. The findings,

however, have been inconsistent (Bauer et al., 2009; Hestvik et al., 2023; Koravand et al., 2017; Mohideen & Thangaraj, 2023; Pinto et al., 2024; Rocha-Muniz et al., 2015; Roggia & Colares, 2008; Sharma et al., 2025). Sharma et al. (2025), for example, recorded MMN responses to a /da/–/ga/ contrast in 85 children with LiD grouped according to cognitive profiles. They reported that MMN was difficult to detect in children with APD. Mohideen and Thangaraj (2023) used pure-tone stimuli in which the deviant stimulus was shorter than the standard and found that APD/dyslexic children had smaller MMN amplitudes than controls. In contrast, Pinto et al. (2024) found no group differences in MMN amplitude when comparing children with CAPD and TD children aged 9 to 12 years, using a standard /da/ and a deviant /ta/. Koravand et al. (2017) also examined MMN responses to both verbal and non-verbal deviants in children aged 9 to 12 years with APD and TD controls. In their different conditions, the standard stimulus was either speech /ba/, non-verbal /ba/, or a 1-kHz pure tone, while the deviant was either speech /da/, non-verbal /da/, or a 2-kHz pure tone. They likewise found no significant group differences. Thus, electrophysiological evidence of auditory change detection deficits in APD remains mixed, and very little is known about electrophysiological responses to gap deviants in this population.

In most MMN studies, the deviant differs from the standard from its onset and continues to differ throughout its duration. In some studies, however, the change is brief and occurs at different times after stimulus onset. For example, a standard may last 400 ms, while the deviant also lasts 400 ms but contains a brief 50-ms frequency change at either 100 ms or 300 ms. In such cases, the timing of the deviant feature relative to the onset of the stimulus varies. A number of studies have examined the effect of the timing of stimulus change (Alain et al., 1994; Grimm & Schröger, 2005; Hoonhorst et al., 2012; Näätänen et al., 2004; Schröger et al., 1992; Tamakoshi et al., 2016; Timm et al., 2011; Weise et al., 2007, 2010, 2011; Yabe et al., 2001; Yabe et al., 2005). These studies have generally reported a gradual decrease in MMN amplitude as the onset of change is delayed. For example, Näätänen et al. (2004) found that when the deviant occurred earlier in time, MMN amplitude was larger, whereas delaying the onset of the change led to a progressive reduction in MMN amplitude. This effect, known as the temporal distance or temporal position effect, has been most consistently reported for duration deviants (Grimm et al., 2004, 2006; Grimm & Schröger, 2005), but it has also been observed for other types of stimulus changes. Schröger et al. (1992), for instance, showed that a brief frequency modulation at 150 ms elicited a larger MMN than one at 250 ms. Grimm et al. (2006) found that only frequency modulations occurring within

the first 300 ms of a sound elicited a clear MMN, while changes occurring later were much reduced or absent. Importantly, participants were still able to behaviourally detect these later-occurring deviants as well as earlier ones (Grimm et al., 2006; Weise et al., 2007, 2010; Timm et al., 2011; Hoonhorst et al., 2012). Therefore, the MMN reduction cannot be explained simply by an inability to perceive the later change.

The temporal integration theory has been proposed to explain this effect (Näätänen & Winkler, 1999; see also Weise et al., 2023). According to this account, the initial analysis of sound features such as intensity, frequency, and location allows them to be integrated into a single perceptual unit or auditory object (Arnott & Alain, 2002; Bizley & Cohen, 2013). This integration occurs within a temporal window of integration (TWI) that begins at sound onset and forms a representation of the sound in sensory memory. A major constraint of this process is that integration appears to be limited to approximately 150 to 300 ms. As a result, early portions of a sound contribute more strongly to its representation than later portions. This temporal limit has been attributed either to the restricted capacity of the auditory store or to the brief duration of auditory sensory memory (Cowan, 1984; Grimm & Schröger, 2005; Näätänen & Winkler, 1999).

There may, however, be an important exception to this temporal distance effect: the onset time of a gap in a deviant stimulus. Abrupt transient changes within a stimulus are thought to initiate new integration windows, thereby segmenting a long-duration sound into separate parts (Weise et al., 2007). In this way, the usual temporal constraints of sound representation may be overcome. Weise et al. (2023) noted that a transient change strong enough to initiate a new integration window should also elicit an N1-P2 response. In previous studies showing strong temporal distance effects for frequency deviants, sudden transient changes were intentionally avoided by using gradual frequency modulations. For example, Grimm and Schröger (2005) introduced a brief frequency modulation into a 1000-ms standard tone gradually, thereby avoiding a strong transient. In Weise et al. (2007), a frequency change occurring at 550 ms did not elicit an MMN, presumably because it occurred outside the main integration period. However, when a 25-ms gap was inserted at 350 ms, the same subsequent frequency change elicited an MMN. This finding suggested that the gap initiated a new integration window, allowing the later change to be automatically detected.

Not all gaps, however, appear to produce a sufficiently salient transient to reset auditory integration. Yabe et al. (2001) presented 170-ms, 1000-Hz tones composed of five short segments. Occasionally, one of the later segments was omitted, creating a silent period or gap. If a transient gap reliably initiated a new integration window, each gap should have elicited an N1-P2 in addition to an MMN, regardless of its temporal position. This was not observed. A gap occurring in the fifth position elicited only a very small MMN and no N1-P2. Weise et al. (2023) suggested that the rise-and-fall time used in that study may not have been fast enough to generate a sufficiently salient transient. However, Tamakoshi et al. (2016) replicated Yabe et al. (2001)'s findings using even faster rise-and-fall times and still found no N1-P2. These results suggest that not all gaps reliably initiate a new integration window.

Taken together, these findings directly motivated the present thesis. First, it remains unclear whether the timing of silent gap onset influences the deviant-related response in the same way that temporal position influences MMN for other deviant types. Second, ERP studies on the timing of stimulus change have not yet been conducted in TD and APD children using gap deviants. Third, because conventional oddball paradigms are time-consuming, a more efficient method is required, particularly for use with children and clinical populations.

The present thesis, therefore, employed a multi-deviant paradigm (Näätänen et al., 2004), which allows multiple deviant stimuli to be presented within a single recording sequence. This approach is considerably more time-efficient than the traditional oddball design, in which only one deviant is presented per sequence (Gomes et al., 1997). To study the effect of temporal position using a single-deviant oddball design, separate recording sessions would be needed for each deviant onset time. Because MMN amplitude is small, typically around 1 μ V, and embedded in much larger EEG background activity, a large number of stimulus presentations is required for reliable averaging. In most MMN studies, the deviant must be presented 100 to 200 times, while the standard is presented well over a thousand times. Even with relatively rapid stimulus presentation, a single recording session may last 20 to 30 minutes. If several deviant onset times are studied separately, testing time becomes extremely long, which is especially problematic for young children and clinical populations (Duda-Milloy et al., 2019).

In 2004, Näätänen introduced a multi-feature paradigm in which several different deviants, each representing a change in a different feature of the standard stimulus, were presented within

the same sequence. This approach substantially reduced testing time relative to repeated single-deviant oddball paradigms. Duda-Milloy et al. (2019) later used a related multi-deviant paradigm to examine the effect of gap duration on MMN. In that study, six different gap durations were presented within the same sequence. Because the same feature of the standard was manipulated, namely the insertion of a gap, the paradigm was referred to as multi-deviant rather than multi-feature (Duda-Milloy et al., 2019; Duda et al., 2020; Duda et al., 2025). In those studies, the gap was always inserted in the centre of the standard stimulus, and the results showed that MMN measures obtained using the multi-deviant paradigm were comparable to those obtained using the traditional one-deviant oddball condition. Importantly, MMN amplitude increased with increasing gap duration. These findings suggest that a multi-deviant gap-detection paradigm can yield reliable data in a much shorter time.

In the present thesis, a time-efficient multi-deviant paradigm was used in which a brief silent gap occurred at six different onset times, ranging from 15 to 400 ms after the onset of the deviant stimulus. One concern, however, was that although the onset time of the gap varied across deviants, its duration remained constant. In the Duda-Milloy et al. study, by contrast, all deviants differed in duration. It was therefore not known whether the multi-deviant approach would also be suitable for studying temporal position when deviant duration remained identical. For this reason, results obtained from separate oddball sequences were compared with those obtained from a single multi-deviant sequence.

While a few studies have recorded the MMN to gaps, several important questions remain unanswered. In particular, it is still unclear whether the temporal position of a silent gap (sooner or later after the onset of the actual deviant) affects the deviant-related response in the same way as the temporal position effect reported for other types of deviants. In addition, this question has not yet been examined in TD and APD children using a time-efficient electrophysiological paradigm. The present thesis was designed to address these gaps by examining the effects of gap onset time on the DRN in adults, TD children, and children with APD, using both behavioural and electrophysiological measures.

Based on the literature reviewed above, it was hypothesized that the temporal onset of a silent gap would influence the deviant-related response. More specifically, gap deviants occurring earlier in the stimulus were expected to elicit larger DRN amplitudes than those occurring later,

consistent with the temporal position effect reported for other types of auditory deviants. However, because gaps may create salient transient changes that can initiate a new integration window, it was also expected that this effect might be reduced or differ from that reported for non-gap deviants. It was further hypothesized that this reduction would occur earlier in children than in adults, reflecting maturational differences in auditory temporal integration and a more limited auditory sensory memory capacity in children. Finally, it was hypothesized that children with APD would exhibit reduced deviant-related responses relative to TD children, particularly at gap onset times if APD is associated with impaired auditory processing and/or reduced auditory sensory memory capacity. Alternatively, if poorer behavioural gap-detection performance in APD primarily reflects attentional demands rather than impaired auditory encoding, then electrophysiological responses recorded under passive listening conditions would be expected to show minimal group differences.

1.2 Objective of the thesis

The main purposes of this thesis were to create a time-efficient novel multi-deviant paradigm to investigate the effects of gap duration and temporal position, and to determine how these two factors influence the automatic detection of the gap by the change detection system. The present thesis was designed to address these questions in adults, TD children, and children with APD, using both behavioural and electrophysiological measures. The MMN was recorded passively by having participants engage in a visual task, watching a video while ignoring the auditory sequence. This is important. Traditional behavioural gap-detection methods require sustained active attention throughout the task. Children may have greater difficulty maintaining attention over prolonged testing periods, and behavioural performance may therefore be influenced by attentional and other task-related demands in addition to auditory temporal processing. Specifically, the thesis focused on evaluating the DRN and on determining how this response varies as a function of the temporal onset of the gap. By comparing electrophysiological and behavioural measures, the thesis also explored whether these measures showed different patterns in children with APD, providing preliminary information that may help guide future investigation of the factors contributing to temporal processing difficulties. First, the objective was to investigate the effects of the temporal position of a gap on the DRN using a multi-deviant paradigm and to compare the responses obtained with this paradigm with those obtained using traditional single-

deviant oddball sequences. Previous studies of the effect of temporal position have used oddball paradigms that were repeated for each temporal position (Grimm & Schröger, 2005; Grimm et al., 2006; Weise et al., 2010, 2011; Timm et al., 2011). These studies also primarily used other types of acoustic changes, such as frequency modulation, rather than gap deviants. This initial study compared the effects of gap timing using both a multi-deviant paradigm and multiple repetitions of a single-deviant oddball paradigm. These results were also compared with the effects of the temporal position of the gap on an active behavioural gap-detection task. The duration of this testing was very long. For this reason, only young adults participated in this study.

Second, the thesis aimed to examine developmental differences in auditory temporal processing by comparing adults and TD children. The effects of temporal position on change detection have not yet been examined in children. In particular, this study investigated whether DRN amplitude varied as a function of gap onset time across the two age groups, with the goal of characterizing maturational differences in temporal resolution and temporal integration.

Third, the thesis aimed to explore whether DRN responses differed between children with APD and their TD peers using the novel multi-deviant paradigm. This phase investigated whether the electrophysiological index of gap processing, DRN, differed between the two groups, and whether any such differences depended on the gap onset time. Because this was a small exploratory pilot study, the behavioural and electrophysiological results were considered together to explore possible patterns of group differences and to provide preliminary insight into factors that may contribute to poorer gap detection in children with APD.

1.3 Overview of the thesis

Chapter 2 presents a review of the literature relevant to this thesis. It begins with an overview of APD and then reviews temporal processing, with particular focus on the two key aspects examined in this thesis: temporal resolution and temporal integration. The chapter summarizes behavioural studies of temporal resolution and temporal integration in both TD children and children with LiD. It then reviews electrophysiological research on MMN elicited by gap-inserted paradigms, including studies in normal young adults, TD school-age children, and children with APD.

Chapter 3 includes the first study conducted to address the research objectives. This chapter describes the development of the novel multi-deviant gap detection paradigm in normal-hearing

adults. Specifically, it examines whether silent gaps at different onset times within a noise stimulus can reliably elicit the DRN, and compares responses obtained with the multi-deviant paradigm with those elicited by a traditional single-deviant paradigm. Behavioural gap-detection performance was also examined as a function of gap duration and onset time. This study therefore provided a basis for using the multi-deviant paradigm in the subsequent studies with children. Chapter 4 presents the second study, which examines developmental differences in auditory temporal processing by comparing TD children with adults. This chapter investigates how DRN amplitude changes as a function of gap onset time across the two groups and evaluates whether children and adults differ in the neural processing of gaps occurring at different temporal positions. This study addresses the second objective of the thesis by characterizing maturational differences in temporal resolution and temporal integration using the novel paradigm.

Chapter 5 presents the third study, which focuses on children with APD and compares them with their TD peers using both electrophysiological and behavioural measures. This chapter evaluates whether DRN responses differ between the two groups, whether group differences in DRN amplitude depend on gap onset time and gap duration, and how electrophysiological findings relate to behavioural gap-detection performance. Because of the exploratory nature and small sample size of the study, the behavioural and electrophysiological findings are used to describe possible patterns of group differences and to provide preliminary insight into factors that may contribute to poorer gap detection in children with APD. Finally, Chapter 6 provides a general discussion of the findings from all three studies. It integrates the results across adults, TD children, and children with APD, and discusses their implications for understanding auditory temporal processing and integration, developmental changes in gap detection, and possible factors contributing to behavioural and electrophysiological differences in children with APD. The chapter concludes by considering the theoretical and clinical significance of the work and outlining directions for future research.

Chapter 2: Literature Review

This chapter reviews the literature related to auditory temporal processing and gap detection. It begins with APD and LiD in children. This section explains why the diagnosis of APD is still controversial and why LiD is sometimes used as a broader term. The chapter then reviews auditory temporal processing and its main domains, with a focus on temporal resolution. Behavioural measures of temporal resolution are discussed in TD children and in children with APD. The effect of gap position on behavioural gap detection is also reviewed. This is important because gap detection may depend not only on the gap's duration but also on its position within the stimulus. The chapter then discusses ERPs as objective measures of auditory change detection. It focuses on MMN and DRN responses to gap-inserted stimuli. Finally, the chapter reviews how gap duration and gap position affect electrophysiological responses, and how these responses develop in TD children and children with APD. Together, these sections provide the background for the present thesis and identify the research gap addressed in this work.

2.1 Auditory processing disorder (APD)

Children may present to pediatricians and audiologists with LiD despite having clinically normal audiometric thresholds and no obvious peripheral hearing loss. A subgroup of these children is referred for assessment of (central) APD (CAPD). However, the diagnosis of APD remains controversial. Recent literature continues to emphasize that APD is a heterogeneous construct, that diagnostic outcomes depend strongly on the criteria and test battery used, and that there is still no universally accepted gold-standard diagnostic tool or reference standard (American Academy of Audiology [AAA], 2010; ASHA, n.d.; Gitto et al., 2025; Hind et al., 2011; Wilson, 2013). Reported prevalence also remains highly variable. Current American Speech-Language-Hearing Association (ASHA) guidance summarizes prevalence estimates in school-age children ranging from 0.2% to 6.2%, and previous work has shown that the proportion of referred children meeting diagnostic criteria can vary markedly depending on the testing protocol and cut-off used (American Academy of Audiology [AAA], 2010; ASHA, n.d.; Gitto et al., 2025; Hind et al., 2011; Wilson, 2013).

Historically, three broad perspectives have been proposed regarding the origins of APD. The first views APD primarily as a bottom-up disorder, reflecting impaired neural processing

within the central auditory nervous system despite normal peripheral hearing sensitivity (ASHA, 2005). The second emphasizes top-down influences and interprets many listening problems as arising from broader cognitive mechanisms such as attention, memory, executive function, and language processing rather than from a purely auditory deficit (Ferguson et al., 2011; Moore et al., 2010). A more integrative perspective, reflected in the British Society of Audiology guidance and supported by more recent multidisciplinary work, considers APD in relation to both central auditory mechanisms and other neural systems that shape listening, including cognition, language, and attention (Moore et al., 2018).

In parallel, several authors have argued that the term LiD is often more appropriate when referring to the presenting complaint itself (Dillon & Cameron, 2021; Moore, 2018; Moore et al., 2025). In this view, LiD functions as a broader, symptom-based umbrella term for difficulty understanding or responding to auditory information, especially in everyday situations such as listening in noise or following spoken instructions, whereas APD refers more specifically to auditory processing deficits that may contribute to that complaint. Importantly, LiD may arise from auditory, cognitive, language, attention, or peripheral hearing factors, either in isolation or in combination (Bamiou et al., 2001; Dawes et al., 2008; Dillon & Cameron, 2021). Recent work in children with normal audiograms supports this distinction, showing that caregiver-reported LiD is associated with weaknesses across listening and cognitive measures and with a broad range of co-occurring clinical concerns beyond the auditory domain (Moore et al., 2025; Petley et al., 2021). In the present thesis, the term APD is used when referring to children who have received or are being considered for a clinical diagnosis of APD. The broader term LiD is used when referring to the presenting listening complaint, which may reflect auditory, cognitive, language, attention, or peripheral hearing factors. Therefore, the thesis recognizes LiD as the broader clinical concern, while focusing more specifically on temporal auditory processing mechanisms that may contribute to APD-related listening difficulties.

Because temporal processing is one of the auditory mechanisms that may contribute to LiD in children with APD, the next section focuses on auditory temporal processing and its main subdomains.

2.2 Auditory Temporal Processing

Auditory temporal processing refers to the perception, encoding, and interpretation of acoustic information that changes over time (Musiek et al., 2005). Because speech unfolds over multiple timescales, efficient temporal processing is essential for perceiving rapid phonemic contrasts such as voice onset time (VOT), as well as slower suprasegmental cues such as lexical stress, rhythm, and prosody (Chermak & Musiek, 1997). More generally, temporal processing is thought to support a wide range of auditory abilities, including auditory discrimination, localization and lateralization, pattern perception, and listening in degraded or competing acoustic conditions (ASHA, n.d.; Chermak & Musiek, 1997; Shinn et al., 2009).

Auditory temporal processing is commonly divided into four major domains: temporal resolution, temporal ordering or sequencing, temporal integration, and temporal masking (Chowsilpa et al., 2021; Musiek et al., 2005). Among these, temporal ordering and temporal resolution remain the most established in clinical assessment, whereas temporal integration and temporal masking are less standardized and have fewer routinely used clinical tools (Chowsilpa et al., 2021). Temporal ordering is typically assessed using pattern perception tasks such as the Frequency Pattern Test and Duration Pattern Test (Musiek, 1994), whereas temporal resolution is most commonly evaluated using gap-detection measures such as the Random Gap Detection Test (RGDT; Keith, 2000) and the Gaps-in-Noise (GIN) test (Musiek et al., 2005).

2.3 Temporal resolution

Temporal resolution is defined as the auditory system's ability to detect and encode rapid changes in the envelope of a sound stimulus (Shinn et al., 2009). Temporal resolution plays a significant role in speech perception, as it involves processing temporal information at different rates. For example, lexical and syntactic information is conveyed by changes in the speech envelope, and variations in gap duration can affect phonemic discrimination (Hoover et al., 2015). This ability helps distinguish dips in fluctuation noise, enabling the detection and assembly of target speech segments and thereby contributing to speech perception in noise (Bernstein & Grant, 2009).

Several methods have been suggested for evaluating temporal resolution. Various tests have been created for clinical assessments, including the RGDT (Keith, 2000), the Adaptive Test of Temporal Resolution (ATTR) (Lister et al., 2006), and the GIN (Musiek et al., 2005). The

RGDT consists of various subtests in which pairs of tone or noise bursts are played in a fixed, nonadaptive format with nine items per interval. Gap durations are randomly presented, and the listener's task is to indicate whether they heard one or two bursts during each interval. Gap durations typically vary on the order of milliseconds (Keith, 2000). The GDTs are typically around 2-3ms in highly trained listeners (Phillips, 1999), and slightly higher for less experienced listeners (Phillips & Smith, 2004). In ATTR, the gap duration changes based on the participant's responses. This can enhance the precision of measuring GDTs (Lister et al., 2006). The GIN test comprises a series of 6-sec segments of broadband noise bursts, each containing 0 to 3 silent gaps of different durations (from 2ms to 20ms) embedded at different positions within the stimulus. The listener is tasked with pressing a button upon hearing a gap, and scoring is based on the shortest gap duration at which the listener accurately recognizes a minimum of four out of six gaps, along with the overall percentage of correct responses for all gap durations. Typically, the test is designed within the same channel, meaning that the stimulus preceding the gap shares the same spectrum as the stimulus following the gap (Musiek et al., 2005). As with RGDT and ATTR tests, the goal of GIN is to find the smallest detectable gap for a listener, called the GDT.

The position of a silent gap is in the middle of the acoustic signal for the RGDT and ATTR evaluations (Keith, 2000; Lister et al., 2006), however, it changes in the GIN test (Musiek et al., 2005). Therefore, the patient's task in the GIN test is straightforward: they just need to push a button when a gap in noise is perceived. On the other hand, unlike the RGDT, the GIN involves continuous noise segments interrupted by gaps near the listener's detection threshold, requiring quick responses without visual cues or pauses for contemplation, potentially affecting its interpretation as a measure of temporal resolution (Hoover et al., 2015).

Both RGDT and GIN may be influenced by cognitive factors in addition to auditory temporal resolution. In the RGDT, listeners judge whether one or two sounds are heard for each stimulus pair (Keith, 2000), whereas the GIN requires continuous monitoring of noise and an immediate response when a gap is detected (Musiek et al., 2005). The sustained monitoring and rapid-response requirements of the GIN may involve vigilance and processing speed (Hoover et al., 2015). Thus, gap-detection performance may reflect cognitive demands in addition to auditory temporal resolution. Hoover et al. (2015) proposed that differences between GIN and traditional GDT thresholds may partly reflect working memory, sustained attention, and processing speed. They further hypothesized that reduced abilities in these cognitive domains could increase the

discrepancy between the two measures, although these factors were not directly assessed in their study. Some children with APD have also been reported to show poorer working-memory abilities than TD children, although this is not a consistent characteristic of all children with APD (Ferguson et al., 2011; Tomlin et al., 2015). Working memory may influence performance on some auditory-processing tasks, but the strength of this relationship varies across tasks and individuals (Tomlin et al., 2015).

2.3.1 Behavioural assessment of temporal resolution in typically developing children

Behavioural studies of temporal resolution in TD children have reported different developmental patterns. Several studies using the GIN suggest that temporal-resolution performance is relatively mature by school age, with little or no effect of age, ear, or gender across the age ranges examined (Amaral & Colella-Santos, 2010; Ismaail et al., 2019; Marculino et al., 2011; Perez & Pereira, 2010; Shinn et al., 2009). In contrast, studies using other psychophysical or adaptive gap-detection paradigms have reported age-related improvements extending through childhood and, in some cases, into adolescence (Boboshko et al., 2023; Buss et al., 2014, 2017; Gudkar et al., 2026; Wightman et al., 1989). Together, these findings suggest that the developmental course of temporal resolution depends partly on the test format and acoustic characteristics of the stimuli. Several earlier studies reported no substantial age, ear, or gender effects on GIN performance in normal-hearing children. Shinn et al. (2009) showed that children between 7 and 18 years of age could complete the GIN successfully and that their performance did not differ significantly across age groups or between ears. Perez and Pereira (2010) reported similar findings in 11- and 12-year-old children. They found a mean gap threshold of 5.05 ms and a mean correct score of 71.70%, with no significant differences across age, ear, or gender. They also reported that in 78.27% of the sample, the gap threshold was 5 ms or less, and suggested this value as a normal reference for that age range. Likewise, Amaral and Colella-Santos (2010) found no significant differences among children aged 8 to 10 years. They proposed cut-off values of 6.1 ms for the mean gap threshold and 60% for correct responses. Marculino et al. (2011) also found no differences between ears or genders in 9-year-old children, with mean GDTs of 4.4 ms for the right ear and 4.2 ms for the left ear. Ismaail et al. (2019), using a larger sample divided into four age groups from 6 to 16 years, again found no significant effects of age, ear, gender, list, or retest on GIN performance, and concluded that temporal resolution measured by the GIN is already adult-like by school age.

However, not all studies support a completely age-independent view. Wightman et al. (1989) showed that children aged 3 to 7 years old, especially the younger ones, had higher gap-detection thresholds than adults when gaps were presented in half-octave-band noise centered at 400 Hz and 2000 Hz, with poorer performance in the lower-frequency condition. Later, Buss et al. (2014) and Buss et al. (2017) examined gap detection using psychophysical paradigms with different envelope and frequency conditions (Buss et al., 2014; Buss et al., 2017). Buss et al. (2014) examined gap detection in 5.2 to 15.6-year-old TD children and 12 adults using continuous noise bands centred at 2000 Hz. They compared four stimulus types: a 25-Hz-wide Gaussian noise, a 1000-Hz-wide Gaussian noise, a 25-Hz-wide low-fluctuation noise with reduced inherent envelope modulation, and a 25-Hz-wide staccato noise with enhanced envelope modulation. For the narrowband stimuli, adults showed the poorest performance in the staccato condition, intermediate performance in the Gaussian condition, and the best performance in the low-fluctuation condition. Children showed the same general pattern but were less affected by envelope fluctuations than adults. In addition, children reached adult-like performance at a younger age for the 1000-Hz-wide Gaussian noise than for the 25-Hz-wide low-fluctuation noise. These findings suggest that both envelope characteristics and the availability of cues across frequency influence gap-detection performance, and that the ability to use broad spectral cues matures relatively early in childhood. Buss et al. (2017) found lower thresholds for wideband noise when the onset and offset transitions bounding the gap were brief (4-ms raised-cosine ramps) rather than more gradual (40-ms ramps). They also found lower thresholds for narrowband noise centred at 5000 Hz than at 500 Hz. Performance reached adult levels only after about 11 years of age for all four stimulus conditions, although some younger children already performed at adult levels. These findings suggest that the developmental course of temporal resolution is not fully captured by a single clinical test and may depend on stimulus type. More recent studies have also suggested that some maturation continues during childhood. Boboshko et al. (2023), using the RGDT, found a significant age effect in full-term children between 6 and 11 years of age. The youngest group, aged 6 to 7 years, performed more poorly than the older groups. This result does not necessarily contradict the earlier GIN findings, but it does suggest that temporal resolution may appear stable or age-dependent depending on the test format. In the same direction, Gudkar et al. (2026) established normative data for five new non-speech auditory processing tests in normally hearing children aged 6 to 11 years. Two of these tests assessed gap detection: an adaptive within-channel

(WC) and an adaptive between-channel (BC) gap-detection test. In the WCGD test, the leading and trailing markers were identical broadband white-noise bursts, each 300 ms in duration, with 0.3-ms onset and offset ramps. In the BCGD test, the markers had non-overlapping spectra. The leading marker was band-pass filtered noise from 1500 to 4000 Hz with a duration of 30 ms, whereas the trailing marker was a harmonic complex from 100 to 1000 Hz with a duration of 400 ms. The BCGD stimulus was designed as a simplified non-speech approximation of the burst-vowel structure of stop consonants, so that the gap functioned in a way similar to VOT. Across the full test battery, Gudkar et al. found a significant effect of age, but not sex, on all thresholds. These findings suggest that temporal auditory abilities continue to mature during the primary school years, even though many children may already perform within the normal clinical range. Therefore, temporal resolution in TD children appears relatively mature by school age, but may not be fully stable across all tasks and stimulus conditions.

2.3.2 Behavioural assessment of temporal resolution in children with APD

Behavioural studies generally suggest that children with APD perform more poorly than TD children on temporal-resolution tasks (Balén et al., 2009; Dias et al., 2012; Iliadou et al., 2014; Jain et al., 2025; Phillips et al., 2010). However, the findings are not fully uniform across paradigms. The literature is also relatively limited, especially when only studies on children with diagnosed APD are considered. Most available studies have used behavioural gap-detection tasks such as the RGDT, the GIN test, or experimental WC and BC paradigms (Balén et al., 2009; Phillips et al., 2010; Dias et al., 2012; Iliadou et al., 2014; Jain et al., 2025).

One of the earlier studies in this area was conducted by Balén et al. (2009). They compared children with normal peripheral hearing and normal auditory processing, children with conductive hearing loss, and children with APD, all between 7 and 10 years of age. The control group had hearing thresholds of 15 dB HL or better, normal tympanometry, and normal performance on the auditory-processing test battery. Children in the conductive hearing loss group had mild-to-moderate hearing loss with type-B or type-C tympanograms in at least one ear. Children in the APD group had normal pure-tone thresholds and tympanometry but abnormal performance on at least two tests from the auditory-processing battery. Temporal resolution was assessed using random gap detection test (RGDT). The children with APD and children with conductive hearing loss performed worse than the normal-hearing group at all tested frequencies. However, gap-

detection thresholds did not differ significantly between the APD and conductive hearing-loss groups. The authors concluded that APD and conductive hearing loss negatively affected temporal-resolution performance and that the gap-detection test was sensitive to these alterations (Balen et al., 2009). Dias et al. (2012) extended this line of work by assessing 131 individuals with APD and 94 controls between 5 and 25 years of age using the RGDT. They reported significant age-group differences in both the APD and control groups. They also found that 48% of the children with APD failed the RGDT, and that the failure rate was highest in the 5- to 6-year-old group, where it reached 86%. In addition, the authors found strong correlations among the four RGDT frequency conditions and suggested that a single frequency may be sufficient for clinical testing. They also recommended that the RGDT not be administered to children younger than 7 years because immature or nonauditory factors may affect performance at that age (Dias et al., 2012).

Phillips et al. (2010) examined auditory gap detection in children referred to APD assessment and compared them with TD controls. Their study included both WC and BC paradigms. The WC task used markers with similar spectral characteristics before and after the gap, whereas the BC task used markers with different spectral characteristics. The authors found that WC gap thresholds were very similar across groups. In contrast, BC gap thresholds differed significantly, with the largest difference observed between the control and APD-positive groups. Their results suggested that not all gap-detection paradigms are equally sensitive to group differences in APD (Phillips et al., 2010).

Iliadou et al. (2014) compared two clinical tests of auditory temporal resolution in children with CAPD. Their CAPD group included 22 children between 7.5 and 17 years of age. They administered both the RGDT and the GIN test. In 13 of the 22 children with CAPD, it was not possible to calculate an RGDT threshold because of inconsistent responses. When inconsistent cases were excluded, RGDT thresholds differed significantly across the study populations, and the authors also found significant differences between RGDT and GIN thresholds within the CAPD group. These findings suggest that different behavioural temporal-resolution tests are not interchangeable because they place different task demands on the child. The RGDT requires the child to judge whether two sounds were perceived as one or two, whereas the GIN requires the child to monitor continuous noise and signal when a brief gap is detected; therefore, inconsistent performance on one test does not necessarily imply the same outcome on the other (Iliadou et al., 2014).

More recently, Jain et al. (2025) compared 12 children with APD and 12 TD children between 10 and 12 years old using adaptive gap-detection tasks. They measured monaural gap-detection thresholds under three stimulus conditions. The first used 500-ms broadband Gaussian noise, with a silent gap inserted symmetrically around the stimulus's midpoint. The second was a WC condition, in which the leading and trailing markers were both narrowband noises centred at 2 kHz. The third was an across-channel condition, in which the leading marker was centred at 2 kHz and the trailing marker was centred at 1 kHz. Significant group differences were found only in the across-channel condition. No significant differences were found in the broadband noise or WC conditions. The authors suggested that the across-channel task is more demanding because it requires comparing temporal information across two different auditory channels, whereas the other two conditions rely on markers within the same spectral channel. Their results therefore suggest that group differences in APD may depend on the acoustic structure of the stimuli and the specific temporal demands of the task (Jain et al., 2025).

Taken together, the available behavioural literature supports the view that temporal resolution can be affected in children with APD. At the same time, the findings show that the magnitude and even the presence of group differences may depend on the task used. Simpler paradigms do not always separate children with APD from controls, whereas more demanding paradigms may be more sensitive in some studies.

2.3.3 The effect of gap position on the behavioural gap detection threshold

Studies have shown that behavioural gap-detection performance is affected by the temporal position of the gap within the stimulus (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999). In general, they suggest that gaps are more difficult to detect when they occur near the onset of the stimulus than when they occur later.

For example, Forrest and Green (1987) examined 100-ms broadband-noise bursts in which the gap occurred 10, 30, 70, or 90 ms after stimulus onset. They reported little overall effect of gap position, except that performance was poorer when the gap occurred about 30 ms after onset. Thus, even in short noise bursts, the temporal placement of the gap could influence the threshold. Phillips et al. (1997) examined this question more directly by varying the duration of the leading marker from 5 to 300 ms while keeping the trailing marker fixed at 300 ms. They showed that gap-detection thresholds improved as the duration of the leading marker increased, especially when the

leading and trailing markers were spectrally different and therefore likely activated different perceptual channels. Their findings suggested that when gap detection depends on comparing activity across different channels, a longer pre-gap segment provides a stronger basis for the temporal comparison needed to detect the silent interval.

Studies in children have also shown that early gap placement is more difficult. Diedler et al. (2007) tested TD children with a mean age of 8.7 years using 100-ms white-noise bursts containing gaps of 3, 6, 10, or 30 ms inserted either 5 or 50 ms after stimulus onset. They found that thresholds were higher when the gap was closer to the onset of the noise burst. This result showed that the temporal placement of the gap affects psychoacoustic performance even in school-age children. More recently, Gay et al. (2020) examined 8- to 19-year-olds using 200-ms bandpass-noise stimuli in which the gap occurred 10, 15, 20, 30, or 50 ms after onset. They found that thresholds improved with age at all gap positions. They also found that younger listeners did not show the same benefit from later gap onset times as older listeners. These findings suggest that the effect of gap position is present across development, but that the ability to benefit from a longer leading marker continues to mature through adolescence (Diedler et al., 2007; Gay et al., 2020).

Physiological work is consistent with this behavioural pattern. Eggermont (1995) recorded responses from small clusters of neurons in the cat auditory cortex while presenting a 1-s wideband noise containing either an early gap, placed 5 ms after onset, or a late gap, placed 500 ms after onset. Neural gap-detection thresholds were below 5 ms for the late-gap condition, but were about 40 to 50 ms for the early-gap condition. Eggermont suggested that the strong onset response to the initial part of the noise suppresses neural responses to the trailing marker when the gap occurs too early. Although this was an animal study, it provides a useful physiological explanation for why early gaps are often more difficult to detect behaviourally (Eggermont, 1995).

Negri et al. (2024) provided additional evidence that behavioural gap-detection thresholds depend on how the gap is embedded within the stimulus. In a preliminary experiment, they tested 18 young adults with normal hearing (NH) using a two-alternative forced-choice GIN task. The stimulus was a 400-ms noise burst presented at 75 dB SPL. They used either broadband noise, low-pass filtered at 30 kHz, or one-octave narrowband noise centered at 1, 2, 4, 8, or 16 kHz. In half of the trials, a silent gap was inserted pseudorandomly at 100, 200, or 300 ms after stimulus onset. Gap salience was also manipulated by varying the depth of the gap, so that the temporal

position and perceptual prominence of the gap were less predictable across trials. They found that when gap position and salience varied, detection became more difficult, reflected in higher thresholds and shallower psychometric functions. More specifically, detection was poorest when the gap occurred late in the stimulus, at 300 ms after onset, whereas performance was best for the 200-ms position and intermediate for the 100-ms position. This effect was mainly evident for longer gaps (3 ms or more), whereas for very short gaps (1–2 ms), gap position had little effect on hit rate. Although this study was conducted in young adults rather than children, it supports the general view that behavioural gap detection depends not only on gap duration itself, but also on the temporal placement and acoustic salience of the gap within the stimulus (Negri et al., 2024).

In summary, the literature suggests that the gap-detection threshold is influenced by more than gap duration alone. The gap position within the stimulus is an important factor. In many studies, gaps near stimulus onset are more difficult to detect than gaps that occur later. This effect appears to interact with other stimulus properties, including the duration of the leading marker, the spectral similarity between the leading and trailing markers, and the overall predictability of the gap. Therefore, behavioural gap-detection thresholds should be interpreted in relation to the acoustic context in which the gap is presented, rather than as a fixed measure that is independent of stimulus design.

2.4 Event-related potentials and mismatch negativity

Event-related potentials (ERPs) provide an objective means of examining auditory processing. ERPs are small changes in electrical brain activity recorded from the scalp and time-locked to the presentation of a sensory event. They appear as a waveform with positive- and negative-going deflections that are usually described in terms of polarity, latency, and amplitude (Näätänen & Picton, 1987). Because ERPs do not always require an overt behavioural response, they are useful when behavioural performance may be influenced by factors such as attention, motivation, or task demands (Duda-Milloy et al., 2019; Näätänen et al., 2011).

In auditory research, ERPs are often recorded in a passive paradigm. In this type of paradigm, the participant is asked to attend to a non-auditory activity, such as watching a silent video or reading, while ignoring the auditory stimuli. Even under these conditions, auditory ERP responses can still be elicited reliably. This is one of the main advantages of ERPs over behavioural measures, especially in children and clinical populations (Muller-Gass et al., 2005, 2006;

Näätänen, 1990). Auditory stimuli typically elicit a series of obligatory cortical responses, commonly labelled P1, N1, P2, and N2. These components reflect early stages of auditory processing and are mainly related to the stimulus's physical properties (Näätänen & Picton, 1987).

The MMN is another auditory ERP component, but unlike P1, N1, P2, and N2, it is related to the detection of change. MMN is usually recorded using an auditory oddball paradigm, in which a frequently occurring standard stimulus is presented and, at rare times, a deviant stimulus is introduced by changing one of its acoustic features. The deviant elicits, in addition to the obligatory responses, another negative-going deflection, the MMN. MMN is commonly quantified in a deviant-minus-standard difference wave. It is typically largest over frontocentral scalp regions and, when a nose reference is used, it reverses polarity at the mastoids (Näätänen, 1990; Näätänen et al., 1978).

The MMN is generally considered an index of automatic auditory change detection. In the classical account, the features of the standard stimulus are extracted and stored in a brief auditory sensory memory trace. Each incoming stimulus is then compared with that representation, and a mismatch response (MMR) is elicited when it deviates from the regularity established by the standards (Näätänen, 1990). A more recent view proposes that MMN can also reflect violation of a rule-based pattern extracted from the auditory sequence, rather than only mismatch with a simple memory trace (Winkler et al., 2009). Regardless of the theoretical account, MMN is widely used as an objective measure of auditory discrimination because it can be elicited without requiring the listener to respond actively to the sounds.

The MMN can be elicited by changes in several stimulus dimensions, including frequency, intensity, duration, location, and speech-related features (Näätänen et al., 2007). Its amplitude usually increases as the acoustic difference between the standard and deviant becomes larger (Näätänen, 1992; Näätänen et al., 2005). A more time-efficient way of eliciting MMN responses was proposed by Näätänen et al. (2004). In this multi-deviant, or multi-feature, paradigm, several different deviants are presented within a single sequence rather than in separate oddball runs. In the original version of the paradigm, the deviants differed from the standard in only one dimension, such as frequency, intensity, duration, location, or gap insertion. The MMNs obtained with this new paradigm were reported to be equal in amplitude to those obtained with the traditional oddball paradigm (Näätänen et al., 2004).

In gap-detection studies, the negative response elicited by an infrequent gap deviant has often been labelled as an MMN. However, this response may not always be a pure MMN. In a typical gap paradigm, the deviant is physically identical to the standard until the time at which the gap occurs. The onset of the gap, therefore, acts not only as a signal of deviance but also as an abrupt acoustic transient. These abrupt changes elicit an obligatory N1 response. At the same time, because the gap marks a change from the preceding standard stimuli, it also elicits an MMN. For this reason, the negativity observed in response to a gap deviant is better viewed as a composite response, that is, an N1+MMN, rather than as a pure MMN alone (Duda-Milloy et al., 2019; Näätänen & Picton, 1987). This composite negativity has been referred to as a DRN (Duda-Milloy et al., 2019; Woldorff et al., 1998).

In the present thesis, the term DRN is used to emphasize that the response to a gap is not interpreted as a pure MMN, but as a deviant-related negative waveform that includes both automatic deviance detection and the obligatory cortical response to the abrupt onset of the gap.

2.4.1 Effect of gap duration on automatic deviance detection

The duration differences have been used to evoke MMN as a measure of auditory discrimination, without any attention to its possible role in manifesting temporal auditory processing or resolution ability (Colin et al., 2009; Jaramillo et al., 2000; Okazaki et al., 2006). Other temporal manipulations used to elicit MMN include variations in the rise time of a stimulus, removing one component in a tone pair, decreasing the ISI, and modification of the silent period in a stop consonant within a nonsense word (Lyytinen et al., 1992; Näätänen et al., 1993; Nordby et al., 1988; Pihko et al., 1997; Tervaniemi et al., 1994). Studies have shown that very brief silent gaps within a stimulus, including tone pips (Desjardins et al., 1999), modulated tones (Mulle, 2012), pure tones (Bertoli et al., 2001; Heinrich et al., 2004; Uhlén et al., 2017), noise (Duda-Milloy et al., 2019; Todd et al., 2011), or the combination of a harmonic tone and noise (Takegata et al., 2009) can also trigger MMN. These studies were designed to establish an electrophysiological GDT as a measure of temporal resolution, employing minimal differences between standard and deviant stimuli. Using a gap paradigm to elicit MMN may offer advantages over the duration-difference approach. In the gap paradigm, the duration of the deviant stimulus remains consistent with the standard, thereby avoiding alterations in rhythm or intensity that may arise with duration disparities (Bertoli et al., 2002).

A few studies have investigated the timing of brain responses to gaps in auditory stimuli using MMN. Bertoli et al. (2001) studied MMN responses to silent gaps in pure-tone stimuli. While frontal and central MMN responses were observed to long-duration gap durations (9 or 15ms), no MMN was detected when gap sizes were at or below behavioural detection thresholds in participants with NH. Besides, MMN amplitude increased and latency decreased with longer gap durations, indicating the sensitivity of the ERP response to the relative detectability of gap stimuli (Bertoli et al., 2001). Desjardins et al. (1999) evaluated eight right-handed young adults with NH in order to investigate the possibility of determining GDT using MMN. They inserted three gap sizes (4, 5, or 7ms) between two 2000 Hz tone pip markers. The study revealed that MMN was detected even at gap size near the behavioural GDT, and its amplitude increased by increasing the gap size (Desjardins et al., 1999). Uther et al. (2003) investigated auditory temporal resolution in 10 normal young adults using MMN and its magnetic counterpart (MMNm). The standard stimulus was a 2000-Hz pure tone with a duration of 100ms, and the deviant contained a silent gap of varying duration, including 5, 7, or 11ms. The results showed that smaller gap sizes (3ms) elicited less pronounced MMN and MMNm compared to larger ones (5, 7, or 11ms) (Uther et al., 2003). Heinrich et al. (2004) examined 12 young adults with NH (two withdrawals after behavioural testing) to investigate whether the MMN can differentiate between auditory mechanisms responsible for WC and BC processing. The study used pure tones of 1000 Hz and 2000 Hz as leading and trailing markers to investigate the MMN in WC and BC conditions. Notably, despite a significant fivefold difference in behavioural GDTs between the two conditions, the amplitude and latency of MMN were comparable (Heinrich et al., 2004).

Takegata et al. (2009) evaluated gap detection using MMN and compared conditions with spectrally symmetrical, slightly asymmetrical, and widely asymmetrical markers in 14 young adults. The trailing marker in all conditions was a harmonic tone with four sinusoidal partials. In the two asymmetrical conditions, band-pass filtered white noise was used as the leading marker; in the slightly asymmetrical setup, the noise was divided into four frequency bands (each with a bandwidth of 100 Hz), and the central frequencies of these bands aligned with the sinusoidal partials of the trailing marker. In the highly asymmetrical scenario, the noise underwent filtering using a band-pass filter ranging from 650 to 3000 Hz, approximately aligned with the frequency range of the trailing marker. Conversely, the leading marker in the symmetrical condition mirrored the trailing marker. The results indicated that slightly asymmetrical markers reduced MMN

amplitude, whereas widely asymmetrical markers affected both amplitude and latency, suggesting non-uniform effects across different magnitudes of asymmetries.

Duda-Milloy et al. (2019) developed the multi-deviant gap detection paradigm in which six deviants with different silent gap durations were presented in the middle of the stimulus (Duda-Milloy et al., 2019). The gap duration ranged from 2 to 40 ms. They compared the amplitudes of DRN between the multi-deviant and one-deviant conditions. The results indicated comparable DRN parameters between the two conditions (except when the mastoid was used as a reference). The amplitudes of DRN increased with longer gap duration. However, no DRN was detected when the gap duration was 2ms, possibly because it is below the behavioural GDT. This study showed that a multi-deviant WC gap-detection paradigm can be more time-efficient than a single-deviant one, achieving the same results in a very short time.

Duda et al. (2025) further examined the effect of gap duration by adding background masking noise to a multi-deviant gap paradigm. In 14 normal-hearing adults, they presented an 80 dB SPL standard stimulus and deviants containing silent gaps from 2 to 40 ms under no-masker, 60 dB SPL masker, and 80 dB SPL masker conditions. They measured the DRN together with the following P2. The DRN–P2 amplitude increased with gap duration in the no-masker and low-masker conditions, but not in the high-masker condition. These findings suggest that electrophysiological sensitivity to gap duration is preserved under quiet and low noise conditions, but becomes less reliable when masking noise is strong (Duda et al., 2025).

Despite variations in stimuli, recording techniques, and analytical approaches across studies, the consistent observation of MMN to gap duration and its relationship with GDTs underscores the potential utility of MMN as an electrophysiological marker for temporal processing abilities in young adults. Moreover, recent methodological advances, such as the multi-deviant paradigm introduced by Duda-Milloy et al. (2019), offer promising avenues for more efficient assessment of temporal processing using MMN, highlighting its potential in both research and clinical applications.

2.4.2 Temporal distance effect in automatic deviance detection

Most MMN studies use a standard oddball contrast in which the deviant differs from the standard from the beginning of the stimulus and remains different for its entire duration. In this case, the onset of the deviant and the onset of the acoustic change coincide. A different situation

arises when the standard and deviant are physically identical at stimulus onset, and the change occurs only later within the ongoing sound. In such paradigms, the timing of the acoustic change becomes an important experimental variable. Studies using this approach have shown that MMN amplitude depends not only on the nature of the deviance but also on when the deviance occurs relative to stimulus onset (Grimm et al., 2006; Grimm & Schröger, 2005; Timm et al., 2011; Weise et al., 2023). This effect is commonly referred to as the temporal distance effect or temporal position effect (Weise et al., 2023).

Early evidence for this effect came from studies using long or internally structured non-speech sounds. Schröger et al. (1992) used short complex sound patterns rather than single tones. Each stimulus was a pattern of eight consecutive 50-ms segments, each with a different frequency (720, 500, 638, 1040, 1175, 565, 815, and 920 Hz). Thus, the whole sound lasted about 400 ms. In the standard pattern, the same sequence of eight frequencies was repeated many times as participants performed a visual search task while ignoring the sounds. In the deviant pattern, only the segment at position 4 or at position 6 within that eight-part sequence was increased by 20% in frequency, while the rest of the pattern remained the same. The authors reported that this single within-pattern frequency change elicited an MMN, showing that the auditory system automatically encoded the internal spectro-temporal structure of the sound pattern even when attention was directed away from it. They also reported that a larger, more easily discriminable frequency change elicited a larger MMN than a smaller, less discriminable one (Schröger et al., 1992).

Grimm and Schröger (2005) used 440-Hz sine-wave standards of 1000 ms duration and created two types of deviants. One was a duration deviant, in which the tone was shortened from 1000 to 600 ms. The other was a transient frequency-modulation deviant, in which the tone changed from 440 to 480 Hz for 50 ms and then returned to 440 Hz. This brief frequency modulation could occur at one of several temporal positions within the long tone (at 100, 200, 300, 400, 500, 600, 700, 800, or 900 ms after sound onset). They found that MMN amplitude was clearly affected by the timing of the internal change. Earlier deviations elicited larger MMNs, whereas later deviations elicited smaller or no MMNs. MMN was absent in the ignore condition when frequency modulations occurred 400 ms after sound onset or later. Grimm et al. (2006) compared two types of within-stimulus deviants: duration deviants and frequency-modulation deviants. They used a passive oddball design with 440-Hz pure tones presented binaurally. In separate blocks, the standard sounds were 167, 333, 500, or 667 ms long. In the duration condition,

the deviant was a shortening of the sound by 40%, yielding deviant durations of 100, 200, 300, or 400 ms, respectively. In the frequency-modulation condition, the standard and deviant had the same overall duration, but the deviant contained a brief 50-ms frequency glide, during which the tone increased from 440 to 480 Hz over 25 ms and then returned to 440 Hz over the next 25 ms. The onset of this modulation was again placed at 100, 200, 300, or 400 ms after sound onset. This design allowed the authors to compare whether deviance detection depended only on the time of occurrence within the sound or also on the type of feature that changed. They found that MMN was present for both deviant types when the critical change occurred at 100, 200, or 300 ms, but it was no longer reliable at 400 ms. For the frequency-modulation deviants in particular, MMN was strong at the early positions, smaller by 300 ms, and absent at 400 ms. Based on this pattern, they concluded that the initial roughly 300 ms of a sound make the strongest contribution to automatic auditory deviance detection (Grimm et al., 2006; Grimm & Schröger, 2005).

Later studies reached similar conclusions. Timm et al. (2011) further examined within-stimulus frequency deviants by testing both the presence and the absence of a brief frequency modulation. They used 550-ms, 440-Hz sine waves that either contained a 50-ms frequency modulation (in which the frequency rose from 440 to 480 Hz and then returned to 440 Hz) or remained unmodulated. The modulation occurred either 100 or 400 ms after sound onset. In one set of conditions, the standard tone was unmodulated, and the deviant contained the modulation. In the other set, the standard contained the modulation, and the deviant omitted it. A clear MMN was found only when the deviant contained an early modulation at 100 ms. No reliable MMN was observed for the late modulation or for the conditions in which the deviant was defined by the absence of the modulation. This suggests that automatic deviance detection was sensitive not only to when the critical event occurred within the sound, but also to whether that event involved the addition of a transient spectral change (Timm et al., 2011). Hoonhorst et al. (2012) used tones that all lasted 500 ms. One stimulus was a fixed 1000-Hz tone that remained constant throughout the 500 ms. The other stimulus contained a 100-ms upward frequency sweep within the tone. In this sweep stimulus, the tone first remained at 1000 Hz, then changed gradually over 100 ms to 1100 Hz, and then stayed at 1100 Hz for the rest of the stimulus. The sweep began either 100 ms or 280 ms after the sound onset. Thus, in the early-sweep stimulus, the first 100 ms were steady at 1000 Hz, the next 100 ms contained the sweep, and the final 300 ms were steady at 1100 Hz. In the late-sweep stimulus, the first 280 ms were steady at 1000 Hz, the next 100 ms contained the sweep,

and the final 120 ms were steady at 1100 Hz. For each sweep onset time, the authors used two oddball arrangements. In the cued condition, the standard was the fixed 1000-Hz tone, and the deviant was the sweep tone. In this case, the deviant contained an explicit acoustic event, namely the onset of the frequency sweep. In the cueless condition, the roles were reversed: the sweep tone was the standard, and the fixed 1000-Hz tone was the deviant. Here, the deviant was defined by the absence of the expected sweep rather than by the presence of a new acoustic cue. MMNs were larger for cued than for cueless deviants, but they were reduced when the critical change occurred later in the tone. In the late cueless condition, the MMN disappeared, even though behavioural discrimination remained at ceiling. This showed that the reduction in MMN for late within-stimulus change could not be explained simply by poor conscious detectability (Hoonhorst et al., 2012).

One influential interpretation of these findings is based on the TWI. According to this account, sound features are extracted and combined into a single perceptual representation within a short period after sound onset. This representation is formed in a limited-capacity sensory memory system, so the earliest parts of a sound contribute more strongly to the final auditory object than later parts (Cowan, 1984; Grimm & Schröger, 2005; Näätänen & Winkler, 1999). Näätänen and Winkler (1999) proposed that this integration process is constrained by a brief temporal window. Weise et al. (2023) explained that auditory edges, especially sound onsets, open an integration window of roughly 350 ms. Within this window, the sound's internal dynamics are integrated into a representation. Information occurring beyond that window contributes less to the same representation unless another event creates a new segment. This framework explains why a late deviance may be behaviourally detectable but still fail to elicit a strong MMN: it falls outside the main integration window for automatic auditory object formation (Näätänen & Winkler, 1999; Weise et al., 2023).

An important question, however, is whether all internal transients can reset auditory integration. Weise et al. (2007) addressed this question using long 650-ms, 440-Hz pure tones. In one condition, the tone was continuous from onset to offset. In another condition, the same tone contained a brief 25-ms silent gap inserted 350 ms after sound onset. In both conditions, the deviant was a short frequency modulation placed near the end of the tone, between 550 and 600 ms after onset. During this 50-ms interval, the frequency increased from 440 to 480 Hz over 25 ms and then decreased back to 440 Hz over the next 25 ms. Thus, the late deviance was identical in both

conditions. The only difference was whether the tone had contained an earlier internal transient. When the tone was continuous, this late deviance did not elicit an MMN. When the tone contained the earlier gap, the same late deviance did elicit an MMN. They, therefore, argued that the internal transient segmented the long tone into smaller parts and effectively initiated a new integration window for the later portion of the sound. In a second experiment, they replaced the silent gap with a short noise segment and again observed MMN to the late deviance. This showed that the critical factor was not silence itself, but the presence of an internal transient that restructured the ongoing sound (Weise et al., 2007). Weise et al. (2011) further examined the segmentation account using long 900-ms, 440-Hz pure tones that contained a brief 20-ms silent gap at 440–460 ms after sound onset. This gap divided each tone into an initial and terminal segment. Standard tones contained only the gap, whereas deviant tones contained two brief 50-ms frequency modulations in which the frequency rose from 440 to 540 Hz and then returned to 440 Hz. In the Initial condition, both modulations occurred before the gap, at 50 and 300 ms after sound onset. In the Terminal condition, both occurred after the gap, at 550 and 800 ms after sound onset, corresponding to 90 and 340 ms after the onset of the terminal segment. In the Split condition, one modulation occurred late in the initial segment and the other early in the terminal segment. MMN was larger for deviances that occurred early within a segment and smaller for those that occurred late within a segment, regardless of whether the segment was initial or terminal. These findings supported the idea that auditory transients can parse unattended sounds into smaller units, allowing later changes to be represented automatically (Weise et al., 2011).

At the same time, not all gaps appear to function as strong reset cues. Yabe et al. (2001) used short 170-ms sound patterns made of five consecutive 1000-Hz tone segments. In the standard pattern, all five segments were present. In the deviant patterns, one internal segment was missing and was replaced by silence. The missing segment could occur at 26–62 ms, 62–98 ms, 98–134 ms, or 134–170 ms after sound onset. Therefore, the deviance was defined by where the silent interval occurred within the sound. They found that when the missing segment occurred later in the pattern, the MMNm was smaller and its peak was later. This suggested that automatic discriminative sensitivity becomes weaker toward the end of the TWI (Yabe et al., 2001). In later work, Yabe et al. (2005) used a short complex sound of about 170 ms made of consecutive 1000-Hz tone segments. They created a full pattern, an early-omission pattern in which the segment from 20 to 50 ms was missing, and a late-omission pattern in which the segment from 110 to 140

ms was missing. These patterns were presented in four oddball arrangements, with the roles of standard and deviant reversed across blocks. When the omission pattern served as the deviant, the MMNm latency difference between early and late omissions more closely matched the actual temporal difference. However, when the full pattern served as the deviant and the omission pattern served as the standard, the latency difference became much smaller. They also found a significant amplitude effect across conditions. In particular, MMNm amplitude was larger in the early-in-full condition than in the full-in-early condition, and the smallest response was observed for the full-in-late condition. These findings suggested that the temporal distance between early and late events may be represented as shorter in auditory sensory memory than in real time, and that later omitted information is represented more weakly (Yabe et al., 2005). Tamakoshi et al. (2016) used 176-ms complex sounds made of eight consecutive 1000-Hz tone segments with 2-ms rise and fall times. The full stimulus contained all eight segments. In the early-gap and late-gap stimuli, the second or fifth segment, respectively, was omitted and replaced by silence. In the early-filled and late-filled stimuli, the same segment positions were occupied not by silence but by a weak tone segment presented at one-quarter intensity. These stimuli were presented in oddball sequences with 85% standards and 15% deviants, and the roles of standard and deviant were reversed across blocks. MMN latency showed the same asymmetrical pattern for both gap and filled conditions. When the full stimulus served as the standard, MMN occurred earlier for early than for late changes, whereas this difference was reduced when the full stimulus served as the deviant. MMN amplitude was also larger for early than for late changes only when the full stimulus was the standard (Tamakoshi et al., 2016). Thus, these findings do not support a simple view in which every gap automatically resets integration.

Taken together, the literature shows that automatic deviance detection within long sounds is strongly constrained by the timing of the internal change. Earlier changes usually elicit larger MMNs than later ones. For relatively simple long sounds, this sensitivity often drops markedly after about 300 to 400 ms. However, this temporal constraint can be modified when the sound contains internal transients that segment it into smaller units. The resulting picture is therefore not that late change is inherently undetectable, but that the auditory system does not treat all parts of a long sound equally when building an automatic representation. This framework is directly relevant to studies of gap detection, because it predicts that the electrophysiological response to a gap should depend not only on gap duration, but also on when the gap occurs within the sound.

2.4.3 Development of MMN to gap-inserted stimuli in typically developing children

The findings discussed above suggest that the processing of internal gaps depends on how the auditory system represents ongoing sounds within the TWI. An important next question is whether MMN responses to gap-inserted stimuli can be reliably measured in TD children, and how these responses change with age.

Only a small number of studies have examined MMN to gap deviants in children, and most have used multi-feature paradigms rather than a single-gap-deviant design. In these paradigms, the gap deviant is presented together with other deviant types, such as changes in frequency, duration, or intensity. This approach shortens the recording time and is therefore practical for pediatric testing.

Petermann et al. (2009) recorded MMN from 16 TD children aged 5 to 7 years using a multi-deviant design that included frequency, duration, intensity, and gap deviants. Their study showed that the multi-deviant paradigm could be successfully applied in children and that the resulting MMN responses were distributed mainly over frontocentral scalp areas. They also emphasized that this type of design allows objective analysis of several MMN features within a relatively short test session (Petermann et al., 2009).

Partanen et al. (2013) extended this approach to speech-like material. They studied 30 children aged 4 to 12 years, including both preschool and school-age children, and used a multi-feature paradigm based on the pseudoword [tatata]. The deviant changes were introduced in the middle syllable and included vowel duration, fundamental frequency, gap, intensity, and vowel identity. They found that gap and vowel-duration changes elicited the largest frontocentral MMN responses, and these two deviant types also produced MMNs in most individual participants (Partanen et al., 2013).

In a longitudinal study by Putkinen et al. (2014), MMN and P3a responses were recorded from children at 7, 9, 11, and 13 years of age. Basic auditory processing was assessed with a multi-feature paradigm that included frequency, duration, intensity, location, and gap deviants. The results showed that MMN amplitudes to frequency, gap, and intensity deviants increased with age, whereas no comparable age-related increase was found for duration deviants. This suggests that neural discrimination of some basic sound changes, including silent-gap changes, continues to mature during school age (Putkinen et al., 2014).

More recently, Rapaport et al. (2023) used pediatric Magnetoencephalography and compared 18 younger children (mean age of 4.1 years) and 19 older children (mean age of 6.2 years). Their passive multi-feature oddball paradigm used harmonic tones as standards, and the deviants differed in frequency, intensity, duration, or by containing a silent gap in the middle of the tone. The gap deviant was created by removing 7 ms from the middle of the stimulus, including 1-ms fall and rise times. The older children showed larger mismatch-field responses than the younger children, especially in the right inferior frontal gyrus, whereas no age-group differences were found in the right primary auditory cortex or right superior temporal gyrus. These findings support the idea that predictive auditory processing becomes stronger with development and that frontal involvement increases with age (Rapaport et al., 2023).

Although this subsection focuses on school-age children, earlier work also showed that gap-inserted deviants can elicit MMN even in infancy. Trainor et al. (2001) reported MMN in 6- to 7-month-old infants using stimuli with silent gaps of 4, 8, or 12 ms. They suggested that at least some aspects of temporal resolution are already measurable very early in life (Trainor et al., 2001).

In general, these studies show that gap-inserted stimuli can reliably evoke MMN in TD children. They also suggest that the response changes with development. In particular, older children tend to show larger MMRs, and frontal regions appear to contribute more strongly with age. At the same time, the available literature is still limited. Most child studies have used multi-feature paradigms, and only a few have focused specifically on gap deviants. Thus, while gap-based MMN appears feasible in children, more work is still needed to clarify how age, stimulus context, and paradigm design influence the response.

2.4.4 MMN responses in children with APD: evidence from gap and speech paradigms

Although gap-inserted paradigms can elicit MMN reliably in TD children, much less is known about how children with APD respond to the same type of deviance. Only a small number of studies have examined MMN in children with APD, and the gap-specific literature is especially limited. Among them, Bauer et al. (2009) compared 32 children with APD and 13 TD controls with an age range of 5 to 7 years old using a multi-deviant MMN design. The paradigm included three types of sound deviants, one of which was a gap deviant. At the group level, the incidence of MMN was higher in the control group. The control group also showed earlier peak latencies at

frontal, central, and temporal sites. In addition, the area under the curve showed an asymmetric pattern in the APD group, with a tendency toward left-hemispheric dominance. These findings suggest that passive detection of gap-related change is less consistent, and possibly slower, in children with APD than in TD children (Bauer et al., 2009).

Hestvik et al. (2023) did not use a gap deviant. Instead, they examined a temporal speech contrast that is also relevant to rapid auditory processing. They studied 5 children with APD and 5 TD children, all between 7 and 10 years of age. The stimuli were synthesized /dæ/ and /tæ/ syllables that differed in VOT. For the /d/ tokens, VOT ranged from 0 to 20 ms in 5-ms steps. For the /t/ tokens, VOT ranged from 60 to 80 ms in 5-ms steps. The study compared two conditions. In the single-token condition, the same exact standard token was repeated. In the varying-standards condition, different tokens from the same speech category were used as standards. The TD group showed MMN in both conditions. In contrast, the APD group showed a reduced or absent MMN in the single-token condition, whereas the response in the varying-standards condition appeared more typical. The authors suggested that the children with APD had difficulty forming a stable short-term memory trace for the exact acoustic details of the repeated speech sound (Hestvik et al., 2023).

In summary, the available evidence suggests that children with APD often show reduced MMN consistency and prolonged latency, but the literature on gap-evoked MMN in this group remains very limited. In particular, the effect of gap position on MMN in children with APD has not yet been examined directly.

2.5 Summary and Research Gap

The literature shows that temporal resolution is an important auditory ability and that gap detection is commonly used to assess this ability. Behavioural studies show that gap detection can be affected by gap duration, gap position, stimulus characteristics, and task demands. These factors are especially important in children and in children with APD, because behavioural performance may be influenced not only by auditory sensitivity, but also by attention, memory, motivation, and response demands. Therefore, behavioural gap-detection results do not always provide a pure measure of auditory temporal processing.

Electrophysiological studies provide another way to examine gap detection. Silent gaps can elicit a DRN, and previous studies have shown that DRN amplitude increases as gap duration

increases. However, much less is known about whether the temporal position of the gap also affects the DRN. Studies using other types of auditory deviants have shown that automatic deviance detection becomes weaker when the change occurs later in the stimulus. This has been explained by the TWI. However, it is still unclear whether the same temporal position effect occurs for silent gaps, because gaps are abrupt acoustic changes and may initiate a new integration window.

This question has not been examined directly in TD children. It is also not known whether children with APD show reduced or less stable DRN responses to gaps occurring at different temporal positions. This is an important gap in the literature, because children with APD often show poorer behavioural gap detection, but it is unclear whether this reflects a bottom-up auditory temporal-processing deficit, top-down task demands, or both.

Chapter 3: The effect of the temporal position of a gap on its automatic detection

Authors:

Shaghayegh Omidvar¹, Kenneth Campbell², Claude Alain^{3,4}, Steven Aiken⁵, Amineh Koravand^{1*}

Affiliations:

¹ School of Rehabilitation Sciences, University of Ottawa, Ottawa, Canada,

² School of Psychology, University of Ottawa, Ottawa, Canada

³ Department of Psychology, University of Toronto, Toronto, Ontario, Canada

⁴ Rotman Research Institute, Baycrest Academy for Research and Education, Toronto, Ontario, Canada

⁵ School of Communication Sciences and Disorders, Dalhousie University, Halifax, Nova Scotia, Canada

***Corresponding address:**

Faculty of Health Sciences, University of Ottawa, 200 Lees Ave, Ottawa, Canada, ON K1S 5S9

Abstract

The presentation of a rarely occurring auditory stimulus that contains a silent interval (or gap) among a sequence of frequently occurring stimuli that do not contain a gap can elicit a negative-going event-related potential known as the Deviant-Related Negativity (DRN). This study investigated how the temporal position of the gap (i.e., temporal distance from stimulus onset) influences DRN amplitude using a novel multi-deviant paradigm. Seventeen young adults were presented with a sequence in which six gap deviants alternated with a standard stimulus. The standard stimulus was a 500-ms duration white noise burst. The deviants were constructed by inserting a gap in six different temporal positions, ranging from 15 to 400 ms after stimulus onset. In different conditions, gap duration was either 10 or 30 ms. Participants were also presented with single-deviant sequences in which the gap occurred at 100 or 300 ms. The amplitude of the DRN was larger for 30-ms than for 10-ms duration gaps; however, it was much reduced for gaps occurring at 300 or 400 ms after stimulus onset. There was no significant difference in DRN amplitude between the multi- and single-deviant paradigms. These results confirm the hypothesis that auditory integration occurs within a limited temporal window of less than 300 ms. Moreover, the multi-deviant paradigm successfully elicited measurable DRNs across multiple gap positions within a single recording sequence, highlighting its potential as a time-efficient tool for assessing temporal processing.

Keywords: Mismatch negativity (MMN), deviant-related negativity (DRN), gap detection, temporal distance effect

3.1 Introduction

Temporal resolution is a key component of central auditory processing and plays a crucial role in speech perception, particularly in challenging listening environments. It refers to the ability to detect and resolve rapid changes in acoustic signals over time (Moore, 2008; Shinn et al., 2009). These changes may include brief variations in amplitude, stimulus onset and offset, or short silent intervals within an ongoing sound. Temporal resolution is therefore relevant to the processing of rapidly changing acoustic information in speech. In fluctuating background noise, it may also help listeners take advantage of brief reductions in masker energy to access portions of the target speech (Alamatsaz et al., 2024; Bernstein and Grant, 2009). Temporal resolution is often studied using gap-detection methods, where a listener is asked to press a button when they hear a silent period (or “gap”) within a continuous or long-duration auditory stimulus. Normal-hearing young adults can detect gaps as short as 3 to 5 ms when inserted into moderate-intensity white noise (Musiek et al., 2005; Giannela Samelli, and Schochat, 2008). Standardized clinical behavioural gap detection tests typically insert gaps either in the middle of a short-duration sound or well after the onset of a long-duration sound, but rarely near the sound’s onset or offset (Phillips, 1998). A series of behavioural studies has indicated that gap detection is poorer when the gap is inserted near the beginning or near the end of a stimulus but better when it is inserted in the middle of a stimulus (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999). For example, Gay et al. (2020) assessed gap-detection thresholds using 200-ms bandpass-noise bursts (200–6000 Hz), with gaps positioned 10 to 50 ms after stimulus onset. They found that thresholds were highest for early-position gaps. Snell and Hu (1999) inserted a 10- to 130-ms gap into a 150-ms duration, 6-kHz, low-pass noise burst. The gap duration needed to be longer when the gap occurred near the beginning or end of the noise burst, and shorter when it occurred near the centre. This temporal asymmetry in gap detectability may be partly explained by neural refractoriness and desynchronization in phasic ON-type units (i.e., neurons that respond to stimulus onset) following the onset of the leading noise burst, as well as an offset masking effect, whereby neural responses to the end of the gap are masked by responses to the trailing noise offset (Walton et al., 1997). A limitation of using behavioural assessments of gap detection abilities is that they require the individual to actively engage in the testing session for relatively long periods. Sustaining attention for long periods may not be possible for children, older adults, or those with neurological or psychiatric disorders. Moreover, performance on behavioural testing is often affected by non-sensory factors such as

motivation, attention, and response bias (willingness to respond “yes” to near-threshold gaps) (Silman et al., 2000; Gyldenkerne et al., 2014).

Scalp recording of event-related potentials (ERPs) provides a non-invasive means of examining auditory processing by measuring changes in the brain’s electrical activity that are time-locked to sensory or cognitive events (Näätänen & Picton, 1987). (Avivi-Reich et al., under revision; Bertoli et al., 2001; Bertoli et al., 2002; Desjardins et al., 1999; Duda et al., 2020, 2025; Duda-Milloy et al., 2019; Heinrich et al., 2004; Ross et al., 2010; Tamakoshi et al., 2016; Todd et al., 2011; Uther et al., 2003; Yabe et al., 2005). ERPs are the changes in the electrical activity of the brain (the EEG) that are elicited by a physical stimulus or a cognitive event. ERPs consist of a series of negative- and positive-going components thought to reflect different aspects of processing. In auditory research, these responses can be used to examine neural processing of different acoustic features, including temporal characteristics of sound. A component called the Mismatch Negativity (MMN) has been recently employed in the study of gap detection. The MMN is typically recorded using a so-called “oddball” paradigm. The participant is presented with a frequently occurring “standard” stimulus. At rare (or odd) and unpredictable times, a feature of the standard is changed to form a “deviant.” The onset of any auditory stimulus will elicit an obligatory response, labelled the “N1,” peaking at about 100 ms, followed by a later positivity, the P2, at about 180 ms. Both the standard and deviant thus elicit the N1-P2 complex. The deviant also elicits an additional negative component, the MMN (Näätänen, 1992). The MMN peaks from 100 to 200 ms, depending on the extent to which the deviant differs from the standard. In the classic Näätänen et al. (2004) model, the change detection system is claimed to compare the encoded features of an incoming stimulus with those in sensory memory. If they match, the sensory memory is strengthened, but further processing is deemed redundant and ceases. If they fail to match, an acoustic change is detected, and further processing is warranted; the output of the change detection system is reflected in the MMN. More recent descriptions (e.g., Winkler, 2007; Winkler et al., 2009) maintain that a rule-based predictive model of the preceding pattern in the auditory sequence is formed. It is a violation of this prediction that elicits the MMN. A large advantage of the use of the MMN is that it still occurs in the absence of attention or indeed regardless of what the participant “is doing” (Muller-Gass et al., 2006; Näätänen et al. 1993; Sussman, 2007, 2017). The MMN is thus often recorded “passively” while the participant is engaged in another “diversion.”

Prior MMN studies of gap detection have often used an oddball paradigm in which participants are presented with a frequently occurring “standard” stimulus that lacks a gap and a “deviant” stimulus that contains a gap. The gap deviant elicits an MMN response that increases with gap duration (Bertoli et al., 2001; Desjardins et al., 1999; Uther et al., 2003). While most studies categorize the negativity triggered by the infrequent gap deviant as an MMN, it may not be a “true” MMN (Campbell and MacDonald, 2011; Palmer and Musiek, 2013; Pratt et al., 2007). The onset of the gap also elicits an N1 wave. The occurrence of the gap deviant additionally signals a change from the standard (not containing a gap). It thus elicits an MMN. Therefore, the gap elicits a composite negativity, the N1+MMN (Duda-Millooy et al., 2019). This composite negativity has been labelled as a deviant-related negativity (DRN) (Woldorff et al., 1998). The label “DRN” will therefore be used in the present study¹. In most MMN studies, the deviant stimulus differs from the standard stimulus from stimulus onset, and this difference is maintained throughout the stimulus duration (Näätänen et al., 2007). In some studies, however, the change in the standard stimulus is relatively brief and does not persist throughout the stimulus. For example, the standard duration might be 400 ms. The deviant might also last 400 ms, but a brief 50-ms frequency change might occur at 100 ms or at 300 ms. The time of occurrence of stimulus changes relative to the standard thus varies. Previous studies have examined the effect of the time when stimulus change occurs (Alain et al., 1994; Grimm & Schröger, 2005; Hoonhorst et al., 2012; Näätänen et al., 2004; Schröger et al., 1992; Tamakoshi et al., 2016; Weise et al., 2007, 2010, 2011; Timm et al., 2011; Yabe et al., 2001; Yabe et al., 2005) and have reported a gradual decrease in MMN amplitude as change onset time increases. Näätänen et al. (2004) used duration deviants in which the deviant tone was half the duration of the standard (standard/deviant pairs of 200/100, 400/200, 800/400, and 1600/800 ms). They found that when the deviant occurred earlier in time (e.g., a 100-ms change), the MMN amplitude was larger. As the onset of change was delayed (e.g., 800 ms), the MMN amplitude gradually decreased. This pattern was consistent across experiments, showing that MMN amplitude diminishes as the temporal distance to the change increases. The effect of the timing of stimulus change has been called the temporal distance (or “position”) effect (see the review by Weise et al., 2023). This effect has been consistently observed for duration deviants across studies (Grimm et al., 2004; 2006, Grimm & Schröger, 2005). The temporal distance effect

¹ When reference is made to previous studies, their naming convention will, however, be used (e.g., MMN instead of DRN).

is not unique to duration deviants. Other studies have examined the effects of temporal distance on other features. Schröger et al. (1992) presented a long-duration standard stimulus. A brief 50-ms frequency modulation at 150 ms elicited a larger MMN than one at 250 ms. Grimm and Schröger (2005) varied the onset time of a frequency-modulated standard stimulus, with deviance occurring from 100 to 900 ms after stimulus onset. Importantly, only frequency modulations within the first 300 ms of the sounds elicited a distinctive MMN. The MMN was much reduced when the change occurred at 400 ms. The MMN was absent to changes occurring from 500 to 900 ms after stimulus onset. Other studies have also reported decreased MMN to frequency modulations after 300 ms (Hoonhorst et al., 2012; Timm et al., 2011; Weise et al., 2007, 2010, 2011). Participants were often also asked to detect the occurrence of the different deviants. They were able to do so with ease. Deviants that occurred after 300 ms were detected equally as well as those that occurred earlier (Grimm et al., 2006; Hoonhorst et al., 2012; Timm et al., 2011; Weise et al., 2007, 2010). Therefore, this MMN reduction cannot be explained by an inability to perceive change after 300 ms.

A temporal integration theory has been proposed to explain this effect (Näätänen and Winkler, 1999; see review by Weise et al., 2023). The initial analysis and extraction of sound features (e.g., intensity, frequency, and location) enable their integration into a single perceptual unit, related to the formation of an auditory object (Arnott and Alain, 2002; Bizley and Cohen, 2013). A temporal window of integration (TWI) begins at sound onset, forming a sound representation within sensory memory. A major constraint is that this integration is confined to a 150 to 300-ms period. Early portions of a sound, therefore, appear to contribute more to its representation than later parts. It has been suggested that this temporal limit results from the restricted capacity of the auditory store or the very short phase of auditory sensory memory (Cowan, 1984; Näätänen & Winkler, 1999; Grimm & Schröger, 2005).

There may be an exception to the temporal distance effect, the onset time of a gap in a deviant stimulus. This is because abrupt transient changes within these stimuli are thought to initiate new integration windows, effectively creating segments within the long-duration sound (Weise et al., 2007). As such, the temporal constraints of sound representation may be overcome. Weise et al. (2023) note that a stimulus that initiates a transient change will elicit an N1-P2. In the frequency deviance studies discussed earlier in which a strong temporal distance effect was observed, the effects of a sudden transient change were avoided by gradually modulating the

frequency change. For example, Grimm and Schröger (2005) introduced a 50-ms frequency modulation into a 1000-ms, 440 Hz standard tone by increasing the frequency to 480 Hz for 25 ms, then returning it to 440 Hz over the next 25 ms. In the Weise et al. (2007) study, a frequency modulation of the standard occurred at 550 ms. This deviant did not elicit an MMN, presumably because the frequency change occurred after the integration period. However, when a 25-ms gap was inserted at 350 ms, the same frequency change at 550 ms, which had previously not elicited an MMN, now did elicit one. This was explained by the fact that the presentation of a transient-the gap- initiated a new integration window, allowing for the automatic detection of frequency change. The gap created a transient change, indexed by the N1-P2. In a related study, Yabe et al. (2001) presented 170-ms, 1000 Hz tones composed of five 36-ms segments, each with a 4-ms rise-and-fall time. Occasionally, the 2nd, 3rd, 4th or 5th tones were omitted (i.e., the segment was heard as a silent period or a gap). If a transient gap initiated a new integration window, then each gap should have elicited an N1-P2 in addition to an MMN, regardless of its time of onset. This was not the case. The gap occurring in the 5th position, beginning at 134 ms, elicited only a very small MMN amplitude. The gap, however, did not elicit an N1-P2. Weise et al. (2023) suggested that the 4-ms rise-and-fall time may not have been sufficient to initiate a new integration period. Tamakoshi et al. (2016), however, replicated the Yabe et al. (2001) results using a faster 2-ms rise-and-fall time. Nevertheless, this gap did not elicit an N1-P2 and probably did not initiate a new integration window. These studies show that not all gaps reliably produce a perceptually salient transient capable of resetting auditory integration.

These findings directly informed the design of the present study. We aimed to investigate how gap onset time and gap duration influence the DRN, and whether the effect of gap onset time differs as a function of gap duration. The present study used a multi-deviant paradigm (Näätänen et al., 2004). We also examined whether a multi-deviant paradigm could be used to study the temporal position of gap deviants by comparing DRNs obtained with the multi-deviant paradigm with those obtained using traditional single-deviant oddball sequences. This paradigm is time-efficient, allowing multiple deviants to be presented in a single sequence (Näätänen et al., 2004). Duda-Milloy et al. (2019) modified the original Näätänen et al. (2004) paradigm for use with gap deviants. The standard stimulus was a 200-ms white noise burst. A gap deviant was created by inserting a silent interval in the center of the standard. Six different deviants were used, varying in duration from 2 to 40 ms. The DRN amplitude was larger for the longer-duration gaps. Follow-up

studies have replicated these results (Duda et al., 2020, 2025). The various deviants shared the same feature, introducing a silent period in the centre of the standard. This procedure differs from that used in the original Näätänen et al. (2004) multi-feature paradigm, in which each deviant is created by changing a different feature of the standard. In the study by Duda-Milloy et al. (2019), although the duration of the various gaps varied, the actual physical features of the deviants (a silent period) did not. For this reason, they compared their multi-deviant results to those obtained when the various deviants were also presented in separate oddball sequences. The DRNs' amplitudes did not significantly differ between the oddball and multi-deviant paradigms. In the present study, a brief silent gap, either 10 or 30 ms, occurred at six different onset times, varying from 15 to 400 ms after the onset of the deviant stimulus. A concern in the present study was that while the onset of gap deviants occurred at different times, all deviants were identical and, unlike the gaps used in the Duda-Milloy study, their duration did not vary within each multi-deviant sequence. It was unknown whether the multi-deviant paradigm could be used in such a situation. For this reason, the results of separate oddball sequences were compared to those obtained in a single multi-deviant sequence.

Statement of Hypotheses: When the frequency of a long-duration deviant is gradually modulated, and this change occurs after 300 ms, the amplitude of the MMN/DRN is much reduced or absent. Weise et al. (2023) hypothesized that this temporal position effect only applies to changes that occur relatively slowly (gradual modulation). The temporal position effect may not apply to very abrupt transient changes. A silent gap inserted into a deviant stimulus has an abrupt onset and offset. (1). If the Weise et al hypothesis is true, the DRNs elicited by gap deviants should not be subject to the temporal position effect. It would therefore be expected that a robust DRN will be elicited regardless of the time of occurrence of the gap deviant. (2). Alternatively, if the Weise et al hypothesis is not true, then it would be expected that the temporal position effect will apply to any deviant, regardless, even if its onset is abrupt. Late-occurring gap deviants should therefore elicit very small or absent DRNs. In addition, it is not known if the temporal distance effect will interact with the duration of the gap. For this reason, in different conditions, the gap duration was either 10 or 30 ms. Finally, based on Duda-Milloy et al. (2019), DRN amplitudes obtained with the multi-deviant paradigm were expected to be comparable to those obtained with the single-deviant oddball paradigm.

3.2 Methods

3.2.1 Participants

A total of 17 adults (12 females) aged 22 to 37 years (mean = 29.6, Standard deviation [SD] = 5.5 years) volunteered to participate in the study. The sample size was deemed appropriate, assuming a power of 0.8, based on a power analysis of effect size within the Grimm and Schröger (2005) study, using their means and SDs for the MMN when a frequency modulation occurred at either 100 or 400 ms. Based on their effect size, a sample of 11 participants was deemed adequate. These estimates were calculated using G*Power software, version 3.1.9.7.28 (Faul et al., 2007). The sample size in the present study was comparable to that used in prior studies of the gap-elicited DRN (Duda-Milloy et al., 2019; Duda et al., 2020; Duda et al., 2025; Tavakoli et al., 2021).

Participants were recruited from English-speaking graduate students at the University of Ottawa. All participants were fluent in English, and many also spoke other languages. None of the participants reported a history of neurological or psychiatric disorders. All participants reported normal hearing. Normal hearing levels were verified using standard audiological procedures. Their hearing threshold did not exceed 15 dB HL at octave frequencies from 250 to 8000 Hz (ANSI, 1996). Middle ear function was also assessed to ensure normal tympanometric peak pressure (between -100 and +50 daPa) in both ears (Gelfand, 2016), using a tympanometer. Written informed consent was obtained prior to the beginning of the experiment. The University of Ottawa Research Ethics Board approved the study in accordance with the Canadian Tri-Council (Health, Natural, and Social Sciences) guidelines on ethical conduct involving humans.

3.2.2 Procedure and stimuli

Participants sat in a sound-attenuated and electrically shielded booth. They were instructed to watch a silent, subtitled video of their choice and to ignore the auditory stimuli. The auditory stimuli were therefore processed passively. Auditory stimuli were delivered binaurally via insert-earphones (E-A-RTONE 3A). **Multi-deviant paradigm.** The multi-deviant sequence consisted of the presentation of frequently occurring standards alternating with six different gap deviants. The standard stimulus was a broadband noise burst with a peak intensity of 80 dB SPL and a 500 ms duration, with instantaneous rise- and fall-times. The broadband noise ranged in frequency from near 0 Hz to 22 kHz. Sound intensity was calibrated using a Bruel and Kjaer Type 2235 sound level meter and a 2 cm³ coupler (GRAS RA0045-S1). Six different deviants were created by

inserting a silent interval (a “gap”) in the standard. The gap had an instantaneous onset and offset. Broadband noise made it less likely that spectral splatter produced by the gap could be detected (Brown and Nicholls, 1997). The gap occurred at either 15, 50, 100, 200, 300, or 400 ms after the onset of the deviant. The sequence lasted about 14 minutes, with 472 standards (including 10 initially presented) and 77 of each deviant. Because the standards and deviants alternated, the probability of occurrence of each stimulus type was 50%. However, the probability of occurrence of each of the six different gap deviants was 8.3%. The inter-stimulus interval (offset-to-onset) was 400 ms. The deviants were ordered pseudo-randomly such that in an array of 12 alternating standards and deviants, each of the six deviants was presented in random order (see Figure 3.1). The last deviant in the array and the first in the subsequent array could not be the same. Moreover, the same random order of presentation of deviants within an array could not occur a second time. Gap duration was either 10 or 30 ms in separate conditions. The two multi-deviant sequences were each repeated three times in random order. A brief rest period was provided between sequences. The timing and presentation of all stimuli were controlled by a computer running E-Prime® (Psychology Software Tools, version 2.0).

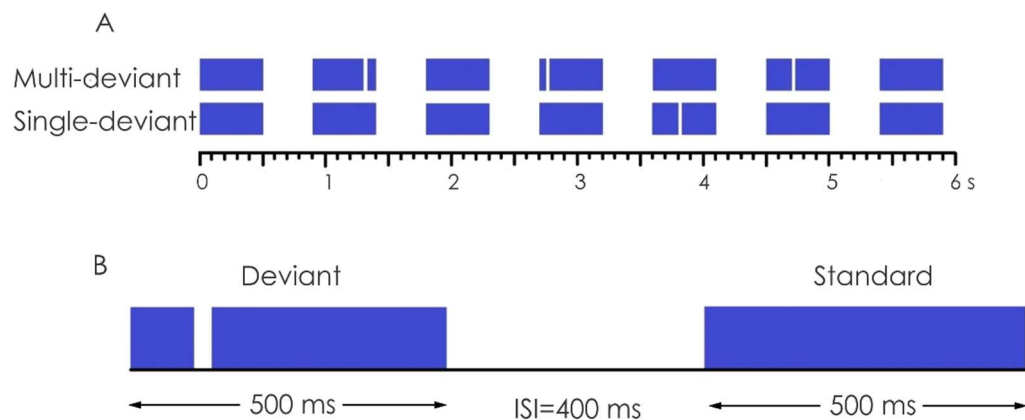


Figure 3.1 Multi-deviant and single-deviant sequences. A: In the multi-deviant sequence, the standard and deviant stimuli alternated. Three gap onset times are shown. In the single-deviant sequence, a gap was presented at either 100 or 300 ms, depending on the condition. B: Timing of stimulus presentation. For both the multi- and single-deviant sequences, the duration of all stimuli was 500 ms. The ISI was 400 ms.

Single deviant oddball paradigm. Two single-deviant (or oddball) paradigms were also run. The same standard used in the multi-deviant paradigm was presented on 91.7% of trials. Only a single deviant was presented in the sequence occurring on the remaining 8.3% of trials (Figure 3.1). The single deviant thus had the same probability of occurrence as each deviant in the multi-

deviant paradigm. The order of stimulus presentation was pseudorandomized, ensuring that at least three standards were presented between deviants. Again, ten standards were presented at the beginning of each sequence. Each sequence, therefore, included 857 standards and 77 deviants. Every single deviant sequence also lasted about 14 minutes. In different conditions, the gap occurred at either 100 or 300 ms. Only a single 10-ms gap condition was used in order to limit total testing time. The 100- and 300-ms gap onset time conditions were each presented three times. The six sequences were presented in random order. The duration of the EEG recording session was about three hours.

The order of presentation of the multi- and single-deviant conditions was counterbalanced across participants.

3.2.3 Active behavioural detection task

A separate condition was run in which the participant was asked to actively detect the occurrence of the deviant. In this condition, the standards (500-ms broadband white noise bursts) and the 10 and 30-ms duration deviants were presented in a single sequence. Stimuli were presented binaurally via insert earphones at a peak intensity of 80 dB SPL, as in the electrophysiological conditions. A silent gap was inserted at 15, 50, 100, 200, 300, or 400 ms after the onset of the deviant. The sequence thus included standards and 12 different deviants (2 gap durations $\times$ 6 different onset times). A total of 50 standards and 120 deviants were presented in random order. The stimuli were presented with a fixed 1500 ms ISI to allow participants time to push the left button of a mouse if they judged that a standard (no gap) had been presented and the right button if the deviant (containing a gap) had been presented. Hits (correctly detecting the gap deviant), correct rejections (correctly detecting the standard), false positives (detecting the standard as a gap deviant), and misses (detecting the gap deviant as a standard) were computed. Ten practice trials were presented to ensure the participant understood the instructions.

3.2.4 Electrophysiological recording and analyses

The EEG was recorded from 30 active silver-silver chloride electrodes attached to an electrode cap placed according to the International 10-10 system (Pivik et al., 1993). Electrodes were positioned on the midline and lateral areas of the frontal, central, parietal, temporal, and occipital regions. The reference electrode was located on the tip of the nose. Vertical eye movement and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of

the left eye. Inter-electrode impedance was below 25 k Ω . The continuous EEG activity was sampled at 500 Hz and stored on disk.

The data were subsequently analysed using Brain Products' Analyzer 2.3 software. The EEG and electrooculography (EOG) data were digitally filtered using a band-pass filter set at 0.3 to 20 Hz. The continuous data were visually inspected to identify channels contaminated by high-amplitude noise. These channels were replaced by interpolating the data of surrounding electrode sites (Perrin et al., 1989). A maximum of four channels was replaced by interpolation. Independent Component Analysis (ICA; Makeig et al., 1996), was used to identify eye movement and blink artifacts statistically independent of the EEG activity. These components were then subtracted from the raw EEG to minimize contamination from eye movements.

The continuous EEG was partitioned into 1000-ms epochs, beginning 100 ms before stimulus onset. The average of the 100-ms pre-stimulus period served as a baseline, from which ERP components were measured. Any epoch containing activity exceeding ± 100 μ V was then rejected from further analysis. Rejections based on this criterion were relatively low because the most common type of artifact, eye blinks and movements, had already been corrected. The single epochs were then sorted according to electrode site and stimulus type (standard, six deviants, two gap durations) and averaged.

3.2.5 Quantification and statistical analyses

The onset of standard and deviant stimuli elicited obligatory N1 and P2 ERP components. The gap within the deviant also elicited an additional negativity, the DRN, consisting of a composite N1+MMN. The onset of gaps occurring from 15 to 100 ms elicited a DRN that overlapped with the N1 and P2 elicited by the onset of the deviant stimulus. For this reason, the DRN was best observed in a difference wave computed by subtracting the standard from the deviant waveform. This subtraction process removes processing common to the standard and deviant (including the N1 and P2 elicited by the onset of both stimuli), leaving processing unique to the deviant (i.e., the processing of the gap inserted into the deviant). The amplitude of the DRN for each participant was quantified by averaging all data points within a ± 25 -ms window around the peak identified in the grand-average waveform.

The use of the difference wave, however, assumes that differences in the DRN across two gap durations, or two paradigm conditions, resulted from differential processing of only the

deviant. However, this assumption is only valid if the processing of standard stimuli does not vary across conditions. Therefore, a one-way analysis of variance (ANOVA) was used to compare the standard N1 amplitudes between the multi- and single-deviant conditions, as well as in the 10- and 30-ms conditions at both Fz and Cz.

For the multi-deviant paradigm, a 3-way ANOVA with repeated measures on gap duration (10, 30 ms), gap onset time (15, 50, 100, 200, 300, and 400 ms), and electrode site (left, midline, right). A separate ANOVA was run for the frontal and central regions of interests (ROIs). When the oddball sequences were run, only a 10-ms gap occurring at 100 or 300 ms was presented. The DRN results for these deviants were compared with those within the multi-deviant paradigm, but only for the 10-ms gap data occurring at 100 or 300 ms. A 3-way ANOVA with repeated measures on gap onset time (100, 300 ms), paradigm (single-, multi-deviant), and electrode site (left, midline, right) was run on these DRN data. Again, separate ANOVAs were run at frontal and central ROIs.

A problem with the ANOVA procedure is that it is used to determine whether the amplitude of the DRN differs significantly across conditions. The temporal position effect, however, predicts that a DRN may be absent for late-occurring stimulus changes. An ANOVA can determine if the amplitude of the DRN for these late occurring deviants is significantly different from that of earlier occurring deviants. It cannot, however, determine if the amplitude of the DRN is absent. To determine whether a deviant elicited a DRN, confidence intervals (CIs) were computed to assess whether the amplitude of the DRN was significantly below the zero-voltage pre-stimulus baseline (i.e., a negative-going deflection). The CIs were calculated using data from the ROI, including frontal (Fz, F3, F4) and central (C3, Cz, C4) ROIs, where the DRNs are typically largest. A DRN response was considered to be present only if both the maximum and minimum amplitude values were less than 0 (i.e., a negative value). Because the DRN was expected to be negative-going, a one-tailed test of significance ($p < .05$) was used for the CI testing.

3.3 Results

3.3.1 Electrophysiological data

3.3.1.1 Multi-deviant paradigm

3.3.1.1.1 Standard ERP

Figure 3.2 A. illustrates the standard waveforms at Fz and Cz in the 10- and 30-ms conditions. A small amplitude N1 deflection (a negative-going deflection) is apparent around 100 ms, followed by a larger P2 deflection (a positive-going deflection) at approximately 180 ms. A one-way ANOVA revealed no significant differences in N1 amplitude between the multi- and single-deviant conditions, or between the 10- and 30-ms conditions at Fz and Cz, $F < 1$. Similarly, no significant differences were found for the amplitude of the standard P2 at both Fz and Cz, $F < 1$.

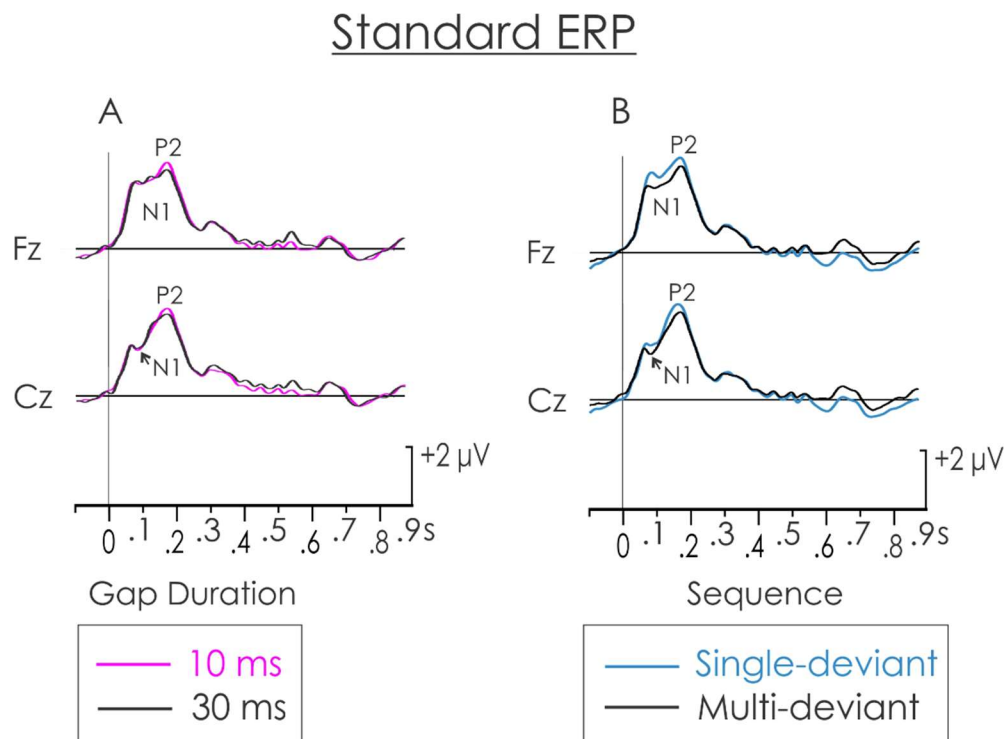


Figure 3.2. The grand average ERP waveforms following the presentation of the standard stimulus. A. The standard ERP is illustrated for the 10- and 30-ms gap duration conditions. The amplitude of N1 was very small because of the rapid rate of stimulus presentation. It was followed by a positivity, P2. Their amplitudes did not significantly differ between the 10- and 30-ms duration gap conditions. B. The standard ERP in the single- and multi-deviant conditions. Again, the amplitudes of N1 and P2 following the presentation of the standard did not differ significantly between the two paradigms.

3.3.1.1.2 Deviant-Related Negativity (DRN)

The grand averages of the deviant-standard difference waves for 10- and 30-ms gaps in the multi-deviant paradigm are shown in Figure 3.3. A fronto-central maximum DRN was elicited approximately 100–150 ms after gap onset.

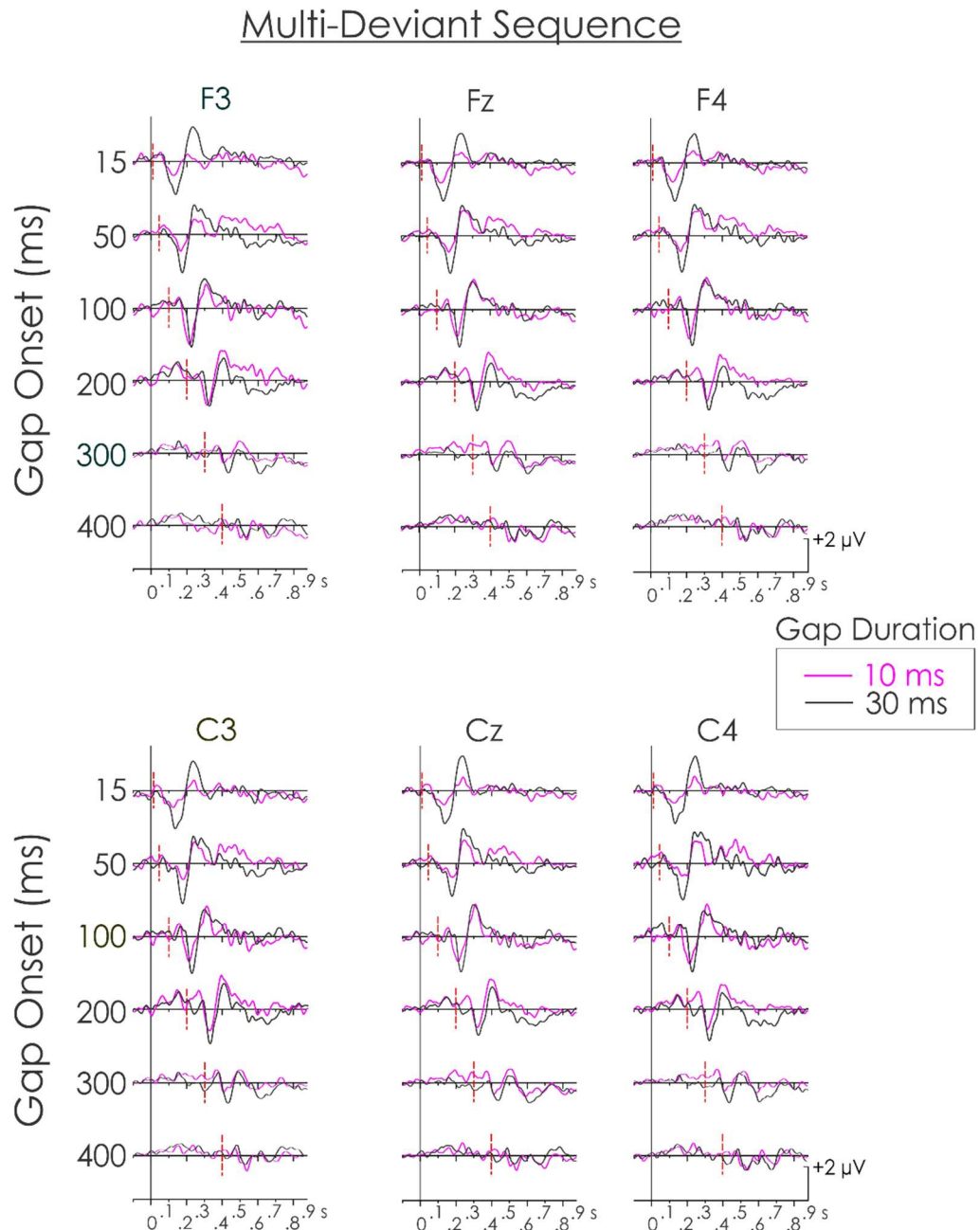


Figure 3.3 Grand averaged ERP deviant-standard difference waves in the multi-deviant condition as a function of gap duration and gap onset time. The onset of the deviant stimulus containing the gap is indicated by the solid vertical black line at 0 ms. The onset of the actual gap is indicated by the red dashed line. A DRN is apparent approximately 100–150 ms after gap onset. The DRN was significantly reduced when the gap duration was 10 ms. The DRN was much reduced when the gap occurred at 300 or 400 ms.

The amplitude of the DRN increased with increasing gap duration from 10 to 30 ms at frontal and central ROIs. The mean DRN data are presented in Figure 3.4 for the frontal and central ROIs. When the 30-ms duration gap was presented, the DRN gradually decreased as its onset time increased from 15 to 400 ms. When the 10-ms gap was presented, the amplitude of the DRN generally decreased as gap onset times increased, but the changes were not as consistent as those observed with the 30-ms duration gap.

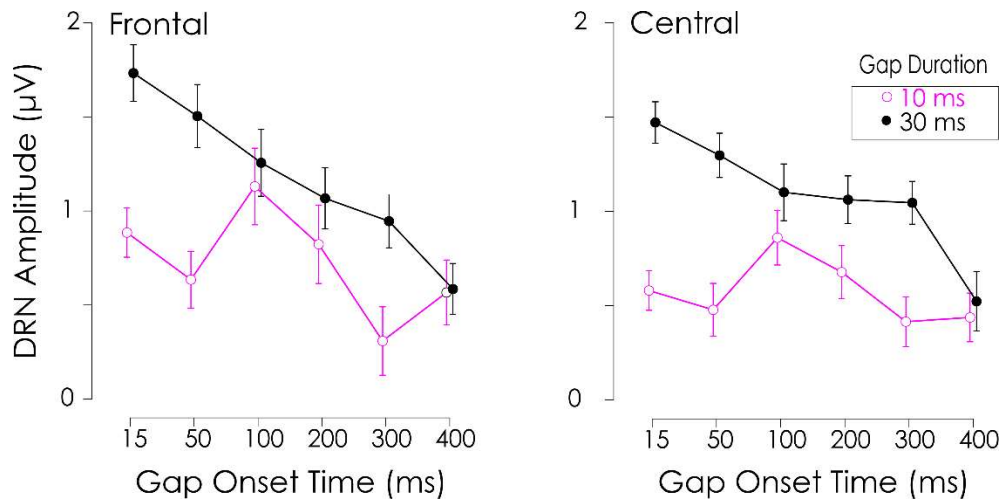


Figure 3.4 Mean amplitude of the DRN for the 10- and 30-ms duration gaps as a function of gap onset time. Error bars represent the standard error (SE) of the mean.

The ANOVA revealed a significant main effect of gap duration at the frontal ROI, $F(1,16) = 10.64$, $p = .005$, $\eta^2_p = .40$, and the central ROI, $F(1,16) = 12.83$, $p = .002$, $\eta^2_p = .44$, respectively. DRN amplitude was significantly larger following the presentation of the 30-ms than the 10-ms duration gap. The main effect of gap onset time was significant at the frontal ROI, $F(5,80) = 3.91$, $p = .003$, $\eta^2_p = .20$. The main effect of gap onset time was not significant at the central ROI, $F(5,80) = 1.58$. The interaction between gap onset time and electrode site was, however, significant ($F(10,160) = 2.12$, $p = 0.03$, $\eta^2_p = .12$). Simple main effects testing indicated that the main effect of gap onset time was significant at Cz ($p < .03$) but not at either C3 or C4 ($p = .32$ and $.41$, respectively). The interaction between gap duration and gap onset time was not significant at either frontal or central ROIs ($p = .14$ and $.30$, respectively).

Holm post hoc tests were performed on the frontal ROI data to determine DRN differences across the various gap onset times. The DRN was significantly larger when the gap occurred at 15 ms than at 400 ms ($p < .001$), whereas no significant differences were observed between other gap onset times. The Holm post hoc procedure, however, is very conservative. It reduces the likelihood of finding differences across comparisons. Its use would therefore be more likely to support the null hypothesis that predicts that the amplitude of the DRN to late occurring deviants will not be different from that of early occurring deviants. Previous studies, however, have found that the DRN significantly decreases in amplitude when gap onset times occur later (i.e., the null hypothesis is not supported). For this reason, multiple t-tests were run without correction for multiple comparisons. These results indicated that the DRN was significantly reduced for the 200-, 300-, and 400-ms onset times compared to the 15-ms onset time ($p < .05$). In addition, the DRN was significantly reduced for the 400-ms onset time compared to the 50- and 100-ms onset times. The DRN was also significantly reduced at the 300-ms onset compared to the 100-ms onset times ($p < .05$).

CI testing revealed a significant DRN for all deviants, except for the 10-ms gap occurring at 300 ms at the frontal ROI, and at 300 and 400 ms at the central ROI.

3.3.1.2 *Single-deviant paradigm*

3.3.1.2.1 Standard ERP

Figure 3.2 B. illustrates the standard ERPs in single and multi-deviant conditions. The N1 amplitude was small, whereas the P2 amplitude was relatively large. Their amplitudes did not significantly differ between these conditions at either Fz or Cz, $F < 1$ in both cases.

3.3.1.2.2 Deviant-Related Negativity (DRN)

Figure 3.5 illustrates the grand-average difference waveforms in the single- and multi-deviant conditions. In the single-deviant condition, the 10-ms duration gap occurred at 100 or 300 ms. These data were compared to the same 100- and 300-ms gap onset times during the multi-deviant condition. The DRN was larger in the single-deviant than in the multi-deviant condition. However, this difference was not statistically significant at either the frontal, $F(1,16) = 3.79$, $p = .07$, $\eta^2_p = .19$, or the central ROI, $F(1,16) = 3.36$, $p = .09$, $\eta^2_p = .17$. The main effect of gap onset time was significant at the frontal ROI, $F(1,16) = 10.98$, $p = .004$, $\eta^2_p = .41$, the amplitude of the

DRN being significantly larger when the gap occurred at 100 than at 300 ms. The DRN at the central ROI was also larger when it occurred at 100 than at 300 ms, but the difference was not significant, $F(1,16) = 2.65$, $p = .12$, $\eta^2_p = .14$. No interactions were significant, $F < 1$ in all cases at both frontal and central ROIs.

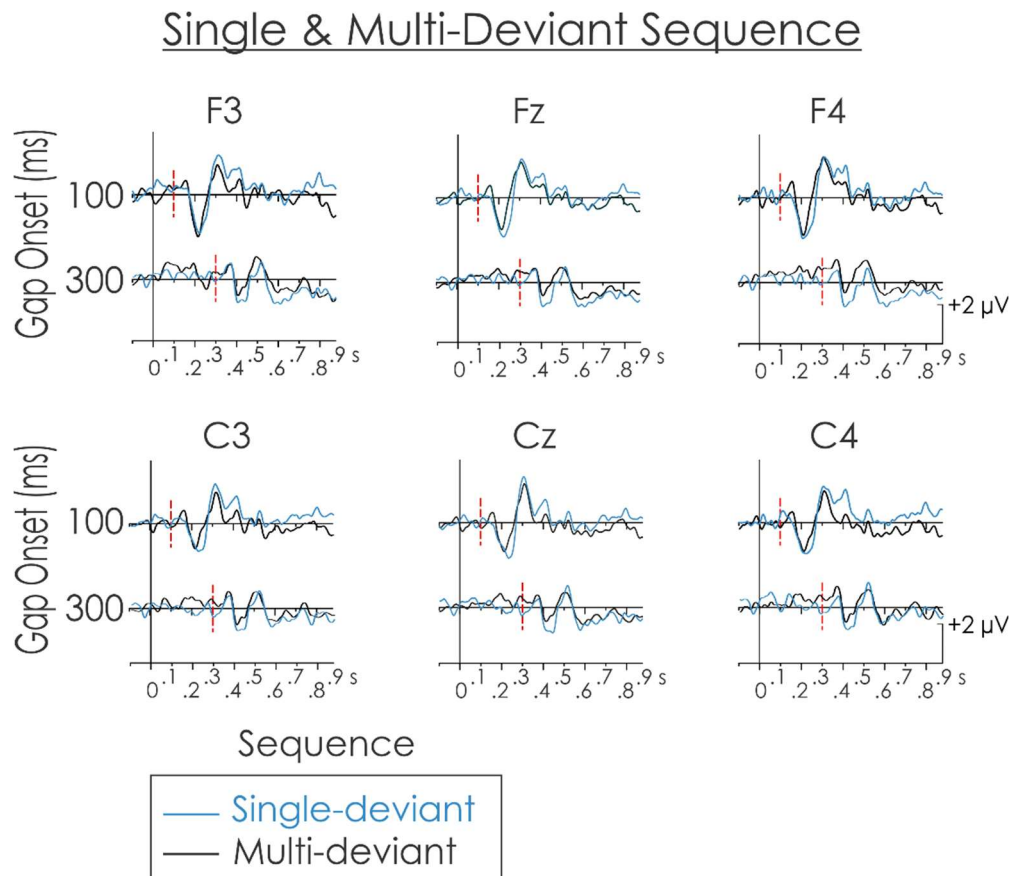


Figure 3.5 The effect of single- and multi-deviant paradigms. The 10-ms duration gap deviant was presented in both paradigms, occurring at 100 or 300 ms after stimulus onset. The amplitude of the DRN was significantly reduced in both paradigms when the gap occurred at 300 ms. The amplitude of the DRN was also reduced in the multi-deviant paradigm, but the difference was not significant. Note, however, that random differences were apparent before the onset of the gap. The DRN was therefore also measured peak-to-peak with respect to the subsequent positivity.

3.3.2 Behavioural data

Behavioural performance was measured using the signal detection measure, d' , based on both the probability of a correct detection of the deviant (i.e., hit rate) and the probability of a standard being detected (incorrectly) as a deviant (i.e., false positive rate). Figure 3.6 presents the mean d' for the 10- and 30-ms gaps as a function of gap onset time.

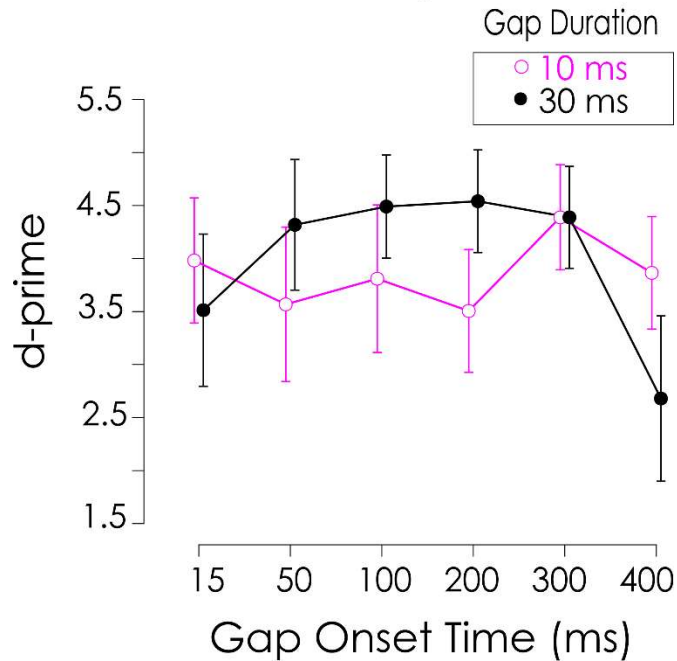


Figure 3.6 Behavioural performance data. Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time. Error bars represent SE.

As may be observed, d' values were very high, regardless of gap onset time. A 2-way ANOVA was used to determine the effects of gap duration and onset time. The main effect of gap duration was not significant, $F(1,16) = 1.88$, $p = .19$, $\eta_p^2 = .10$. On the other hand, the main effect of gap onset time was significant, $F(5,80) = 8.25$, $p < .001$, $\eta_p^2 = .34$. The interaction between gap duration and onset time was also significant, $F(5,80) = 8.35$, $p < .001$, $\eta_p^2 = .34$. Simple main effects testing was used to determine the source of the interaction. For the 10-ms gap, Holm post-hoc tests showed no significant differences in d' across the different gap onset times. However, for the 30-ms gap, d' for a gap occurring at 400 ms was significantly reduced compared to all other onset times ($p < .02$) except for that occurring at 15 ms. No other gap onset comparisons were significant.

Correlations between the behavioural and DRN measures were also computed. No significant correlations were found between d' and DRN values at either the frontal or the central ROI in either the multi- or single-deviant conditions.

3.4 Discussion

The present study investigated the influence of gap duration and onset time on the amplitude of the DRN. The standard was a 500-ms-duration broadband noise burst. The deviant was created by inserting a gap deviant at six different times, ranging from 15 to 400 ms after stimulus onset. The gap duration was either 10 or 30 ms. The stimulus offset (or onset of the gap) was instantaneous.

DRN amplitude was significantly larger for the 30-ms gap than for the 10-ms gap. This replicates a large number of previous studies showing a larger DRN for longer-duration gaps (Bertoli et al., 2001; Alain et al., 2004; Torppa et al., 2014; Duda-Milloy et al., 2019; Duda et al., 2020, 2025).

The multi-deviant paradigm was initially designed for use with deviants having different features. In the present study, all deviants within a given sequence contained the same type of acoustic change—a silent gap of the same duration—and differed only in the temporal position of that gap. There was, therefore, doubt about the efficacy of the multi-deviant paradigm. What varied among the six gap deviants was the timing of their occurrence. The use of the multi-deviant paradigm was, nevertheless, successful. CI testing revealed that a significant DRN was elicited for all 30-ms-duration gaps, and for most 10-ms gap. For the 10-ms gap, a significant DRN was not observed at 300 ms at the frontal ROI, or at 300 and 400 ms at the central ROI. Critically, the multi-deviant results for the 10-ms duration gap at 100 and 300 ms were compared with oddball results in which a single 10-ms gap occurred at either 100 or 300 ms in separate runs. The amplitude of the DRN did not significantly vary between the single- and multi-deviant conditions. However, the DRN was numerically larger in the single-deviant paradigm, and the paradigm effect approached significance at both frontal and central ROIs. Therefore, the present findings should not be interpreted as evidence that the two paradigms are equivalent. Rather, they indicate that the multi-deviant paradigm can elicit a measurable DRN while allowing several gap onset times to be examined within a single, time-efficient recording sequence. The temporal position of the gap did influence the amplitude of the DRN. For the 30-ms gap, the amplitude of the DRN gradually decreased with increasing gap onset delay. The effect was less consistent for the 10-ms gap. The amplitude of the DRN was significantly reduced for the shorter gap duration compared to the longer one across all gap onset times. Residual random background noise would therefore probably

have contaminated the measurement of this DRN. Importantly, a large decrease in the amplitude of the DRN was apparent for gap onsets occurring at 300 and 400 ms. These findings are thus consistent with the temporal distance effect (Grimm et al., 2004, 2006; Grimm & Schröger, 2005; Weise et al., 2007, 2010, 2011; Timm et al., 2011; Hoonhorst et al., 2012). The effect has been explained by temporal integration theory. Sound features are initially extracted and subsequently integrated with previous and subsequent features into a single unit. This single perceptual unit forms the deviant representation in sensory memory. Integration theory, however, limits the integration window to a very short time interval, estimated at no more than 300 ms. The DRN gradually decreased in amplitude from 15 to 300 ms. This is consistent with suggestions that stimulus changes that occur early (e.g., at 15 to 100 ms) contribute more to the representation in sensory memory than those that occur later (e.g., after 200 to 300 ms). Stimulus change that occurs to the long-duration standard after 300 ms after stimulus onset, and certainly after 400 ms, will contribute little to the representation of the deviant stimulus. As such, the DRN/MMN will be absent or much reduced in amplitude (Weise et al., 2023).

A further restriction on the effects of the window for temporal integration is that stimulus change within a long-duration stimulus must be gradual. Studies typically gradually change the features of the standard toward those of the deviant, then gradually change the features of the deviant back toward those of the standard (see the review by Weise et al., 2023). If the deviant represents a rapid transient change to a feature, a new integration window might be initiated. If this transient change occurs after 300 ms, the DRN/MMN will not be attenuated because of the creation of a new integration window. Weise et al. (2023) indicate that such transients elicit N1 and P2 deflections. The gaps in the present study similarly consisted of brief silent interruptions within an ongoing broadband-noise stimulus and had instantaneous onset and offset. As such, when the gap occurred at either 300 or 400 ms, it should have initiated a new integration window. The amplitude of the DRN should, therefore, not have differed from the DRN elicited by gaps that occurred earlier. This was not the case; the DRN was much attenuated. A reduction in the mismatch response for later-occurring gaps has also been reported by Yabe et al. (2001) and Tamakoshi et al. (2016). Yabe et al. (2001) used a 170-ms stimulus in which silent segments occurred at 26–62, 62–98, 98–134, or 134–170 ms after stimulus onset; responses were reduced for the later omissions. Tamakoshi et al. (2016) used a 176-ms tone train in which the second segment (approximately 22–44 ms) or fifth segment (approximately 88–110 ms) was omitted to create the

early- and late-gap conditions, respectively. In both studies, a reduction in the MMN was nevertheless observed when the gap occurred later (Yabe et al., 2001; Tamakoshi et al., 2016). These results appear to be in contrast with those observed by Weise et al. (2007). The stimuli and procedures used in the Weise et al. and Yabe et al. series of studies differed, making direct comparisons difficult. For example, in the Weise et al. (2007) study, when their frequency modulation occurred prior to 300 ms, a large MMN was observed. If the frequency modulation occurred later, the MMN was much reduced or absent. However, when a gap stimulus was introduced 200 ms before the onset of a frequency deviant occurring at 550 ms, the deviant now elicited an MMN. Weise et al. suggest that the introduction of the gap initiated a new window of integration, encompassing the time of the frequency deviant. In the Weise et al. (2007) study, introducing a gap shortly before the onset of the frequency change may have led to a shift in attention from the task at hand (watching a video) to the task-irrelevant auditory channel. The gap might have acted as a warning signal or cue for the subsequent onset of the frequency deviant (Parmentier, 2014). It might have been this shift of attention to the auditory channel and subsequent additional processing of the frequency deviants that were responsible for the elicitation of the large amplitude MMN.

The much-reduced DRN observed for the gap deviants occurring after 300 and 400 ms may result from a large decrease in their perceptibility. This was not the case. The signal detection measure d' , was used to assess the perceptibility of the gap. While the main effect of gap onset time was found, overall participants easily detected the deviants as evidenced by very high d' scores, even for the gap occurring at 400 ms, when the amplitude of the DRN was very much reduced. Moreover, correlations between DRN amplitude and behavioural performance were not significant. Other studies have also reported that stimulus changes to the standard after 300 ms are easily detected, even though the MMN was much reduced or absent (Hoonhorst et al., 2012; Timm et al., 2011). The procedures used to present the stimuli within the multi-deviant paradigm and for the behavioural task were very different. Importantly, task demands were dramatically different. In the single- and multi-deviant paradigms, participants were asked to ignore the rapidly presented auditory stimuli and to attend to a visual task (watching a video). The DRN was thus elicited passively. This process involved an automatic change-detection system comparing the features of the incoming deviant to those of the standard, stored as a representation in a limited-capacity, brief-lasting sensory memory. By contrast, during the behavioural task, participants were asked to

actively attend to the auditory sequence and to signal their detection of the now slowly-presented standard and deviant stimuli. This processing relies on a longer-lasting, much greater capacity working memory (Baddeley & Hitch, 1974; Baddeley, 2007). Information stored in working memory is prevented from fading through active attention involving rehearsal. Weise et al. (2023), commenting on the temporal position effect, suggest that reduced DRN amplitude after 350 ms is a consequence of the limited capacity of the integration process of the auditory sensory store. This constraint is overcome during the active behavioural task involving processes related to a much larger working memory capacity.

These findings have important implications, particularly for speech perception. The automatic detection of brief pauses (or gaps) between phonemes and longer pauses between words is essential for speech perception (Phillips, 1999; Elangovan & Stuart, 2008). The temporal distance effect appears to constrain the automatic detection of gaps to those occurring within 300 ms of stimulus onset. Several studies, however, have indicated that auditory change occurring after 300 ms can elicit a robust MMN (Weise et al., 2007, 2010, 2011). An MMN has also been elicited by a change to a syllable within a standard pseudoword that occurs after 300 ms (Kharlamov et al., 2011; Vidal et al., 2019). The Weise et al. (2010, 2011, 2023) position is that speech consists of a series of concatenated segments formed by the occurrence of transients, such as silent periods or gaps. The occurrence of these transients initiates a new integration window. Thus, when a change occurs to any of these segments, an MMN will be elicited even if the changed segment occurs well after 300 ms. The results of the present study question this explanation, although precisely what defines a transient change remains to be determined.

The duration of the integration window appears to be flexible, as it can be influenced by factors such as attention and learning (Grimm & Schröger, 2005; Kharlamov et al., 2011). Kiss et al. (2008) presented brief-duration reversed speech sound segments. Their detectability was influenced by whether the participants were fluent in the language of the speech segment. Many key temporal cues in speech, such as voice onset time, which marks the brief interval between a stop consonant release and the onset of voicing, depend on millisecond-level timing precision (Phillips, 1998). The present study supports the view that the closer the gap is to the stimulus onset, the more efficiently automatic auditory gap detection operates. Moreover, although the current study employed a passive listening paradigm, future work should examine how task demands and attentional engagement influence DRN amplitude and the temporal constraints of auditory

integration. These extensions would build upon the current findings and further elucidate the mechanisms underlying automatic auditory change detection across the lifespan and in more ecologically valid contexts.

3.5 Limitations

The present study created a deviant stimulus by inserting a brief-lasting silent period (or gap) in a long-duration standard stimulus. The abrupt onset of the gap did not elicit a pure MMN but rather a composite N1+MMN negativity that was labelled a DRN. It was therefore not possible to determine if the decline in this negativity was exclusive to the MMN or whether the N1 was also affected. This limits the extent to which the findings can be attributed specifically to changes in automatic deviance detection or temporal integration.

The present study also examined the effects of gap duration in broadband noise. The findings were obtained using a specific stimulus configuration: 500-ms broadband-noise bursts containing 10- or 30-ms gaps with instantaneous onset and offset. The temporal position effect may depend on properties such as gap duration, stimulus bandwidth, intensity, and the rise-and-fall characteristics of the gap. Therefore, the present results may not generalize to other acoustic stimuli or to more gradual within-stimulus changes.

The comparison between the multi-deviant and single-deviant paradigms was limited to 10-ms gaps occurring at 100 and 300 ms. Therefore, the findings cannot necessarily be generalized to the other gap onset times or to the 30-ms gap condition. In addition, the EEG session was relatively long, lasting approximately three hours, which may have introduced fatigue or changes in alertness across the session despite the use of rest periods and counterbalancing.

The present study employed a young adult sample. The findings therefore cannot be generalized directly to children or older adults, in whom auditory sensory memory and temporal integration may differ (Cabrera et al., 2022; Wang et al., 2005). Future studies should examine whether the same reduction in DRN amplitude for later-occurring gaps is observed across different age groups and under different attentional and stimulus conditions.

3.6 Conclusions

This study demonstrated that a DRN can be reliably elicited using a multi-deviant paradigm when all deviants are created by inserting the same type of acoustic change, a silent gap, into the

same 500-ms broadband-noise stimulus, while the temporal position of the gap varies across deviants. The onset of the gap deviants occurred at different times, ranging from 15 to 400 ms after the onset of the long-duration deviant stimulus. In different conditions, the gap duration was either 10 or 30 ms. As expected, the DRN was larger following presentation of the 30-ms gap than following presentation of the 10-ms gap. Temporal integration theory maintains that stimulus change occurring 300 ms or later after stimulus onset will result in a much reduced or absent MMN/DRN. The results revealed a clear gradual decline in DRN amplitude as gap onset occurred later for the 30-ms gap, whereas this pattern was less consistent for the 10-ms gap. Nevertheless, DRN amplitude was markedly reduced for gaps occurring after 300 ms. This probably reflects a temporal limitation in sensory memory and in auditory deviance-detection mechanisms. Although no statistically significant difference in DRN amplitude was detected between the two paradigms, responses were numerically larger in the single-deviant paradigm. Because the study was not designed or powered to test equivalence, the findings should not be interpreted as demonstrating that the two paradigms produce equivalent responses.

Declaration of conflict of interest

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

Acknowledgements

The authors thank Félix Harvey and Olivia Chanel Zupicic-Grsic for their assistance with data collection. We are also grateful to all the participants for their time and contribution to this study.

References

- Avivi-Reich, M., Arnott, S.R., Alain, C., Rabaglia, C., Schneider, B.A. (Under revision). The influence of auditory training on behavioral and electrophysiological measures of gap detection in younger and older adults. *Attention, Perception and Psychophysics*.
- Alain, C., Woods, D. L., & Ogawa, K. H. (1994). Brain indices of automatic pattern processing. *Neuroreport*, 6(1), 140-144.
- Alain, C., McDonald, K. L., Ostroff, J. M., & Schneider, B. (2004). Aging: a switch from automatic to controlled processing of sounds? *Psychology and aging*, 19(1), 125.
- Alamatsaz, N., Rosen, M. J., & Ihlefeld, A. (2024). Increased reliance on temporal coding when target sound is softer than the background. *Scientific reports*, 14(1), 4457.

- American National Standards Institute (ANSI) (1996), American National Standard specification for audiometers. ANSI S3.6. New York: Author.
- Arnott, S. R., & Alain, C. (2002). Effects of perceptual context on event-related brain potentials during auditory spatial attention. *Psychophysiology*, 39(5), 625-632.
- Baddeley, A. (2007). *Working memory, thought, and action*. Oxford University Press. <https://doi.org/10.1093/acprof:oso/9780198528012.001.0001>
- Baddeley, A. D., & Hitch, G. J. (1974). Working Memory. In G. A. Bower (Ed.), *Recent Advances in Learning and Motivation* (Vol. 8, pp. 47-89). New York: Academic Press.
- Bernstein, J. G., & Grant, K. W. (2009). Auditory and auditory-visual intelligibility of speech in fluctuating maskers for normal-hearing and hearing-impaired listeners. *The Journal of the Acoustical Society of America*, 125(5), 3358-3372.
- Bertoli, S., Heimberg, S., Smurzynski, J., & Probst, R. (2001). Mismatch negativity and psychoacoustic measures of gap detection in normally hearing subjects. *Psychophysiology*, 38(2), 334-342.
- Bertoli, S., Smurzynski, J., & Probst, R. (2002). Temporal resolution in young and elderly subjects as measured by mismatch negativity and a psychoacoustic gap detection task. *Clinical Neurophysiology*, 113(3), 396-406.
- Bizley, J. K., & Cohen, Y. E. (2013). The what, where and how of auditory-object perception. *Nature Reviews Neuroscience*, 14(10), 693-707.
- Brown, S., & Nicholls, M. E. (1997). Hemispheric asymmetries for the temporal resolution of brief auditory stimuli. *Perception & Psychophysics*, 59, 442-447.
- Cabrera, L., Lorenzini, I., Rosen, S., Varnet, L., & Lorenzi, C. (2022). Temporal integration for amplitude modulation in childhood: Interaction between internal noise and memory. *Hearing research*, 415, 108403.
- Campbell, K., & Macdonald, M. (2011). The effects of attention and conscious state on the detection of gaps in long duration auditory stimuli. *Clinical neurophysiology: official journal of the International Federation of Clinical Neurophysiology*, 122(4), 738-747.
- Cowan, N. (1984). On short and long auditory stores. *Psychological bulletin*, 96(2), 341-370.
- Desjardins, R. N., Trainor, L. J., Hevenor, S. J., & Polak, C. P. (1999). Using mismatch negativity to measure auditory temporal resolution thresholds. *Neuroreport*, 10(10), 2079-2082.
- Diedler, J., Pietz, J., Bast, T., & Rupp, A. (2007). Auditory temporal resolution in children assessed by magnetoencephalography. *Neuroreport*, 18(16), 1691-1695.
- Duda-Milloy, V., Tavakoli, P., Campbell, K., Benoit, D. L., & Koravand, A. (2019). A time-efficient multi-deviant paradigm to determine the effects of gap duration on the mismatch negativity. *Hearing Research*, 377, 34-43.
- Duda, V., Campbell, K., & Koravand, A. (2020). Event-related potentials following gaps in noise: The effects of the intensity of preceding noise. *Brain research*, 1748, 147078.
- Duda, V., Augereau, T. M. D., Campbell, K., & Koravand, A. (2025). Does masking alter the mismatch negativity response to gaps? *Brain research*, 1859, 149651.

- Eggermont, J. J. (1995). Neural correlates of gap detection and auditory fusion in cat auditory cortex. *Neuroreport*, 6(12), 1645-1648.
- Elangovan, S., & Stuart, A. (2008). Natural boundaries in gap detection are related to categorical perception of stop consonants. *Ear and hearing*, 29(5), 761-774.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39, 175-191.
- Forrest, T. G., & Green, D. M. (1987). Detection of partially filled gaps in noise and the temporal modulation transfer function. *The Journal of the Acoustical Society of America*, 82(6), 1933-1943.
- Gay, J. D., Rosen, M. J., & Huyck, J. J. (2020). Effects of gap position on perceptual gap detection across late childhood and adolescence. *Journal of the Association for Research in Otolaryngology*, 21(3), 243-258.
- Gelfand, S. A. (2016). *Essentials of audiology* (Fourth ed.). Thieme.
- Grimm, S., Widmann, A., & Schröger, E. (2004). Differential processing of duration changes within short and long sounds in humans. *Neuroscience Letters*, 356(2), 83-86.
- Grimm, S., & Schröger, E. (2005). Pre-attentive and attentive processing of temporal and frequency characteristics within long sounds. *Cognitive Brain Research*, 25(3), 711-721.
- Grimm, S., Roeber, U., Trujillo-Barreto, N. J., & Schröger, E. (2006). Mechanisms for detecting auditory temporal and spectral deviations operate over similar time windows but are divided differently between the two hemispheres. *Neuroimage*, 32(1), 275-282.
- Gyldenkerne, P., Dillon, H., Sharma, M., & Purdy, S. C. (2014). Attend to this: The relationship between auditory processing disorders and attention deficits. *Journal of the American Academy of Audiology*, 25(7), 676-687.
- He, S., Grose, J. H., Teagle, H. F., Woodard, J., Park, L. R., Hatch, D. R., & Buchman, C. A. (2013). Gap detection measured with electrically evoked auditory event-related potentials and speech-perception abilities in children with auditory neuropathy spectrum disorder. *Ear and hearing*, 34(6), 733-744.
- Heinrich, A., Alain, C., & Schneider, B. A. (2004). Within-and between-channel gap detection in the human auditory cortex. *Neuroreport*, 15(13), 2051-2056.
- Hoonhorst, I., Deltenre, P., Markessis, E., Collet, G., Martin, X. P., & Colin, C. (2012). Evidence for a dual versus single origin of the MMNs evoked by cued versus cueless deviants. *Clinical neurophysiology*, 123(8), 1561-1567.
- Keith, R. W. (2000). Random gap detection test. St. Louis, MO: Auditec.
- Kharlamov, V., Campbell, K., & Kazanina, N. (2011). Behavioral and electrophysiological evidence for early and automatic detection of phonological equivalence in variable speech inputs. *Journal of cognitive neuroscience*, 23(11), 3331-3342. <https://doi.org/10.1162/jocn.2011.21606>

- Kiss, M., Cristescu, T., Fink, M., & Wittmann, M. (2008). Auditory language comprehension of temporally reversed speech signals in native and non-native speakers. *Acta neurobiologiae experimentalis*, 68(2), 204–213. <https://doi.org/10.55782/ane-2008-1689>
- Makeig, S., Jung, T. P., Ghahremani, D., & Sejnowski, T. J. (1996). Independent component analysis of simulated ERP data. *Institute for Neural Computation, University of California: technical report INC-9606*
- Moore, B. C. (2008). Basic auditory processes involved in the analysis of speech sounds. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 363(1493), 947–963.
- Muller-Gass, A., Stelmack, R. M., & Campbell, K. B. (2006). The effect of visual task difficulty and attentional direction on the detection of acoustic change as indexed by the mismatch negativity. *Brain research*, 1078(1), 112–130.
- Musiek, F. E., Shinn, J. B., Jirsa, R., Bamio, D.-E., Baran, J. A., & Zaida, E. (2005). GIN (Gaps-In-Noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear and hearing*, 26(6), 608–618.
- Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, 24(4), 375–425.
- Näätänen, R. (1992). Event-related potentials and automatic information processing. *Attention and brain function*, 102–210.
- Näätänen, R., Jiang, D., Lavikainen, J., Reinikainen, K., & Paavilainen, P. (1993). Event-related potentials reveal a memory trace for temporal features. *Neuroreport*, 5(3), 310–312.
- Näätänen, R., & Winkler, I. (1999). The concept of auditory stimulus representation in cognitive neuroscience. *Psychological bulletin*, 125(6), 826.
- Näätänen, R., Pakarinen, S., Rinne, T., & Takegata, R. (2004). The mismatch negativity (MMN): towards the optimal paradigm. *Clinical Neurophysiology*, 115(1), 140–144.
- Näätänen, R., Syssoeva, O., & Takegata, R. (2004). Automatic time perception in the human brain for intervals ranging from milliseconds to seconds. *Psychophysiology*, 41(4), 660–663.
- Palmer, S. B., & Musiek, F. E. (2013). N1-p2 recordings to gaps in broadband noise. *Journal of the American Academy of Audiology*, 24(1), 37–45.
- Parmentier, F. B. R. (2014). The cognitive determinants of behavioral distraction by deviant auditory stimuli: A review. *Psychol. Res.*, 78, 321–338.
- Penner, M. J. (1977). Detection of temporal gaps in noise as a measure of the decay of auditory sensation. *The Journal of the Acoustical Society of America*, 61(2), 552–557.
- Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and clinical neurophysiology*, 72(2), 184–187.
- Phillips, D. P. (1998, November). Sensory representations, the auditory cortex, and speech perception. In *Seminars in hearing* (Vol. 19, No. 04, pp. 319–332). Copyright© 1998 by Thieme Medical Publishers, Inc.

- Phillips, D. P. (1999). Auditory gap detection, perceptual channels, and temporal resolution in speech perception. *Journal of the American Academy of Audiology*, 10(06), 343-354.
- Pivik, R. T., Broughton, R. J., Coppola, R., Davidson, R. J., Fox, N., & Nuwer, M. R. (1993). Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology*, 30(6), 547-558.
- Pratt, H., Starr, A., Michalewski, H. J., Bleich, N., & Mittelman, N. (2007). The N1 complex to gaps in noise: effects of preceding noise duration and intensity. *Clinical neurophysiology*, 118(5), 1078–1087. <https://doi.org/10.1016/j.clinph.2007.01.005>
- Giannella Samelli, A., & Schochat, E. (2008). The gaps-in-noise test: gap detection thresholds in normal-hearing young adults. *International Journal of Audiology*, 47(5), 238-245.
- Ross, B., Schneider, B., Snyder, J., & Alain, C. (2010). Biological markers of auditory gap detection in young, middle-aged, and older adults. *PLoS ONE*, 5(4), e10101. doi:10.1371/journal.pone.0010101.
- Schröger, E., Näätänen, R., & Paavilainen, P. (1992). Event-related potentials reveal how non-attended complex sound patterns are represented by the human brain. *Neuroscience Letters*, 146(2), 183-186.
- Shinn, J. B., Chermak, G. D., & Musiek, F. E. (2009). GIN (Gaps-In-Noise) performance in the pediatric population. *Journal of the American Academy of Audiology*, 20(04), 229-238.
- Silman, S., Silverman, C. A., & Emmer, M. B. (2000). Central auditory processing disorders and reduced motivation: Three case studies. *Journal of the American Academy of Audiology*, 11(02), 57-63.
- Snell, K. B., & Hu, H. L. (1999). The effect of temporal placement on gap detectability. *The Journal of the Acoustical Society of America*, 106(6), 3571-3577.
- Sussman, E. S. (2007). A new view on the MMN and attention debate: the role of context in processing auditory events. *Journal of Psychophysiology*, 21(3-4), 164-175.
- Sussman, E. S. (2017). Auditory scene analysis: An attention perspective. *Journal of Speech, Language, and Hearing Research*, 60(10), 2989-3000.
- Tamakoshi, S., Minoura, N., Katayama, J. i., & Yagi, A. (2016). Entire sound representations are time-compressed in sensory memory: evidence from MMN. *Frontiers in neuroscience*, 10, Article 347.
- Tavakoli, P., Duda, V., Boaf, A., & Campbell, K. (2021). The effects of sleep on objective measures of gap detection using a time-efficient multi-deviant paradigm. *Brain and Cognition*, 152, 105772.
- Timm, J., Weise, A., Grimm, S., & Schröger, E. (2011). An asymmetry in the automatic detection of the presence or absence of a frequency modulation within a tone: a mismatch negativity study. *Frontiers in Psychology*, 2, 189.
- Todd, J., Finch, B., Smith, E., Budd, T. W., & Schall, U. (2011). Temporal processing ability is related to ear-asymmetry for detecting time cues in sound: A mismatch negativity (MMN) study. *Neuropsychologia*, 49(1), 69-82.

- Torppa, R., Huotilainen, M., Leminen, M., Lipsanen, J., & Tervaniemi, M. (2014). Interplay between singing and cortical processing of music: a longitudinal study in children with cochlear implants. *Frontiers in Psychology*, 5, 1389.
- Uther, M., Jansen, D. H. J., Huotilainen, M., Ilmoniemi, R. J., & Näätänen, R. (2003). Mismatch negativity indexes auditory temporal resolution: evidence from event-related potential (ERP) and event-related field (ERF) recordings. *Cognitive Brain Research*, 17(3), 685-691.
- Vidal, Y., Brusini, P., Bonfieni, M., Mehler, J., & Bekinschtein, T. A. (2019). Neural Signal to Violations of Abstract Rules Using Speech-Like Stimuli. *eNeuro*, 6(5), 1-14. <https://doi.org/10.1523/ENEURO.0128-19.2019>.
- Wang, W., Datta, H., & Sussman, E. (2005). The development of the length of the temporal window of integration for rapidly presented auditory information as indexed by MMN. *Clinical neurophysiology*, 116(7), 1695–1706. <https://doi.org/10.1016/j.clinph.2005.03.008>
- Weise, A., Müller, D., Grimm, S., Rübsamen, R., & Schröger, E. (2007). Differential processing of terminal tone parts within structured and non-structured tones. *Neuroscience letters*, 421(2), 163-167.
- Weise, A., Grimm, S., Müller, D., & Schröger, E. (2010). A temporal constraint for automatic deviance detection and object formation: a mismatch negativity study. *Brain research*, 1331, 88-95.
- Weise, A., Ritter, W., & Schröger, E. (2011). The representation of unattended, segmented sounds: A mismatch negativity (MMN) study. *International journal of psychophysiology*, 81(2), 121-126.
- Weise, A., Grimm, S., Rimmele, J. M., & Schröger, E. (2023). Auditory representations for long lasting sounds: Insights from event-related brain potentials and neural oscillations. *Brain and Language*, 237, 105221.
- Woldorff, M. G., Hillyard, S. A., Gallen, C. C., Hampson, S. R., & Bloom, F. E. (1998). Magnetoencephalographic recordings demonstrate attentional modulation of mismatch-related neural activity in human auditory cortex. *Psychophysiology*, 35(3), 283-292.
- Winkler, I. (2007). Interpreting the mismatch negativity. *Journal of psychophysiology*, 21(3-4), 147-163.
- Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: predictive regularity representations and perceptual objects. *Trends in cognitive sciences*, 13(12), 532-540.
- Yabe, H., Koyama, S., Kakigi, R., Gunji, A., Tervaniemi, M., Sato, Y., & Kaneko, S. (2001). Automatic discriminative sensitivity inside temporal window of sensory memory as a function of time. *Cognitive Brain Research*, 12(1), 39-48.
- Yabe, H., Matsuoka, T., Sato, Y., Hiruma, T., Sutoh, T., Koyama, S., Gunji, A., Kakigi, R., & Kaneko, S. (2005). Time may be compressed in sound representation as replicated in sensory memory. *Neuroreport*, 16(2), 95-98.

Chapter 4: Effects of gap onset time and gap duration on the deviant-related negativity in typically developing school-age children

Authors:

Shaghayegh Omidvar¹, Kenneth Campbell², Claude Alain^{3,4}, Steven Aiken⁵, Amineh Koravand^{1*}

Affiliations:

¹ School of Rehabilitation Sciences, University of Ottawa, Ottawa, Canada,

² School of Psychology, University of Ottawa, Ottawa, Canada

³ Department of Psychology, University of Toronto, Toronto, Ontario, Canada

⁴ Rotman Research Institute, Baycrest Academy for Research and Education, Toronto, Ontario, Canada

⁵ School of Communication Sciences and Disorders, Dalhousie University, Halifax, Nova Scotia, Canada

***Corresponding address:**

Faculty of Health Sciences, University of Ottawa, 200 Lees Ave, Ottawa, Canada, ON K1S 5S9

Abstract

Objectives: This study examined whether automatic change detection to within-stimulus gaps, indexed by the deviant-related response (DRN), varies as a function of gap duration and the temporal position of the gap within a long-duration sound, and whether these effects differ between children and adults.

Methods: Fifteen typically developing children (8–12 years) and fifteen adults (23–37 years) completed passive EEG recordings using a multi-deviant sequence in which standards and deviants alternated. All stimuli were 500-ms broadband-noise bursts. Deviants contained a silent gap of either 10 or 30 ms inserted at 15, 50, 100, 200, 300, or 400 ms after stimulus onset. DRNs were derived from deviant–standard difference waves, and the presence of a DRN was evaluated using confidence interval (CI) procedures. In a separate behavioural task, participants performed active gap detection, and sensitivity was quantified using d' .

Results: In both groups, DRN amplitude increased with gap duration and decreased as the gap onset time was delayed. Reliable DRNs were absent for the latest gap position (400 ms) in both groups. In adults, a small DRN was evident at 300 ms, whereas in children, CI testing showed that significant DRNs were limited to the 30-ms gaps occurring at 15 and 50 ms. Behavioural sensitivity was high overall but was lower in children than in adults and did not correlate with DRN amplitude.

Conclusions: Automatic detection of within-stimulus gaps is constrained by the timing of the change, and this temporal constraint appears to be more pronounced in children. The dissociation between DRN outcomes and behavioural performance suggests that active detection may be supported by mechanisms beyond those indexed by the passive DRN.

Keywords: mismatch negativity; deviant-related response; gap detection; auditory temporal resolution; sensory memory; development

4.1 Introduction

Auditory temporal processing refers to the ability to perceive and interpret changes in sound over time or within a limited time interval (Amaral et al., 2015). It involves accurately processing the timing, order, and structure of auditory signals, which are essential for distinguishing fine phonetic elements and achieving clear speech perception. Impairments in temporal processing can disrupt the neural representation of phonemes and hinder speech understanding, potentially contributing to speech and language disorders (Martin et al., 2025). A key component of temporal processing is temporal resolution, which is the minimum time required by the central auditory nervous system to distinguish two successive acoustic events (Musiek et al., 2005). Temporal resolution is commonly assessed using psychoacoustic tasks that measure the ability to detect short, silent intervals (gaps) between two sounds. A participant might, for example, be asked to judge whether a gap has occurred within a continuous or long-duration auditory stimulus. The shortest detectable gap duration is called the gap detection threshold (Musiek et al., 2005).

In adults, the gap detection threshold ranges from 4 to 6 ms (Braga et al., 2015; Giannella Samelli & Schochat, 2008; Heeke et al., 2018; Wong & McPherson, 2015). In children, several studies have shown age-related improvements in gap detection ability; however, the degree of developmental sensitivity varies considerably depending on the acoustic properties of the stimuli (Buss et al., 2014; Buss et al., 2017; Diedler et al., 2007; Gay et al., 2020; Phillips et al., 2010; Trehub et al., 1995). The limitation of relying solely on behavioural gap-detection measures is that they require active participation in a task for a relatively long period. Children's performance on a behavioural task may thus be confounded by an inability to maintain attention throughout the testing session. In addition, behavioural methods require a perceptual decision, which can also be influenced by other nonsensory factors, such as response bias, attention and motivation to carry out the task (Gyldenkerne et al., 2014; Silman et al., 2000).

Many laboratories record auditory event-related potentials (ERPs) to provide an objective measure of information processing (Picton et al., 2000). ERPs are changes in the electrical activity of the brain following the presentation of a physical stimulus or a psychological event. ERPs consist of a series of negative- and positive-going deflections, reflecting different stages of processing. In the auditory domain, these include exogenous, obligatory responses that are largely

determined by the physical characteristics of the auditory stimulus, such as P1, N1, and P2, as well as endogenous responses that reflect information processing in the brain (Picton et al., 2000). Some of these ERP deflections can be elicited independent of attention. Many auditory ERP studies employ a so-called auditory “oddball” paradigm to examine auditory change detection (Näätänen et al., 1993). The participant is presented with a frequently occurring homogeneous “standard” auditory stimulus. At rare (or “odd”) times, a feature of the standard stimulus is changed to form a “deviant” stimulus. In adults, both standard and deviant auditory stimuli elicit a series of obligatory responses, labelled P1, N1, and P2, occurring at about 50, 100, and 180 ms, respectively (Picton et al., 1974; Näätänen & Picton, 1987). When stimuli are presented rapidly (more than once per second), the N1 wave is much reduced (Budd et al., 1998; Čeponien et al., 1998; Pereira et al., 2014). In younger children, a very large positivity is elicited at about 100 ms (Sharma et al., 1997). This may be a late-onset P1 (Bishop et al., 2007; Bruneau & Gomot, 1998; Bruneau et al., 1997; Čeponien et al., 1998; Čeponien et al., 2002; Davies et al., 2010; Mahajan & McArthur, 2012; Ponton et al., 2002; Sharma et al., 1997). The N1 wave may be difficult to observe in young children. Picton and Taylor (2007) suggest that a small N1 wave may be embedded in a much larger positive deflection occurring at about the same time, making the N1 difficult to observe (Picton & Taylor, 2007).

The mismatch negativity (MMN) is an auditory ERP response elicited by a discriminable change in an established auditory regularity; a gap deviant represents one type of acoustic change that can elicit this response (Näätänen et al., 2007). In the original Näätänen model (1990) of auditory perception, the features of the standard stimulus are said to be automatically extracted and stored in sensory memory. Each incoming stimulus is compared to that representation, with deviant sounds generating an MMR between the incoming stimulus and the representation of the standard stimulus (Näätänen, 1990). The MMN occurs between 100 and 250 ms after the onset of the deviant, depending on the extent of acoustic change. Its amplitude is maximum over frontocentral regions of the scalp and inverts in polarity at the mastoids when a nose reference is used (Näätänen et al., 2007; Picton et al., 2000).

Another model of the MMN postulates that the MMN is generated when a stimulus violates an expected occurrence of a rule-based pattern (Winkler et al., 2009). Thus, in a sequence in which two auditory stimuli alternate (a,b,a,b,a,b,a,b), when the rule-based pattern is violated (a,b,a,b,a,a,b,a), the repetition of the same “a” stimulus elicits a MMN even though the stimulus

that preceded it is physically identical (Alain et al., 1994; Nordby et al., 1988). The oddball paradigm is a special case of a rule-based pattern. After the occurrence of a standard, the rule-based pattern leads to the expectation for the occurrence of a subsequent standard; the occurrence of a deviant thus violates this pattern. The output of the change detection system and, thus, the amplitude of the MMN vary directly with the extent of change. Critically, the detection of acoustic change and thus the elicitation of the MMN is claimed to occur automatically, independent of attention. Empirical studies have consistently shown that both the direction and the strength of attention have little influence on MMN amplitude (Muller-Gass et al., 2006; Näätänen, 1990; Näätänen et al., 2005; Sussman, 2007; Sussman et al., 2014). However, when the participants are asked to attend and respond to detect the deviant, additional negativity is elicited at the time of the MMN, referred to as the N2. To minimize overlap between the MMN and N2 components, participants are often asked to attend to a visual task, such as reading a book or watching a video, making the auditory stimuli task-irrelevant (i.e., no response required). In such paradigms, the assumption is that MMN is elicited while the N2 is not, or less so, because the auditory stimuli are not relevant and do not require a response (Fitzgerald & Todd, 2020; Näätänen et al., 2014).

The MMN reaches adult-like morphology early in childhood, reflecting an early maturation of pre-attentive auditory change detection (Themas et al., 2025). In children aged 6 to 13 years, MMN responses to changes in frequency and duration generally show peak latencies that are comparable to, or slightly longer than, adult latencies and exhibit a similar frontocentral negative scalp topography (Gomes et al., 1999; Lohvansuu et al., 2013; Shafer et al., 2000). Studies of early development find that very young children often show a positive mismatch response (MMR) to auditory deviants rather than the adult-like negative MMN (Ahmmed et al., 2008; Lee et al., 2012; Leppänen et al., 2004; Maurer et al., 2003; Näätänen et al., 2014; Putkinen et al., 2014; Shafer et al., 2010). Maurer et al. (2003), for instance, observed that 6–7-year-old children showed a frontal mismatch positivity (accompanied by posterior negativity) in response to deviants, in contrast to the clear frontocentral MMN seen in adults. Shafer et al. (2000) reported that while some 4-year-olds exhibited mature, adult-like MMNs to pure-tone contrasts, younger children frequently showed positive MMRs either in place of or preceding the emerging negative MMN. Similar findings have been documented across a range of stimuli: Ahmmed et al. (2008) noted positive MMRs to pure tones in children aged 7–12, and Lee et al. (2012) also identified positive MMRs to Mandarin speech sounds in 4–6 year-olds alongside developing MMNs. For vowels, Shafer et

al. (2010) also found that 4–5-year-olds tended to produce positive MMRs rather than the adult-like negative response.

Most studies assessing temporal resolution in children have employed oddball-like paradigms (Putkinen et al., 2014; Trainor et al., 2003; Trainor et al., 2001). In these paradigms, the frequently occurring standard does not contain a gap, while a deviant stimulus is created by inserting a brief silent period, often in the centre of the standard stimulus. The initial portion of the deviant, up to the occurrence of the gap, is physically identical to the standard. The occurrence of the gap signals a change. Negativity that is elicited by the gap deviant has been commonly labeled as an MMN. However, as Duda-Milloy et al. (2019) note, this is probably not a “true” MMN but rather a composite negativity. The gap onset (i.e., the offset of the stimulus in which it is embedded) elicits an exogenous N1. However, because the onset of the gap also signals a change from the previous standard stimuli, it will also elicit an MMN. The resulting composite N1+MMN response has been termed a deviant-related negativity (DRN) (Duda-Milloy et al., 2019; Woldorff et al., 1998).

In adults, studies have shown that a supra-threshold gap elicits a DRN occurring about 100 ms after the gap onset, with its amplitude increasing with gap duration (Alain et al., 2004; Bertoli et al., 2001; Desjardins et al., 1999; Duda-Milloy et al., 2019; Duda et al., 2025; Duda et al., 2020; Tamakoshi et al., 2016; Todd et al., 2011; Yabe et al., 2005). For example, Duda-Milloy et al. (2019) used a 200-ms white-noise burst as the standard stimulus, with gap deviants created by inserting a silent interval in the centre of the standard. Six deviants were presented, with gap durations ranging from 2 to 40 ms. As expected, a DRN was difficult to observe following the presentation of the sub-threshold 2-ms gap. It was, however, apparent for the near-threshold 5-ms gap. The DRN amplitude was larger for the longer-duration gaps. Follow-up studies have replicated these findings (Duda et al., 2025; Duda et al., 2020). Only a limited number of studies have examined DRN responses to gap deviants in infants and children. Many studies used oddball-like paradigms (Putkinen et al., 2014; Trainor et al., 2003; Trainor et al., 2001). Trainor et al. (2003) presented a deviant stimulus containing a 16-ms silent gap within a frequently occurring standard that did not contain a gap. They demonstrated that infants as young as 4–6 months exhibit MMN-like responses to the silent gaps, suggesting that temporal sensitivity emerges very early in development. Other studies have employed multi-feature paradigms (Partanen et al., 2013; Petermann et al., 2009; Rapaport et al., 2023) in which a gap deviant was presented among the

four (Petermann et al., 2009; Rapaport et al., 2023) or seven (Partanen et al., 2013) deviants. Each deviant represented a different change in a feature of the standard stimulus. In these studies, the gap deviants reliably elicit an MMN in children. Partanen et al. (2013) examined 30 children aged 4–12 years. The gap deviant consisted of a 100-ms silent interval embedded in the middle syllable of a pseudo-word (/tatata/). The gap deviant elicited a frontocentral MMN in all children, and the MMN amplitude did not differ between pre-school and school-aged children. Similarly, Petermann et al. (2009) investigated children aged 5 to 7 years using a multi-deviant paradigm that included a 16-ms silent gap inserted into the centre of a 100-ms standard stimulus. This gap deviant also elicited an MMN. In a more recent multi-deviant magnetoencephalography study, Rapaport et al. (2023) examined two groups of children (3–7 years) using a 7-ms silent gap as one of the deviants. They reported larger mismatch field amplitudes in the 5–7-year-old children. Unfortunately, their MMN results collapsed across all deviant stimulus types, limiting conclusions specific to gap processing.

Because acoustic information changes continuously over time, successive sound features must be integrated over a limited temporal interval to form a coherent auditory representation (Yabe et al., 1998). Temporal integration is a fundamental aspect of auditory processing. It allows the auditory system to form a perceptual object by initially analyzing and extracting the presently occurring sound features and then integrating the preceding and subsequent features into a single, unified representation, which is stored in sensory memory (Näätänen & Winkler, 1999). The amount of information that can be stored in sensory memory is very limited (Cowan, 1984). As more information accumulates over time, storage capacity will eventually be exceeded (Arnott & Alain, 2002; Bizley & Cohen, 2013; Poeppel, 2003). The period during which integration occurs is referred to as the temporal window of integration (TWI) (Yabe et al., 1998). The duration of the TWI varies depending on the type of auditory information being integrated, such as phonemic, syllabic, or simpler non-speech information. It may be very short (20–40 ms) for phonemes, but longer (125–250 ms) for syllables (Poeppel, 2003). For simpler, less complex auditory stimuli, the TWI may be as long as 300–400 ms (Weise et al., 2023). If an acoustic change occurs within TWI, a single integrated sound will be perceived. On the other hand, when the change occurs outside of the TWI, two sounds will be perceived (Näätänen, 1992; Winkler et al., 2009).

In MMN studies using frequency or intensity deviants, the acoustic difference between the standard and deviant usually occurs immediately at stimulus onset and persists throughout the

stimulus. This is not the case for a gap deviant. When a gap is created to form a deviant stimulus, it is often placed in the centre of the standard stimulus (Duda-Milloy et al., 2019; Linnavalli et al., 2022; Partanen et al., 2013). Nevertheless, changes in speech can occur at any time, from shortly after stimulus onset to very late after its onset. Some studies have examined the effects of changing only a small portion of a feature (often frequency) of the standard and inserting this change at various temporal positions (Alain et al., 1994; Grimm et al., 2006; Grimm & Schröger, 2005; Hoonhorst et al., 2012; Näätänen et al., 2004; Schröger et al., 1992; Tamakoshi et al., 2016; Timm et al., 2011; Weise et al., 2010; Weise et al., 2007; Yabe et al., 2001; Yabe et al., 2005). These studies have consistently reported a gradual decrease in MMN amplitude as the change occurs later within the stimulus. For example, Grimm et al. (2006) created deviant stimuli by inserting a brief (50-ms) change in the frequency of the standard at different onset times. The standard's duration also changed. The 50-ms frequency change occurred 100 ms after the sound onset of a 167-ms standard, 200 ms after the onset of a 333-ms standard, 300 ms after the onset of a 500-ms standard, or 400 ms after the onset of a 667-ms standard. Their results showed that an MMN was elicited only when the frequency change occurred within 300 ms following stimulus onset. Similar temporal constraints on MMN elicitation have been reported in other studies.

Our recent study in adults revealed that DRN amplitudes vary as a function of when the gap occurs within the stimulus, with marked reductions at 300 and 400ms onset times (Omidvar et al., 2026). In this paradigm, standards and deviants were alternated, and six different gap deviants were presented. What distinguished the six deviants was that the gap was inserted into the standard stimulus at different temporal positions: 15, 50, 100, 200, 300, or 400 ms after the onset of the deviant. In separate conditions, the gap duration was either 10 or 30 ms. The results were similar to those of previous studies using brief frequency modulations presented at different temporal positions within longer stimuli (Grimm & Schröger, 2005; Grimm et al., 2006; Timm et al., 2011). However, in our study, reliable DRNs were not uniformly absent at the latest gap positions; response detectability depended on gap duration and recording region. ERP studies examining the effect of stimulus change timing have not yet been conducted in children. The present study therefore employed the same multi-deviant paradigm, with 10- and 30-ms gaps presented at six different onset times, to examine the effects of group (children vs. adults), gap duration, and gap onset time on the DRN response. The aims were to determine how DRN amplitude

varies with gap onset time and gap duration, and whether these effects differ between children and adults.

4.2 Methods

4.2.1 Participants

The study included two groups: 15 adults (5 males) aged 23–37 years (Mean = 30.9, Standard deviation [SD]= 5.2 years) and 15 children (8 males) aged 8–12 years (Mean = 9.9, SD = 1.4 years). The adult participants were selected from the 17 adults who participated in Study 1 (Chapter 3), with two participants randomly removed to obtain a sample of 15 adults for the present study. None of the participants had a history of psychiatric, neurological, attentional, or cognitive disorders, seizures or head trauma. No family history of language or learning impairments was reported. The Auditory Processing Domains Questionnaire (APDQ; O'Hara & Mealings, 2018) was also completed by parents to screen potential difficulties in auditory processing, language, or attention. In addition, the Auditory Continuous Performance Test (ACPT; Keith, 1994) was administered to measure sustained auditory attention and exclude children with possible attention deficits.

Normal hearing levels were verified using standard audiological procedures. Hearing thresholds did not exceed 15 dB HL at octave frequencies from 250 to 8000 Hz (American National Standards Institute [ANSI], 2010). Middle ear function was also assessed to ensure normal tympanometric peak pressure (between -100 and +50 daPa) in both ears (Gelfand, 2016), using a tympanometer. The University of Ottawa Research Ethics Board approved the study (file number # H-07-24-10412). Written informed consent was obtained from the parents or guardians of all child participants prior to the experiment. Children received a \$20 gift card as a token of appreciation for their participation. Adult participants volunteered and provided written informed consent.

4.2.2 Procedure and stimuli

Participants in both groups sat in a sound-attenuated and electrically shielded booth. They were instructed to watch a silent, subtitled video of their choice and to ignore the auditory stimuli. A multi-deviant paradigm was used to present the standard and deviant stimuli. The standard stimulus was a 500 ms broadband white noise burst presented at a peak intensity of 80 dB SPL,

with an instantaneous rise- and fall-time. The broadband noise ranged in frequency from near 0 Hz to 22 kHz, although the frequency response of the earphones did attenuate its amplitude, especially for frequencies below 50 Hz and those above 4 kHz. A wide bandwidth was used because stimuli with broader frequency bandwidths have gap-detection thresholds that are similar in children and adults (Buss et al., 2014; Buss et al., 2017). Moreover, the use of broadband noise reduced the likelihood of detecting spectral splatter caused by the abrupt onset and offset of the stimulus (Brown & Nicholls, 1997). The to-be-ignored irrelevant auditory stimuli were delivered binaurally via insert-earphones (E-A-RTONE 3A). Sound intensity was calibrated using a Bruel and Kjaer Type 2235 sound level meter and a 2 cm³ coupler (GRAS RA0045-S1).

In the multi-deviant paradigm, six deviants were created by inserting a silent interval (a “gap”) at different temporal positions within the standard. The gaps occurred at either 15, 50, 100, 200, 300, or 400 ms after stimulus onset and had an instantaneous onset and offset. The standards and deviants alternated; the probability of occurrence of each stimulus type was therefore .50. However, the probability of occurrence of each of the six different gap deviants was .083. The deviants were ordered pseudo-randomly such that in an array of 12 alternating standards and deviants, each of the six deviants was presented once. Their order of presentation within the array was randomized. The last deviant in the array and the first in the subsequent array could not be the same. Moreover, the same random order of presentation of deviants within an array could not occur a second time. The inter-stimulus interval (offset-to-onset) was 400 ms. The sequence lasted about 14 minutes and included 472 standards (including 10 standards presented to establish the memory trace before the alternating standard and deviant stimuli) and 77 of each deviant. Gap duration was either 10 or 30 ms, in separate conditions. The two multi-deviant conditions were each repeated three times in random order. A brief rest period was provided between sequences. The timing and presentation of all stimuli were controlled by a computer running E-Prime® software (Psychology Software Tools, version 2.0).

4.2.3 Electrophysiological recording and analyses

The electroencephalography (EEG) was recorded from 30 active silver-silver chloride electrodes placed according to the International 10-10 system (Pivik et al., 1993) and attached to an electrode cap. Electrodes were positioned on the midline and lateral areas of the frontal, central, parietal, temporal, and occipital regions. Additional electrodes were placed on the left and right

mastoids. The reference electrode was located on the tip of the nose. Vertical eye movement and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. Inter-electrode impedance was below 25 k Ω . The continuous EEG activity was sampled at 500 Hz and stored on disk.

The data were subsequently analysed using Brain Products' Analyzer 2.3 software. The EEG and EOG data were digitally filtered with a band-pass filter from 0.3 to 20 Hz. The continuous data were visually inspected to identify channels contaminated by high-amplitude noise. These channels were replaced by interpolating the data of the surrounding electrode sites (Perrin et al., 1989). A maximum of four channels was replaced by interpolation. Independent Component Analysis (ICA; Makeig et al., 1996) was used to identify eye movement and blink artifacts that were statistically independent of the EEG activity. The vertical EOG activity was computed by subtracting activity recorded at FP1 from that at the infra-orbital ridge, whereas the horizontal EOG channel was computed by subtracting FT9 activity from that of FT10. These statistically independent components were then subtracted from the raw EEG to minimize contamination from eye movements.

The continuous EEG was partitioned into 1000-ms epochs, beginning 100 ms before stimulus onset. The average of the 100-ms pre-stimulus period served as a zero-voltage baseline, from which ERP components were measured. Each single trial epoch was then baseline-corrected to remove slow voltage "drifts". For each of the standard and deviant single trials, the mean amplitude of all data points within the baseline period was subtracted from that of all subsequent data points in the post-stimulus period. Any epoch containing activity exceeding ± 100 μ V was then rejected from further analysis. Rejections based on this criterion were relatively low because the most common type of artifact, eye blinks and movements, had already been corrected. The epochs were then sorted by electrode site, stimulus type (standard, six deviants), and condition (10 and 30 ms gap duration), and averaged.

4.2.4 Behavioural detection

A separate behavioural condition was run following completion of the ERP session. The participant was asked to attend the auditory sequence and actively detect the occurrence of the deviant gap. In this task, the standards (500-ms broadband white noise bursts) and the 10 and 30-ms duration deviants were presented in a single sequence. A silent gap was again inserted 15, 50,

100, 200, 300, or 400 ms after the onset of the deviant. The sequence thus included standards and 12 deviants (2 durations x 6 onset times). A total of 50 standards and 120 deviants (10 for each type of gap deviant) were presented in random order. The stimuli were presented with a fixed 1500 ms inter-stimulus interval (offset-to-onset) to allow participants time to respond. Participants were asked to push the right button of a mouse if they judged the stimulus to contain a gap (i.e., the deviant stimulus) and the left button if they judged it not to contain a gap (i.e., the standard stimulus). Ten practice trials were presented to ensure the participant understood the instructions.

Hits (correctly detecting the gap deviant), correct rejections (correctly detecting the standard), false positives (detecting the standard as a gap deviant), and misses (detecting the gap deviant as a standard) were computed. The hit and false-positive rates were used to compute an index of sensitivity: d' .

4.2.5 ERP Quantification and Analysis

The deviant elicited a DRN in both groups. The DRN is best observed in a difference wave computed by subtracting the ERP response to the standard from that to the deviant. The initial portions of the standard and deviant stimuli were identical up to the gap onset. Processing of the standard and deviant should thus have been very similar prior to the gap onset, so the subtraction method should have removed response activity in this period (including the P1, N1, and P2 elicited by the onset of both stimuli), leaving responses unique to each deviant (i.e., related to the processing of the gap inserted into the deviant). The subtraction method is illustrated in Figure 4.1.

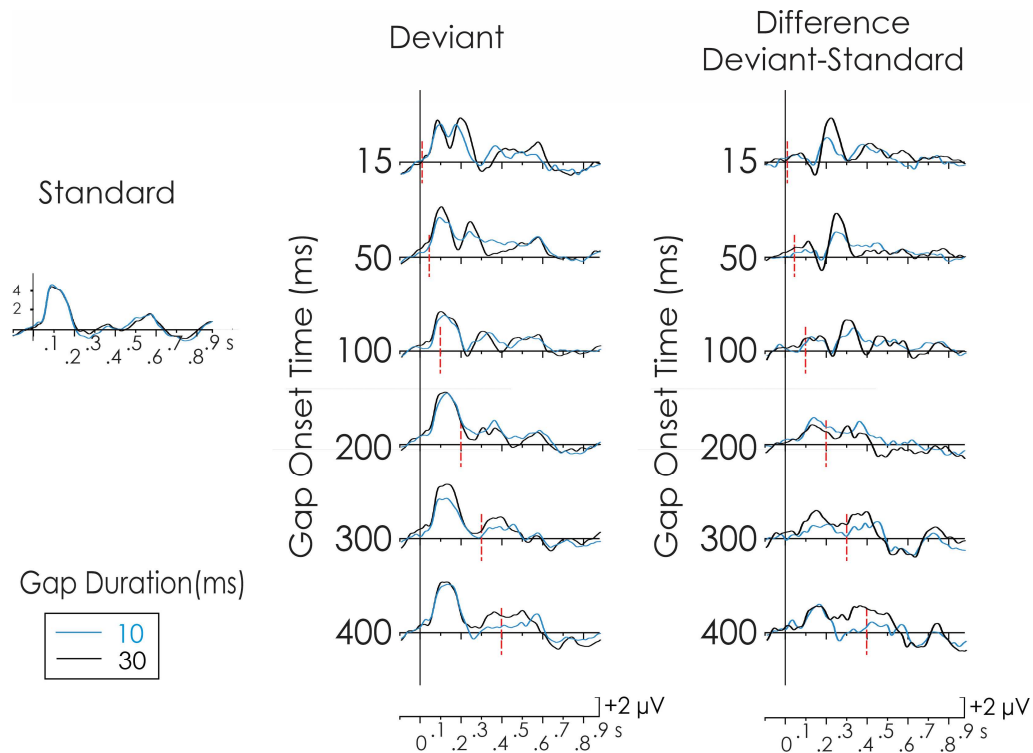


Figure 4.1 “Raw” standard (left column), deviant (middle column), and the deviant-standard difference wave (right column) in children. The standard and deviant were physically identical until the time of onset of the gap (indicated by the red vertical lines). In the left portion of the Figure, the ERPs to the standard stimulus are illustrated. The onset of the standard elicited a large positivity in the children. The middle portion illustrates the ERPs elicited by the different deviant stimuli. The deviant onset also elicited a large positivity. The right portion of the Figure illustrates the deviant-standard difference wave. Processing common to the standard and deviant should thus have been removed in the subtraction process. Thus, the large positive wave common to both the standard and deviant is removed in the difference wave, leaving processing unique to the gap deviant. The overall morphology of the difference wave was relatively similar in children and adults.

The DRN was quantified for each individual using the mean amplitude of all data points within ± 25 ms of the maximum peak identified in the grand-average difference wave for each of the six deviants in the 10- and 30-ms gap duration conditions. Their amplitudes were measured relative to the zero-voltage pre-stimulus baseline. Another common approach for quantifying the DRN is to measure the most negative peak within a predefined latency window. This approach, however, is sensitive to noise because the identified peak reflects the combined contribution of the true DRN and random fluctuations in the waveform. This becomes particularly problematic when the underlying DRN is small, as would be expected for brief gaps. The mean-amplitude measure used in the present study is less affected by this issue because positive- and negative-going drifts

within the measurement window tend to cancel each other out (Luck, 2014). A limitation of the mean-amplitude approach is that it does not readily provide a clear estimate of peak latency.

An analysis of variance (ANOVA) was used to determine whether the amplitude of the DRN varied across the deviants. To determine whether each deviant elicited a DRN, 90% confidence intervals (CIs) were computed on the amplitude of each DRN. The CIs were calculated using data only at the Fz and Cz sites, where the DRN is largest. DRN responses were considered to be present only if both the upper and lower CI limits were less than the zero-voltage pre-baseline level (i.e., a negative value). Because the DRN was expected to have a negative value, a one-tailed test of significance ($p < .05$) was used for the CI testing.

Separate ANOVAs were then conducted at frontal (F3, Fz, F4) and central (C3, Cz, C4) regions of interest (ROIs) for the DRN data. Each was a three-way ANOVA with one between-group factor (children, adults) and two repeated-measures factors: gap onset time (15, 50, 100, 200, 300, 400 ms) and gap duration (10, 30 ms). When the main effect of stimulus onset time was significant, the Holm post hoc procedure was used to identify significant differences among the six onset positions. When a significant interaction was found, the source of the interaction was identified using simple main-effects tests. All statistical analyses were conducted using JASP software, version 0.6 (JASP, 2026).

4.3 Results

4.3.1 Electrophysiological data

4.3.1.1 Standard ERP

One of the purposes of this study was to investigate how the processing of different gap durations affected the DRN. As mentioned previously, it was assumed that whatever differences emerged were a result of differential processing of the deviant stimulus. The DRN was, however, measured in a deviant-standard difference wave. It is thus possible that differences in the processing of the standard rather than the deviant could account for the MMN effects. Figure 4.2 illustrates the standard ERP waveforms at Fz and Cz in the 10- and 30-ms conditions in children and adults. In children, a large-amplitude positivity (P1) is apparent around 95 ms, with N2 peaking at approximately 285 ms. Adults, however, exhibited distinct P1, N1, P2, and N2 components, peaking around 80 ms, 100 ms, 180 ms, and 280 ms, respectively. Because different ERP

components were elicited in the two groups, separate ANOVAs were run for children and adults to compare the effects of gap duration on the processing of the standard stimulus. The separate ANOVAs revealed no significant differences in amplitude between conditions at either frontal or central ROIs, for either group, $F < 1$ in all cases.

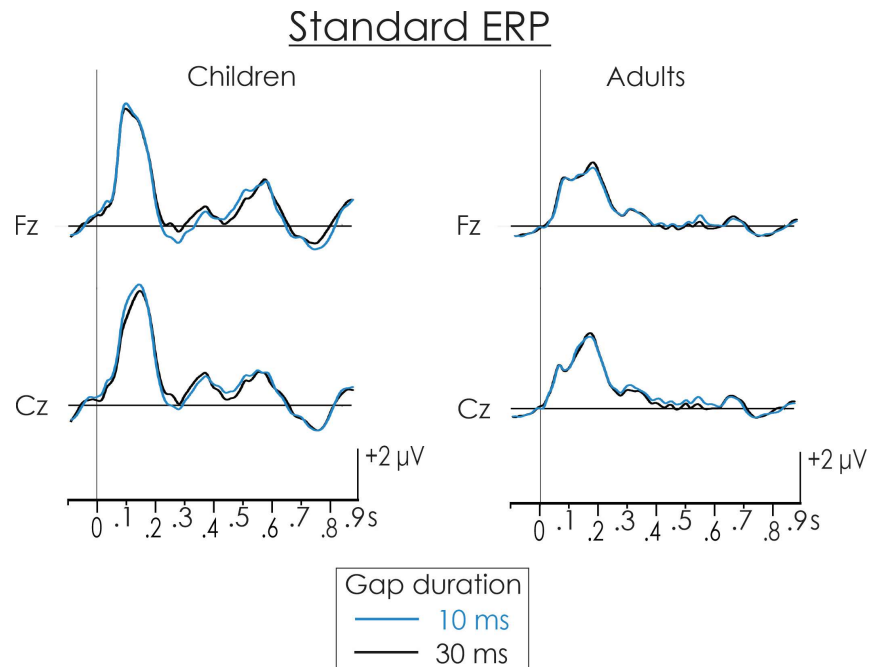


Figure 4.2 Standard ERP waveforms for the 10-ms and 30-ms gap duration conditions in children and adults. The amplitudes of the negative and positive deflections did not differ significantly within each condition.

4.3.1.2 *Deviant-Standard ERPs*

4.3.1.2.1 Deviant-Related Negativity (DRN)

The grand averages of the deviant-standard difference waves for 10- and 30-ms gaps in children and adults are illustrated in Figure 4.3. A fronto-central maximum DRN was elicited approximately 100–150 ms after gap onset. It was much reduced in children, especially for gaps occurring 200 ms and later after the onset of the deviant. This was confirmed by the CI testing. In children, a significant DRN was only elicited for 30-ms duration deviants occurring at 15 and 50 ms after onset, at both Fz and Cz ($p < .05$). In contrast, CI testing in adults revealed a significant DRN for all deviants except for the 10-ms gaps occurring at 300 at Fz and Cz, and for the 10- and 30-ms duration gaps occurring at 400 ms at Cz.

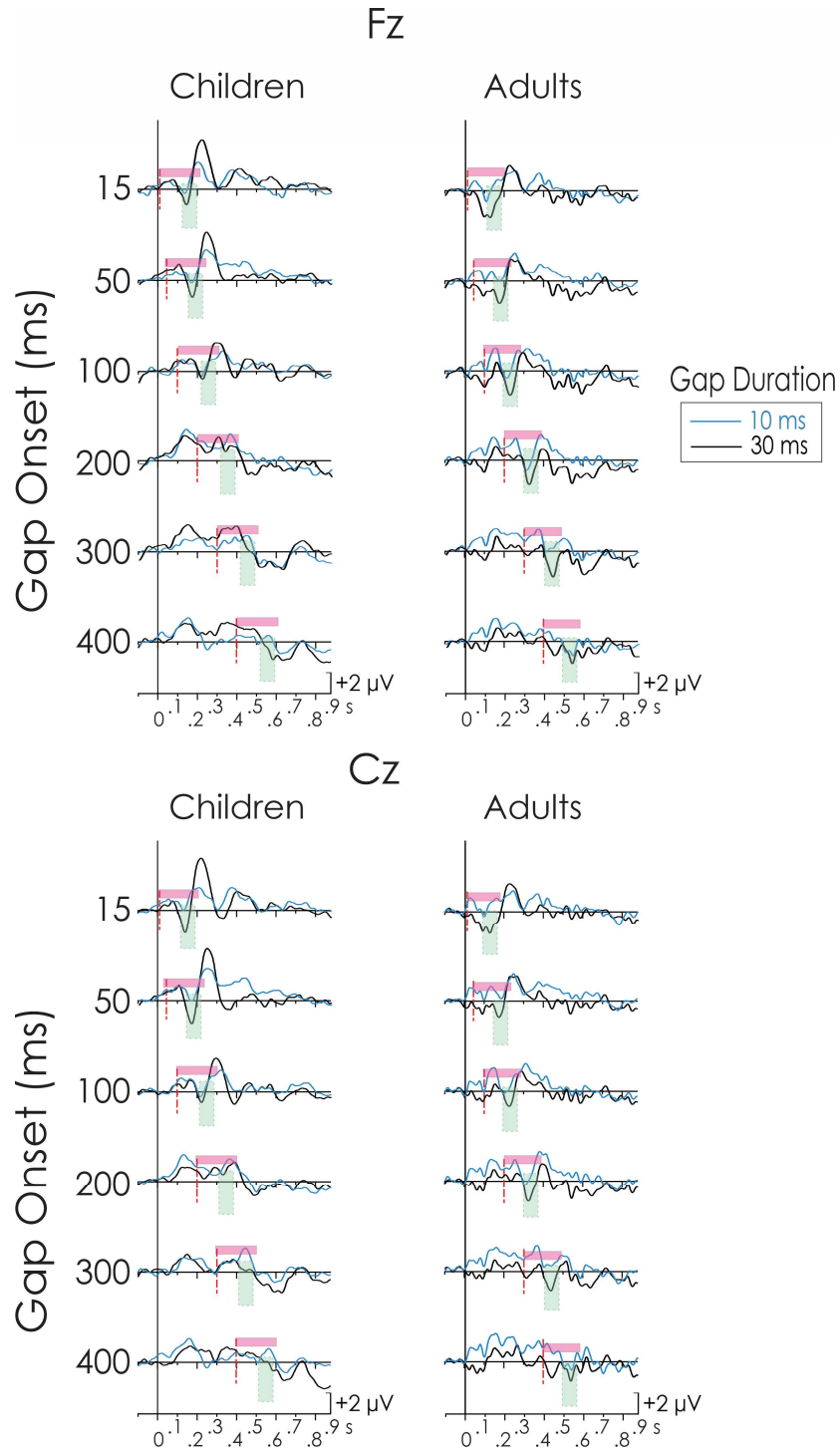


Figure 4.3 Deviant–standard difference waves for children and adults at Fz and Cz, as a function of gap duration and gap onset time. The red dashed line indicates the time of onset of the gap. For children, a clear DRN is visible around 100–150 ms following gaps occurring 15 to 100 ms after the onset of the deviant, although CI testing confirmed a significant DRN only for the 30-ms gaps occurring at 15 and 50 ms. In adults, a clear DRN is visible for gaps occurring between 15 and 300 ms after the onset of the deviant. A subsequent positivity, P2, was larger in children than in adults. The DRN was significantly reduced when the gap duration was 10 ms.

Baseline-to-peak

DRN amplitude was measured with respect to the zero-voltage pre-stimulus baseline (i.e., baseline-to-peak). Figure 4.4 illustrates the mean (and SEs) amplitude as a function of gap duration and gap onset time.

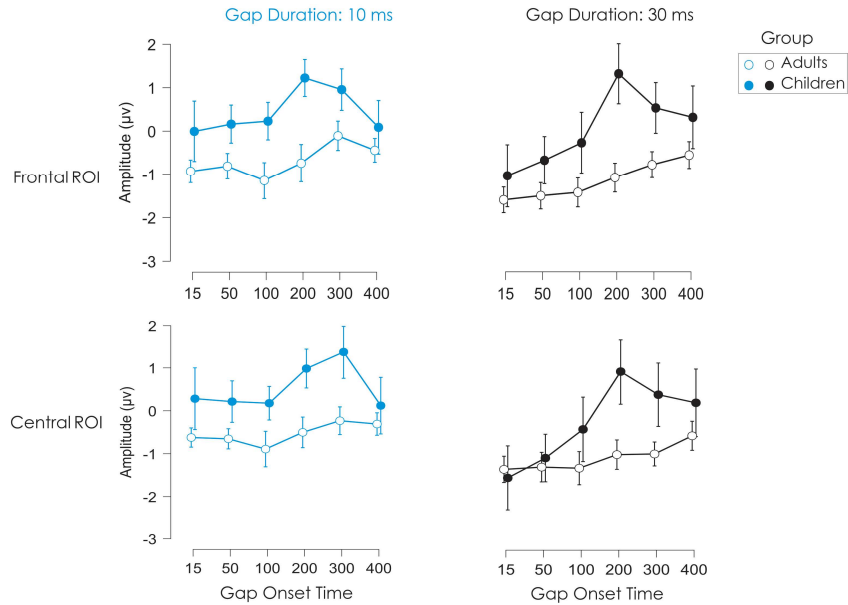


Figure 4.4 Mean baseline-to-peak DRN amplitude in children and adults for the 10- and 30-ms duration gaps as a function of gap onset time at the frontal and central ROIs. Error bars represent the standard error (SE) of the mean.

DRN amplitude was significantly lower in children, at both frontal $F(1, 28) = 9.96$, $p = .004$, $\eta^2_p = .26$, and central ROIs, $F(1, 28) = 5.82$, $p = .02$, $\eta^2_p = .17$. A significant main effect of gap duration was also observed, at both frontal, $F(1, 28) = 5.32$, $p = .03$, $\eta^2_p = .16$, and central ROIs, $F(1, 28) = 9.52$, $p = .005$, $\eta^2_p = .25$. DRN amplitude was significantly larger following the presentation of the 30-ms than following the presentation of the 10-ms duration gap. The interaction between gap duration (10, 30 ms) and group (children, adults) was not significant at either ROI, $F < 1$ in both cases. The main effect of gap onset time was significant at both frontal, $F(5, 140) = 8.42$, $p < .001$, $\eta^2_p = .23$, and central ROIs, $F(5, 140) = 6.13$, $p < .001$, $\eta^2_p = .18$. However, the interaction between group and gap onset time was also significant at both frontal, $F(5, 140) = 2.93$, $p = .02$, $\eta^2_p = .10$, and central ROIs, $F(5, 140) = 2.63$, $p = .03$, $\eta^2_p = .09$. Simple main effect testing was used to isolate the source of the interaction. For both children and adults, the main

effect of gap onset time was significant at both ROIs ($p < .01$ in both groups). Holm post-hoc tests on the children's data revealed that the DRN was significantly lower in amplitude when the gap occurred 200 ms or later than when it occurred 15 or 50 ms after stimulus onset ($p < .05$). Significantly larger DRN amplitudes were also observed at the 100-ms onset times compared to the 200-ms onset times ($p \leq .001$). At the central ROI, children showed significantly larger DRN amplitudes for the 15-, 50-, and 100-ms gap onset times compared to 200 ms ($p \leq .002$) and 300 ms ($p \leq .03$). However, no significant differences in DRN amplitude were observed across gap onset times in adults ($p > .05$). Because several studies have now shown that the MMN/DRN tends to decrease as the deviant's latency increases, multiple uncorrected tests were run to determine differences among the various gap onset times. Uncorrected t-tests were used as a follow-up procedure to further characterize differences among gap onset times.. At both frontal and central ROIs in children, DRN amplitude was significantly larger for gaps occurring at 15 and 50 ms than for gaps at 200 ms or later, particularly for the 30-ms gap duration ($p < .05$). In addition, DRN amplitude was significantly larger for a gap onset time of 100 ms compared with 200 ms ($p \leq .04$). In adults, at the frontal ROI, the DRN was significantly larger for gaps occurring between 15 and 200 ms than for gaps occurring at 300 or 400 ms ($p < .05$). A similar pattern was observed at the central ROIs, where DRN amplitude was significantly larger for gaps between 15 and 200 ms compared with 400 ms ($p < .05$).

Follow-up testing was also run to determine differences between children and adults for each gap onset time. These revealed significantly larger DRNs in adults than in children when the gap occurred between 50 and 300 ms at the frontal ROI ($p \leq .025$), and at 200 and 300 ms at the central ROI ($p = .001$ and $= .003$, respectively). Comparisons were conducted separately for each gap onset time.

Peak-to-peak DRN

The DRN was measured relative to the zero-voltage pre-stimulus baseline. In Figure 4.3, it is apparent that in some conditions, the EEG prior to the onset of the gap was not always stable. Moreover, for children, a large positivity (P2) was apparent following the DRN. This positivity may have overlapped and summed with the DRN, "pulling" it in a positive direction (i.e., reducing the amplitude of the negative-going DRN). This positivity was smaller in adults. In such cases, ERPs are often measured as peak-to-peak rather than a baseline-to-peak measure (Picton et al.,

2000). The data were therefore re-analyzed, measuring the DRN relative to the subsequent P2, which occurred at about 200 ms.

The mean peak-to-peak DRN data are presented in Figure 4.5 for the frontal and central ROIs. Group differences in the amplitude of the DRN, measured peak-to-peak, were no longer significant, $F(1, 28) \leq 1.14$. The main effect of gap duration remained significant at both frontal, $F(1, 28) = 4.72$, $p = .04$, $\eta^2_p = .14$, and central $F(1, 28) = 20.10$, $p < .001$, $\eta^2_p = .42$ ROIs. The DRN was larger for the longer 30 ms duration gap. Again, the interaction between group and gap duration was not significant, at either frontal ($F < 1$) or central ($F(1, 28) = 2.97$, $p = .10$) ROIs. The main effect of gap onset time was again significant at both frontal, $F(5, 140) = 53.61$, $p < .001$, $\eta^2_p = .66$, and central, $F(5, 140) = 39.03$, $p < .001$, $\eta^2_p = .58$ ROIs. However, the group $\times$ gap onset interaction was also significant at both frontal, $F(5, 140) = 12.37$, $p < .001$, $\eta^2_p = .31$, and central, $F(5, 140) = 9.49$, $p < .001$, $\eta^2_p = .25$, ROIs.

The sources of this interaction were similar to those observed for the baseline-to-peak measurement of the DRN. When the ANOVA was run separately for children and adults, the main effect of gap onset time was significant ($p < .01$ in both groups and ROIs). Holm post hoc comparisons, conditional on group, were conducted to determine differences in onset time. In children, at both ROIs, the DRN was significantly larger for gaps at 15 and 50 ms than for those at 100-400 ms ($p \leq .007$). The DRN for the gap occurring at 100 ms was also significantly larger than for those occurring from 200 to 400 ms ($p \leq .002$), and the DRN for the gap occurring at 200 ms was significantly larger than for that occurring at 400 ms ($p \leq .04$). In adults, at the frontal ROI, the DRN was significantly larger for gaps occurring from 15 to 200 ms than for those occurring at either 300 or 400 ms. The DRN was significantly reduced when the gap occurred at 400 compared to 300 ms ($p \leq .02$). At the central ROI, results were essentially identical. As noted above, the overall group differences were not significant. However, when group comparisons were made for each gap onset time, some differences did emerge. The amplitude of the DRN was significantly larger for children when the gap occurred at 50 ms ($p = 0.02$) but significantly smaller when it occurred at 200 and 300 ms ($p = 0.02$).

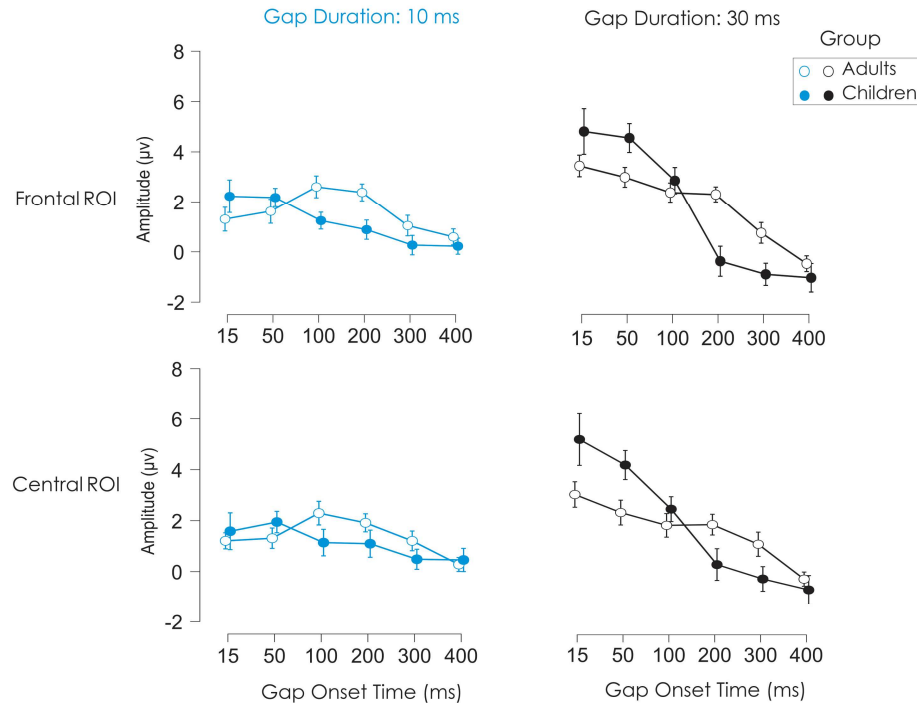


Figure 4.5 Mean peak-to-peak DRN amplitude in children and adults for the 10- and 30-ms duration gaps as a function of gap onset time at the frontal and central ROIs. Error bars represent the standard error (SE) of the mean.

4.3.2 Behavioural data

Behavioural performance was measured using the signal detection measure d' , based on both the probability of a correct detection of the deviant containing a gap (i.e., a hit) and the probability of a standard being incorrectly detected as containing a gap (i.e., a false positive).

Figure 4.6 illustrates the mean d' for the 10- and 30-ms duration gaps as a function of gap onset time. As may be observed, d' values were overall very high, although they were lower in children than in adults. A 3-way ANOVA was used to determine the effects of gap duration and gap onset time for the two groups. Adults performed significantly better than children, $F(1, 28) = 9.81$, $p = .004$, $\eta^2_p = .26$. The main effect of gap duration was not significant, $F(1, 28) = 1.29$, $p = .26$, $\eta^2_p = .04$. On the other hand, the main effect of gap onset time was significant, $F(5, 140) = 6.44$, $p < .001$, $\eta^2_p = .19$. The interaction between gap duration and gap onset time was, however, also significant, $F(5, 140) = 14.56$, $p < .001$, $\eta^2_p = .34$. For the 30-ms gap duration, d' at the 15-ms and 400-ms onsets was significantly lower than all other onset times ($p \leq .03$). The interactions between group and gap duration ($F(1,28) = 0.06$, $p = .80$), as well as, group and gap onset time

($F(5,140) = 1.28$, $p = .28$), were not significant. Pearson correlations were computed between mean d' values and DRN amplitudes (baseline-to-peak and peak-to-peak) for each condition within each group. No significant correlations were found at either Fz or Cz for the two gap durations ($p > .05$ for all comparisons).

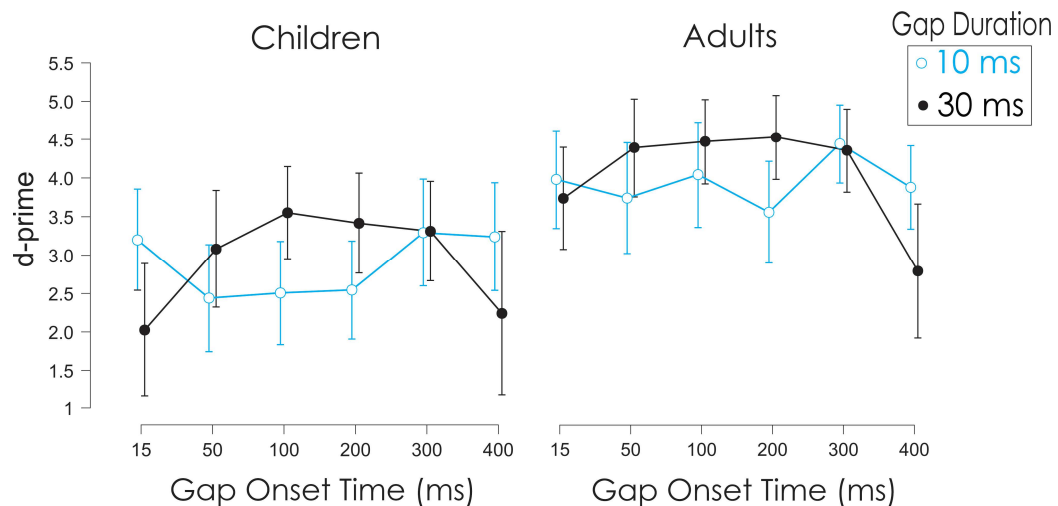


Figure 4.6 Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time in children and adults. Error bars represent the SE of the mean. As may be observed, d' was quite high overall regardless of gap duration and the onset time of the gap. Performance was nevertheless significantly poorer in children.

4.4 Discussion

The present study investigated the influence of gap duration and onset time on the amplitude of the DRN in children and adults. The standard was a 500-ms-duration broadband noise burst. The deviant was created by inserting a 10- or 30-ms gap deviant at 6 temporal positions, ranging from 15 to 400 ms after stimulus onset.

The morphology of the standard ERP differed significantly between children and adults. In adults, the usual P1-N1-P2 complex was observed, although N1 amplitude in adults was very small. This was probably due to the rapid stimulus presentation rate: each stimulus occurred 400 ms after the previous stimulus offset (Pereira et al., 2014). In children, a large positivity (rather than negativity) was observed with a latency of 80-100 ms. These developmental differences are consistent with previous research (Bishop et al., 2007; Mahajan & McArthur, 2012; Shafer et al., 2015; Sussman et al., 2008).

The DRN was measured in a deviant-standard difference wave. It was assumed that differences in DRN between children and adults were due to differential processing of the deviant. The response to the onset of the standard, however, was very different in children than in adults. It is thus possible that group differences in the DRN might partly result from differences in how the standard stimuli are processed. However, as is apparent in Figure 4.1, this was unlikely. The onset of both the standard and deviant elicited ERPs was initially quite similar, even though the waveform morphology was different between children and adults. The deviant-standard subtraction procedure removed processing that was common to both the standard and the deviant.

In this study, the overall DRN amplitude in children was significantly lower than in adults. This finding is similar to that of previous ERP research in children that has reported that the MMN is often less negative-going (or more positive-going) in younger children (Bishop et al., 2011; Oades et al., 1997). The MMN in children, however, undergoes dramatic changes with age. In early childhood, MMN is often recorded as a positivity, rather than a negativity. By around 10 years of age, the MMN in children and adults is quite similar (Ahmmed et al., 2008; Leppänen et al., 2004; Maurer et al., 2003; Näätänen et al., 2014; Putkinen et al., 2014; Shafer et al., 2010). The mean age of the children in the present study was about 10 years, yet their DRNs were reduced in amplitude compared to those of the adults. However, the child group ranged from 8 to 12 years of age, and individual differences related to age may have contributed to variability in DRN amplitude and morphology within this relatively small sample ($n = 15$). This may also be due to the nature of the deviant stimulus. Most studies of acoustic change in children have employed frequency, intensity, duration, and complex speech or non-speech deviants (Ahmmed et al., 2008; Gomes et al., 1999; Leppänen et al., 2004; Linnavalli et al., 2022; Lohvansuu et al., 2013; Lovio et al., 2009; Maurer et al., 2003; Shafer et al., 2010; Themas et al., 2025). These will elicit a “purer” MMN. A gap deviant was employed in the present study. As with the standard, the abrupt gap onset elicits a positivity in children but a negativity in adults. These onset responses overlap and summate with the MMN. The composite DRN might therefore be more positive-going (or less negative-going) in our children. Also, as is apparent in Figure 4.3, a P2 occurring after the DRN was larger in children than in adults. This may explain why when the DRN was measured from baseline to peak, it was smaller in 10-year-old children, while when it was measured peak-to-peak (from the negativity to the following positivity), there were no differences in its amplitude between

children and adults. The peak-to-peak results are very similar to those reported in other studies (Gomot et al., 2000; Kraus et al., 1993; Shafer et al., 2000).

The effects of gap duration on DRN amplitudes have not been tested in children. Many previous studies have shown that in adults, the amplitude of the DRN increases with increasing gap duration (Alain et al., 2004; Bertoli et al., 2001; Duda-Milloy et al., 2019; Duda et al., 2020; Duda et al., 2025). This finding was replicated in the adult group in the present study and was also observed in children. In contrast, in the behavioural task, detection of the gap was poorer when its duration was 10 rather than 30 ms in most conditions; the difference was not statistically significant in either group.

One of the aims of the present study was to determine the influence of the temporal position of the gap deviant on the DRN. Temporal integration theory posits that sound features are first extracted and then integrated with preceding and subsequent features into a single perceptual unit, and stored in a very limited-capacity sensory memory (Weise et al., 2023; Yabe et al., 1997). The duration of the TWI has not been extensively studied in children. In the present study, the DRN decreased in amplitude as the onset time of the gap deviant was delayed in both children and adults. In children, DRN was apparent only for the 30-ms gaps occurring at 15 and 50 ms after stimulus onset, at both Fz and Cz. In adults, DRN was observed for most conditions, except for the 10-ms gap at 300 ms at both Fz and Cz, and the 10- and 30-ms gaps at 400 ms at Cz. This is similar to what other researchers have observed when brief frequency modulations of the standard occur at these times (Grimm et al., 2006; Grimm & Schröger, 2005; Grimm et al., 2004; Hoonhorst et al., 2012; Timm et al., 2011; Weise et al., 2010; Weise et al., 2007; Weise et al., 2011). The TWI was therefore different in children. These findings may suggest that the temporal window for auditory integration is more limited in children aged 8 to 12. However, the DRN does not directly measure sensory-memory capacity. The behavioural performance of both groups showed a different pattern from the DRN data, with relatively good gap detection even when the DRN was reduced or absent. In children, a DRN was not visible for gap deviants occurring at 200 ms and later. The DRN was much reduced in adults when the gap was inserted at 300 ms and 400 ms. The amplitude of the MMN/DRN is much reduced or absent when a deviant cannot be perceived. It would therefore be expected that when the gap was inserted at 200 ms, or later for children and at 300 or 400 ms for adults, performance would be very poor or at chance levels when asked to actively detect the gap. This was not the case. The signal detection measure d' was used to assess the perceptibility of the

gap. Both groups could easily detect the gap, even if it was inserted well after the onset of the deviant stimulus when a DRN was reduced or absent. Moreover, there were no significant correlations between the amplitude of the DRN and behavioural performance. To our knowledge, no previous study has evaluated DRN/MMN responses to late-onset changes in children. However, studies in adults have shown that stimulus changes with late onsets (after 300 ms) are also easily detected, even when MMN amplitudes are reduced or absent (Hoonhorst et al., 2012; Timm et al., 2011). The differences between electrophysiological and behavioural findings may be attributed to variations in task demands and stimulus presentation procedures. During ERP recordings, participants were instructed to ignore the auditory stimuli and attend to a silent video. Stimulus change was thus detected automatically without the need to direct attention to the auditory channel. In the Näätänen model, such automatic detection of change results from a mismatch between the features of the incoming stimulus and what is stored in sensory memory. In contrast, during the behavioural task, participants were required to actively attend to the auditory sequence to determine whether the current stimulus contained a gap. This type of comparison involves a different working memory system. The capacity of working memory is much larger and longer-lasting than that of sensory memory. Importantly, working memory requires active attention and rehearsal to prevent the fading of previous events (Baddeley, 2007; Baddeley & Hitch, 1974). Weise et al. (2023) suggest that while the integration and storage capacity of auditory sensory memory is very limited, this can be overcome through the involvement of working memory processes. This does come at a cost. The listener must actively attend to the auditory stream.

Children generally demonstrated weaker behavioural performance compared to adults. Since the peak-to-peak DRN amplitudes did not differ significantly between groups, these behavioural differences are likely due to factors beyond sensory memory capacity. As discussed above, the behavioural task required active attention and working memory, whereas the ERP task measured automatic responses during passive listening. These greater task demands may have affected children more than adults. Age-related differences in attentional capacity, working memory, neural maturation, and internal noise may therefore have contributed to the poorer behavioural performance observed in children. More specifically, in children, identifying silent gaps was more difficult when the gaps occurred near the onset or offset of the stimulus (i.e., at 15 and 400 ms) with a gap duration of 30 ms. In adults, a similar pattern was observed only for the late gap onset time (400 ms). For the 10-ms gap, the patterns of performance changes were

inconsistent across groups. These findings are consistent with previous studies (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999), which generally report that gap detection is more challenging when the gap occurs near the beginning or end of the stimulus rather than in the middle.

The results of the present study, together with previous findings, suggest that the duration of the TWI is influenced by factors such as maturation, attention, and learning (Grimm & Schröger, 2005; Kharlamov et al., 2011; Rüsseler et al., 2001). Many critical temporal cues in speech, such as voice onset time, the brief interval between the release of a stop consonant and the onset of voicing, depend on millisecond-level temporal precision (Phillips, 1998). The current findings support the notion that automatic auditory gap detection is more efficient near the stimulus onset, especially in children. Future research might examine whether TWI is more strongly affected in individuals with language and/or listening difficulties. Such investigations could provide valuable insights into the contribution of auditory sensory memory to speech and language development in clinical populations. At the same time, several design constraints may limit the generalizability of these results: The paradigm included limited conditions (two gap durations and six gap onset times) in a broadband noise, so the observed onset-time constraint on DRN may not transfer directly to speech or other complex sounds. In future studies, the stimulus set could be expanded by sampling more gap durations and more finely spaced onset times, allowing better estimation of the boundary at which change detection becomes unreliable in each age group. Replicating the design with speech or speech-like stimuli would also strengthen links to real-world listening demands. In addition, because ERPs were recorded during passive listening while behavioural performance was measured during an active task, the two measures may reflect partially different mechanisms (automatic change detection vs attention-supported detection), which likely contributed to the weak correspondence between behavioural sensitivity and DRN amplitude. To better disentangle sensory memory limits from attention and working memory influences, future studies could manipulate task demands during EEG (passive vs active attention) and evaluate whether top-down engagement can “rescue” responses to late-occurring deviants. An additional limitation was that the behavioural task was always administered after completion of the ERP session. Although participants were given a break between the ERP and behavioural tasks, fatigue or reduced task engagement following the EEG session cannot be excluded as a possible contributor to behavioural performance, particularly to the lower performance observed in

children. Future studies should conduct behavioural and electrophysiological assessments in separate sessions. Finally, extending the paradigm to clinical or at-risk groups (e.g., children with listening difficulties or language/reading challenges) could clarify whether atypical auditory development is characterized by a narrower window of automatic change detection and could help establish the DRN as a potential objective marker of temporal processing constraints.

4.5 Conclusion

The present study showed that the longer gaps elicited larger DRNs, and DRN amplitude generally decreased as the gap occurred later within the stimulus. This effect differed between children and adults, as shown by the significant group $\times$ gap onset time interaction at both frontal and central ROIs. In children, reliable DRNs were observed for fewer gap onset times, whereas in adults, DRNs were present in most conditions and were reduced or absent mainly at later onset times. Behavioural gap detection was also affected by age and gap onset time. Behavioural performance was high overall, but particularly reduced for 30-ms gaps occurring at 15 and 400 ms after stimulus onset. Behavioural sensitivity was not correlated with DRN amplitude. This suggests that behavioural detection and passive DRN responses reflect partly different processes. These differences may be related to variations in task demands and stimulus presentation procedures, including the greater involvement of attention, working memory, and decision-related processes in the behavioural task, although these processes were not directly examined in the present study. Taken together, these findings suggest that good behavioural gap detection can be maintained even when the passive DRN response is reduced or absent, particularly for gaps occurring later within the stimulus.

Conflict of interest

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

Acknowledgements

The authors thank William Khawam for his assistance with data collection. We are also grateful to the children, their parents/guardians, and the adult participants for their time and contribution to this study.

References

- Ahmed, A. U., Clarke, E. M., & Adams, C. (2008). Mismatch negativity and frequency representational width in children with specific language impairment. *Developmental Medicine & Child Neurology*, 50(12), 938-944.
- Alain, C., McDonald, K. L., Ostroff, J. M., & Schneider, B. (2004). Aging: a switch from automatic to controlled processing of sounds? *Psychology and aging*, 19(1), 125.
- Alain, C., Woods, D. L., & Ogawa, K. H. (1994). Brain indices of automatic pattern processing. *Neuroreport*, 6(1), 140-144.
- Amaral, M., Casali, R. L., Boscariol, M., Lunardi, L. L., Guerreiro, M. M., & Colella-Santos, M. (2015). Temporal auditory processing and phonological awareness in children with benign epilepsy with centrotemporal spikes. *BioMed Research International*, 2015(1), 256340.
- American National Standards Institute. (2010). *American national standard specification for audiometers*. American National Standards Institute New York.
- Arnott, S. R., & Alain, C. (2002). Effects of perceptual context on event-related brain potentials during auditory spatial attention. *Psychophysiology*, 39(5), 625-632.
- Baddeley, A. (2007). *Working memory, thought, and action*. Oxford University Press.
- Baddeley, A. D., & Hitch, G. J. (1974). Working Memory. In G. A. Bower (Ed.), *Recent Advances in Learning and Motivation* (Vol. 8, pp. 47-89). Academic Press. [https://doi.org/http://dx.doi.org/10.1016/s0079-7421\(08\)60452-1](https://doi.org/http://dx.doi.org/10.1016/s0079-7421(08)60452-1)
- Bertoli, S., Heimberg, S., Smurzynski, J., & Probst, R. (2001). Mismatch negativity and psychoacoustic measures of gap detection in normally hearing subjects. *Psychophysiology*, 38(2), 334-342.
- Bishop, D. V., Hardiman, M., Uwer, R., & Von Suchodoletz, W. (2007). Maturation of the long-latency auditory ERP: step function changes at start and end of adolescence. *Developmental Science*, 10(5), 565-575.
- Bishop, D. V., Hardiman, M. J., & Barry, J. G. (2011). Is auditory discrimination mature by middle childhood? A study using time-frequency analysis of mismatch responses from 7 years to adulthood. *Developmental Science*, 14(2), 402-416.
- Bizley, J. K., & Cohen, Y. E. (2013). The what, where and how of auditory-object perception. *Nature Reviews Neuroscience*, 14(10), 693-707.
- Braga, B. H. C., Pereira, L. D., & Dias, K. Z. (2015). Normality tests of temporal resolution: random gap detection test and gaps-in-noise. *Revista CEFAC*, 17, 836-846.
- Brown, S., & Nicholls, M. E. (1997). Hemispheric asymmetries for the temporal resolution of brief auditory stimuli. *Perception & Psychophysics*, 59, 442-447.
- Bruneau, N., & Gomot, M. (1998). Auditory evoked potentials (N1 wave) as indices of cortical development. In *Neuroimaging in child neuropsychiatric disorders* (pp. 113-123). Springer.

- Bruneau, N., Roux, S., Guerin, P., Barthelemy, C., & Lelord, G. (1997). Temporal prominence of auditory evoked potentials (N1 wave) in 4-8-year-old children. *Psychophysiology*, 34(1), 32-38.
- Budd, T. W., Barry, R. J., Gordon, E., Rennie, C., & Michie, P. T. (1998). Decrement of the N1 auditory event-related potential with stimulus repetition: habituation vs. refractoriness. *International journal of psychophysiology*, 31(1), 51-68.
- Buss, E., Hall III, J. W., Porter, H., & Grose, J. H. (2014). Gap detection in school-age children and adults: Effects of inherent envelope modulation and the availability of cues across frequency. *Journal of Speech, Language, and Hearing Research*, 57(3), 1098-1107.
- Buss, E., Porter, H. L., Hall III, J. W., & Grose, J. H. (2017). Gap detection in school-age children and adults: Center frequency and ramp duration. *Journal of Speech, Language, and Hearing Research*, 60(1), 172-181.
- Čeponien, R., Cheour, M., & Näätänen, R. (1998). Interstimulus interval and auditory event-related potentials in children: evidence for multiple generators. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 108(4), 345-354.
- Čeponien, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870-882.
- Cowan, N. (1984). On short and long auditory stores. *Psychological bulletin*, 96(2), 341-370.
- Davies, P. L., Chang, W.-P., & Gavin, W. J. (2010). Middle and late latency ERP components discriminate between adults, typical children, and children with sensory processing disorders. *Frontiers in integrative neuroscience*, 4, 593.
- Desjardins, R. N., Trainor, L. J., Hevenor, S. J., & Polak, C. P. (1999). Using mismatch negativity to measure auditory temporal resolution thresholds. *Neuroreport*, 10(10), 2079-2082.
- Diedler, J., Pietz, J., Bast, T., & Rupp, A. (2007). Auditory temporal resolution in children assessed by magnetoencephalography. *Neuroreport*, 18(16), 1691-1695.
- Duda-Milloy, V., Tavakoli, P., Campbell, K., Benoit, D. L., & Koravand, A. (2019). A time-efficient multi-deviant paradigm to determine the effects of gap duration on the mismatch negativity. *Hearing Research*, 377, 34-43.
- Duda, V., Augereau, T. M., Campbell, K., & Koravand, A. (2025). Does masking alter the mismatch negativity response to gaps? *Brain research*, 149651.
- Duda, V., Campbell, K., & Koravand, A. (2020). Event-related potentials following gaps in noise: The effects of the intensity of preceding noise. *Brain research*, 1748, 147078.
- Eggermont, J. (1995). Neural correlates of gap detection and auditory fusion in cat auditory cortex. *Neuroreport*, 6(12), 1645-1648. Fitzgerald, K., & Todd, J. (2020). Making sense of mismatch negativity. *Frontiers in psychiatry*, 11, 468.
- Forrest, T., & Green, D. M. (1987). Detection of partially filled gaps in noise and the temporal modulation transfer function. *The Journal of the Acoustical Society of America*, 82(6), 1933-1943.

- Gay, J. D., Rosen, M. J., & Huyck, J. J. (2020). Effects of Gap Position on Perceptual Gap Detection Across Late Childhood and Adolescence. *Journal of the Association for Research in Otolaryngology*, 21, 243-258.
- Gelfand, S. A. (2016). *Essentials of audiology* (Fourth ed.). Thieme.
- Giannella Samelli, A., & Schochat, E. (2008). The gaps-in-noise test: gap detection thresholds in normal-hearing young adults. *International Journal of Audiology*, 47(5), 238-245.
- Gomes, H., Sussman, E., Ritter, W., Kurtzberg, D., Cowan, N., & Vaughan Jr, H. G. (1999). Electrophysiological evidence of developmental changes in the duration of auditory sensory memory. *Developmental psychology*, 35(1), 294.
- Gomot, M., Giard, M.-H., Roux, S., Barthélémy, C., & Bruneau, N. (2000). Maturation of frontal and temporal components of mismatch negativity (MMN) in children. *Neuroreport*, 11(14), 3109-3112.
- Grimm, S., Roeber, U., Trujillo-Barreto, N. J., & Schröger, E. (2006). Mechanisms for detecting auditory temporal and spectral deviations operate over similar time windows but are divided differently between the two hemispheres. *Neuroimage*, 32(1), 275-282.
- Grimm, S., & Schröger, E. (2005). Pre-attentive and attentive processing of temporal and frequency characteristics within long sounds. *Cognitive Brain Research*, 25(3), 711-721.
- Grimm, S., Widmann, A., & Schröger, E. (2004). Differential processing of duration changes within short and long sounds in humans. *Neuroscience letters*, 356(2), 83-86.
- Gyldenkerne, P., Dillon, H., Sharma, M., & Purdy, S. C. (2014). Attend to this: The relationship between auditory processing disorders and attention deficits. *Journal of the American Academy of Audiology*, 25(07), 676-687.
- Hecke, P., Vermiglio, A. J., Bulla, E., Velappan, K., & Fang, X. (2018). The relationship between random gap detection and hearing in noise test performances. *Journal of the American Academy of Audiology*, 29(10), 948-954.
- Hoonhorst, I., Deltenre, P., Markessis, E., Collet, G., Martin, X. P., & Colin, C. (2012). Evidence for a dual versus single origin of the MMNs evoked by cued versus cueless deviants. *Clinical Neurophysiology*, 123(8), 1561-1567.
- JASP Team. (2026). *JASP* (Version 0.98.1) [Computer software]. <https://jasp-stats.org/>
- Keith, R. W. (1994). *ACPT: Auditory continuous performance test*. Psychological Corporation.
- Kharlamov, V., Campbell, K., & Kazanina, N. (2011). Behavioral and electrophysiological evidence for early and automatic detection of phonological equivalence in variable speech inputs. *Journal of cognitive neuroscience*, 23(11), 3331-3342.
- Kraus, N., McGee, T., Micco, A., Sharma, A., Carrell, T., & Nicol, T. (1993). Mismatch negativity in school-age children to speech stimuli that are just perceptibly different. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 88(2), 123-130.
- Lee, C.-Y., Yen, H.-l., Yeh, P.-w., Lin, W.-H., Cheng, Y.-Y., Tzeng, Y.-L., & Wu, H.-C. (2012). Mismatch responses to lexical tone, initial consonant, and vowel in Mandarin-speaking preschoolers. *Neuropsychologia*, 50(14), 3228-3239.

- Leppänen, P. H., Guttorm, T. K., Pihko, E., Takkinen, S., Eklund, K. M., & Lyytinen, H. (2004). Maturational effects on newborn ERPs measured in the mismatch negativity paradigm. *Experimental Neurology*, 190, 91-101.
- Linnavalli, T., Lahti, O., Törmänen, M., Tervaniemi, M., & Cowley, B. U. (2022). Children's inhibition skills are associated with their P3a latency—results from an exploratory study. *Behavioral and Brain Functions*, 18(1), 13.
- Lohvansuu, K., Hämäläinen, J. A., Tanskanen, A., Bartling, J., Bruder, J., Honbolygó, F., Schulte-Körne, G., Démonet, J. F., Csépe, V., & Leppänen, P. H. (2013). Separating mismatch negativity (MMN) response from auditory obligatory brain responses in school-aged children. *Psychophysiology*, 50(7), 640-652.
- Lovio, R., Pakarinen, S., Huotilainen, M., Alku, P., Silvennoinen, S., Näätänen, R., & Kujala, T. (2009). Auditory discrimination profiles of speech sound changes in 6-year-old children as determined with the multi-feature MMN paradigm. *Clinical Neurophysiology*, 120(5), 916-921.
- Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). MIT press.
- Mahajan, Y., & McArthur, G. (2012). Maturation of auditory event-related potentials across adolescence. *Hearing Research*, 294(1-2), 82-94.
- Makeig, S., Jung, T.-P., Ghahremani, D., & Sejnowski, T. J. (1996). Independent component analysis of simulated ERP data. *Institute for Neural Computation, University of California: technical report INC-9606*.
- Martin, K. C., DeMarco, A. T., Dyslin, S. M., & Turkeltaub, P. E. (2025). Rapid auditory and phonemic processing relies on the left planum temporale. *Cortex*.
- Maurer, U., Bucher, K., Brem, S., & Brandeis, D. (2003). Development of the automatic mismatch response: from frontal positivity in kindergarten children to the mismatch negativity. *Clinical Neurophysiology*, 114(5), 808-817.
- Muller-Gass, A., Stelmack, R. M., & Campbell, K. B. (2006). The effect of visual task difficulty and attentional direction on the detection of acoustic change as indexed by the mismatch negativity. *Brain research*, 1078(1), 112-130.
- Musiek, F. E., Shinn, J. B., Jirsa, R., Bamiau, D.-E., Baran, J. A., & Zaida, E. (2005). GIN (Gaps-In-Noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear and hearing*, 26(6), 608-618.
- Näätänen, R. (1990). The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behavioral and brain sciences*, 13(2), 201-233.
- Näätänen, R. (1992). Event-related potentials and automatic information processing. *Attention and brain function*, 102-210.
- Näätänen, R., Jacobsen, T., & Winkler, I. (2005). Memory-based or afferent processes in mismatch negativity (MMN): A review of the evidence. *Psychophysiology*, 42(1), 25-32.

- Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: a review. *Clinical neurophysiology*, 118(12), 2544-2590.
- Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology*, 24(4), 375-425.
- Näätänen, R., Sussman, E., Salisbury, D., & L. Shafer, V. (2014). Mismatch negativity (MMN) as an index of cognitive dysfunction. *Brain topography*, 27(4), 451-466.
- Näätänen, R., Syssoeva, O., & Takegata, R. (2004). Automatic time perception in the human brain for intervals ranging from milliseconds to seconds. *Psychophysiology*, 41(4), 660-663.
- Näätänen, R., & Winkler, I. (1999). The concept of auditory stimulus representation in cognitive neuroscience. *Psychological bulletin*, 125(6), 826-859.
- Nordby, H., Roth, W. T., & Pfefferbaum, A. (1988). Event-related potentials to breaks in sequences of alternating pitches or interstimulus intervals. *Psychophysiology*, 25(3), 262-268.
- O'Hara, B., & Mealings, K. (2018). Developing the auditory processing domains questionnaire (APDQ): a differential screening tool for auditory processing disorder. *International Journal of Audiology*, 57(10), 764-775.
- Oades, R. D., Dmittenmann-Balcar, A., & Zerbin, D. (1997). Development and topography of auditory event-related potentials (ERPs): Mismatch and processing negativity in individuals 8-22 years of age. *Psychophysiology*, 34(6), 677-693.
- Omidvar, S., Campbell, K., Alain, C., Aiken, S., & Koravand, A. (2026). The Effect of the Temporal Position of a Gap on Its Automatic Detection. *European Journal of Neuroscience*, 63(12), e70585.
- Partanen, E., Torppa, R., Pykäläinen, J., Kujala, T., & Huottilainen, M. (2013). Children's brain responses to sound changes in pseudo words in a multifeature paradigm. *Clinical Neurophysiology*, 124(6), 1132-1138.
- Penner, M. (1977). Detection of temporal gaps in noise as a measure of the decay of auditory sensation. *The Journal of the Acoustical Society of America*, 61(2), 552-557.
- Pereira, D. R., Cardoso, S., Ferreira-Santos, F., Fernandes, C., Cunha-Reis, C., Paiva, T. O., Almeida, P. R., Silveira, C., Barbosa, F., & Marques-Teixeira, J. (2014). Effects of inter-stimulus interval (ISI) duration on the N1 and P2 components of the auditory event-related potential. *International journal of psychophysiology*, 94(3), 311-318.
- Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and clinical neurophysiology*, 72(2), 184-187.
- Petermann, M., Kummer, P., Burger, M., Lohscheller, J., Eysholdt, U., & Döllinger, M. (2009). Statistical detection and analysis of mismatch negativity derived by a multi-deviant design from normal hearing children. *Hearing Research*, 247(2), 128-136.
- Phillips, D., Taylor, T., Hall, S., Carr, M., & Mossop, J. (1997). Detection of silent intervals between noises activating different perceptual channels: some properties of "central"

- auditory gap detection. *The Journal of the Acoustical Society of America*, 101(6), 3694-3705.
- Phillips, D. P. (1998). Sensory representations, the auditory cortex, and speech perception. *Seminars in Hearing*, 19(04), 319-332. Phillips, D. P., Comeau, M., & Andrus, J. N. (2010). Auditory temporal gap detection in children with and without auditory processing disorder. *Journal of the American Academy of Audiology*, 21(06), 404-408.
- Picton, T. W., Bentin, S., Berg, P., Donchin, E., Hillyard, S., Johnson, R., Miller, G., Ritter, W., Ruchkin, D., & Rugg, M. (2000). Guidelines for using human event-related potentials to study cognition: recording standards and publication criteria. *Psychophysiology*, 37(2), 127-152.
- Picton, T. W., Hillyard, S. A., Krausz, H. I., & Galambos, R. (1974). Human auditory evoked potentials. I: Evaluation of components. *Electroencephalography and clinical neurophysiology*, 36, 179-190.
- Picton, T. W., & Taylor, M. J. (2007). Electrophysiological evaluation of human brain development. *Developmental neuropsychology*, 31(3), 249-278.
- Pivik, R. T., Broughton, R. J., Coppola, R., Davidson, R. J., Fox, N., & Nuwer, M. R. (1993). Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology*, 30(6), 547-558.
- Poeppel, D. (2003). The analysis of speech in different temporal integration windows: cerebral lateralization as 'asymmetric sampling in time'. *Speech communication*, 41(1), 245-255.
- Ponton, C., Eggermont, J. J., Khosla, D., Kwong, B., & Don, M. (2002). Maturation of human central auditory system activity: separating auditory evoked potentials by dipole source modeling. *Clinical Neurophysiology*, 113(3), 407-420.
- Putkinen, V., Tervaniemi, M., Saarikivi, K., Ojala, P., & Huotilainen, M. (2014). Enhanced development of auditory change detection in musically trained school-aged children: a longitudinal event-related potential study. *Developmental science*, 17(2), 282-297.
- Rapaport, H., Seymour, R. A., Benikos, N., He, W., Pellicano, E., Brock, J., & Sowman, P. F. (2023). Investigating predictive coding in younger and older children using MEG and a multi-feature auditory oddball paradigm. *Cerebral Cortex*, 33(12), 7489-7499.
- Rüsseler, J., Altenmüller, E., Nager, W., Kohlmetz, C., & Münte, T. F. (2001). Event-related brain potentials to sound omissions differ in musicians and non-musicians. *Neuroscience letters*, 308(1), 33-36.
- Schröger, E., Näätänen, R., & Paavilainen, P. (1992). Event-related potentials reveal how non-attended complex sound patterns are represented by the human brain. *Neuroscience letters*, 146(2), 183-186.
- Shafer, V. L., Morr, M. L., Kreuzer, J. A., & Kurtzberg, D. (2000). Maturation of mismatch negativity in school-age children. *Ear and hearing*, 21(3), 242-251.
- Shafer, V. L., Yan, H. Y., & Datta, H. (2010). Maturation of speech discrimination in 4-to 7-yr-old children as indexed by event-related potential mismatch responses. *Ear and hearing*, 31(6), 735-745.

- Shafer, V. L., Yan, H. Y., & Wagner, M. (2015). Maturation of cortical auditory evoked potentials (CAEPs) to speech recorded from frontocentral and temporal sites: three months to eight years of age. *International journal of psychophysiology*, 95(2), 77-93.
- Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 104(6), 540-545.
- Silman, S., Silverman, C. A., & Emmer, M. B. (2000). Central auditory processing disorders and reduced motivation: Three case studies. *Journal of the American Academy of Audiology*, 11(02), 57-63.
- Sussman, E., Steinschneider, M., Gumenyuk, V., Grushko, J., & Lawson, K. (2008). The maturation of human evoked brain potentials to sounds presented at different stimulus rates. *Hearing Research*, 236(1-2), 61-79.
- Sussman, E. S. (2007). A new view on the MMN and attention debate: the role of context in processing auditory events. *Journal of Psychophysiology*, 21(3-4), 164-175.
- Sussman, E. S., Chen, S., Sussman-Fort, J., & Dinces, E. (2014). The five myths of MMN: redefining how to use MMN in basic and clinical research. *Brain topography*, 27(4), 553-564.
- Tamakoshi, S., Minoura, N., Katayama, J. i., & Yagi, A. (2016). Entire sound representations are time-compressed in sensory memory: evidence from MMN. *Frontiers in neuroscience*, 10, Article 347.
- Themas, L., Nickel, E. S., Lippus, P., Padrik, M., & Kreegipuu, K. (2025). Exploring pitch and length perception: An EEG study on quantity discrimination in preschoolers with developmental language disorder. *Acta psychologica*, 258, 105157.
- Timm, J., Weise, A., Grimm, S., & Schröger, E. (2011). An asymmetry in the automatic detection of the presence or absence of a frequency modulation within a tone: a mismatch negativity study. *Frontiers in psychology*, 2, 189.
- Todd, J., Finch, B., Smith, E., Budd, T. W., & Schall, U. (2011). Temporal processing ability is related to ear-asymmetry for detecting time cues in sound: A mismatch negativity (MMN) study. *Neuropsychologia*, 49(1), 69-82.
- Trainor, L., McFadden, M., Hodgson, L., Darragh, L., Barlow, J., Matsos, L., & Sonnadara, R. (2003). Changes in auditory cortex and the development of mismatch negativity between 2 and 6 months of age. *International journal of psychophysiology*, 51(1), 5-15.
- Trainor, L. J., Samuel, S. S., Desjardins, R. N., & Sonnadara, R. R. (2001). Measuring temporal resolution in infants using mismatch negativity. *Neuroreport*, 12(11), 2443-2448.
- Trehub, S. E., Schneider, B. A., & Henderson, J. L. (1995). Gap detection in infants, children, and adults. *The Journal of the Acoustical Society of America*, 98(5), 2532-2541.
- Weise, A., Grimm, S., Müller, D., & Schröger, E. (2010). A temporal constraint for automatic deviance detection and object formation: a mismatch negativity study. *Brain research*, 1331, 88-95.

- Weise, A., Grimm, S., Rimmele, J. M., & Schröger, E. (2023). Auditory representations for long lasting sounds: Insights from event-related brain potentials and neural oscillations. *Brain and language*, 237, 105221.
- Weise, A., Müller, D., Grimm, S., Rübsamen, R., & Schröger, E. (2007). Differential processing of terminal tone parts within structured and non-structured tones. *Neuroscience letters*, 421(2), 163-167.
- Weise, A., Ritter, W., & Schröger, E. (2011). The representation of unattended, segmented sounds: A mismatch negativity (MMN) study. *International journal of psychophysiology*, 81(2), 121-126.
- Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: predictive regularity representations and perceptual objects. *Trends in cognitive sciences*, 13(12), 532-540.
- Woldorff, M. G., Hillyard, S. A., Gallen, C. C., Hampson, S. R., & Bloom, F. E. (1998). Magnetoencephalographic recordings demonstrate attentional modulation of mismatch-related neural activity in human auditory cortex. *Psychophysiology*, 35(3), 283-292.
- Wong, A. C. W., & McPherson, B. (2015). Adaptive tests of temporal resolution: comparison with the gaps-in-noise test in normal-hearing young adults. *International Journal of Audiology*, 54(1), 29-36.
- Yabe, H., Koyama, S., Kakigi, R., Gunji, A., Tervaniemi, M., Sato, Y., & Kaneko, S. (2001). Automatic discriminative sensitivity inside temporal window of sensory memory as a function of time. *Cognitive Brain Research*, 12(1), 39-48.
- Yabe, H., Matsuoka, T., Sato, Y., Hiruma, T., Sutoh, T., Koyama, S., Gunji, A., Kakigi, R., & Kaneko, S. (2005). Time may be compressed in sound representation as replicated in sensory memory. *Neuroreport*, 16(2), 95-98.
- Yabe, H., Tervaniemi, M., Reinikainen, K., & Näätänen, R. (1997). Temporal window of integration revealed by MMN to sound omission. *Neuroreport*, 8(8), 1971-1974.

Chapter 5: Neural Correlates of Auditory Temporal Resolution in Children with Auditory Processing Disorder Using a Multi-Deviant Gap Detection Paradigm: A Pilot Study

Authors:

Shaghayegh Omidvar¹, Kenneth Campbell², Claude Alain^{3,4}, Steven Aiken⁵, Amineh Koravand^{1*}

Affiliations:

¹ School of Rehabilitation Sciences, University of Ottawa, Ottawa, Canada

² School of Psychology, University of Ottawa, Ottawa, Canada

³ Department of Psychology, University of Toronto, Toronto, Ontario, Canada

⁴ Rotman Research Institute, Baycrest Academy for Research and Education, Toronto, Ontario, Canada

⁵ School of Communication Sciences and Disorders, Dalhousie University, Halifax, Nova Scotia, Canada

***Corresponding address:**

Faculty of Health Sciences, University of Ottawa, 200 Lees Ave, Ottawa, Canada, ON K1S 5S9

Abstract

Objective: Children with auditory processing disorder (APD) often show poorer behavioural gap detection, but behavioural measures can be influenced by attention and motivation. This study aimed to evaluate whether an electrophysiological index of gap processing, deviant-related negativity (DRN), differs between children with APD and typically developing (TD) peers, and whether any effects depend on the temporal position of the gap.

Methods: Six children with APD and six age- and gender-matched TD children (8–12 years) with normal audiometric thresholds completed an active gap-detection task (d') and passive event-related potential (ERP) recording while watching a subtitled video. In the ERP paradigm, 500-ms broadband noise standards alternated with deviants containing a silent gap (10 or 30 ms) inserted at 15, 50, 100, 200, 300, or 400 ms after stimulus onset ($ISI = 400$ ms). DRN was isolated from deviant minus standard ERPs using both baseline-to-peak and peak-to-peak amplitude measures; confidence-interval testing assessed response detectability.

Results: Behaviourally, TD children showed higher d' than APD children across gap durations and onset times. Neurophysiologically, DRN amplitude increased with longer gap duration and decreased for later gap onsets. Overall, DRN amplitude did not differ between groups across most conditions. However, follow-up peak-to-peak analyses showed smaller DRN amplitudes in the APD group when the gap occurred at 15 ms for both the 10- and 30-ms gap durations. Confidence interval analyses suggested more restricted and variable DRN in APD, particularly for shorter gap durations and later gap onsets.

Conclusions: Children with APD showed poorer behavioural gap detection, but DRN amplitude was not consistently reduced across gap durations and onset times. Group differences in DRN were limited to selected conditions and should therefore be considered preliminary. The different patterns observed for behavioural and electrophysiological measures do not establish whether poorer behavioural performance in children with APD reflects bottom-up auditory-processing differences, task-related influences, or a combination of factors. Further studies with larger and better-characterized samples are needed to clarify these possibilities.

Keywords: auditory processing disorder, temporal resolution, gap detection, deviant-related response

5.1 Introduction

Listening difficulties (LiDs) encompass challenges in auditory comprehension of speech, particularly in noisy environments. These difficulties are not exclusive to individuals with hearing impairments but also affect those with cognitive deficits, language deficits, and auditory processing disorders (Dillon & Cameron, 2021). Auditory processing disorder (APD) is an umbrella term describing specific deficits that result in poorer-than-normal perception of auditory stimuli (Dillon & Cameron, 2021). Reported prevalence estimates for APD in school-age children vary considerably because of differences in diagnostic criteria, test batteries, and sampling methods; for example, Nagao et al. (2016) estimated a prevalence of 2.08 per 1,000 children (0.2%) in a Mid-Atlantic school-based cohort, whereas Shreyas and Jain (2024) reported a two-year clinical prevalence of 0.7% among children aged 6–18 years seen in an audiology outpatient service (Nagao et al., 2016; Shreyas & Jain, 2024). Despite this variability, APD remains clinically important because auditory processing deficits can interfere with speech understanding, especially in noise, and may contribute to broader listening, communication, and academic difficulties in children (Alanazi, 2023; American Speech-Language-Hearing Association [ASHA], n.d.; Dillon & Cameron, 2021; Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology, 2012).

The underlying mechanisms of APD remain debated. One hypothesis posits that APD reflects a bottom-up deficit in neural processing within the central auditory nervous system, despite normal peripheral hearing sensitivity (ASHA, 2005). A second hypothesis emphasizes top-down cognitive factors. These include attention, working memory, and executive control. Such processes involve multimodal networks in frontal, parietal, and anterior temporal regions (Dhamani et al., 2013; Ferguson et al., 2011; Moore et al., 2010; Moore & Hunter, 2013). Reflecting both views, the British Society of Audiology (Moore et al., 2018) defines APD as involving deficits in bottom-up auditory processing and top-down mechanisms, particularly auditory attention.

Given these listening and academic difficulties, considerable attention has been directed toward identifying the auditory processes that may be impaired in children with APD. One process of particular interest is temporal processing, which refers to the analysis of timing information in sound (ASHA, n.d.). Among the different aspects of temporal processing, temporal resolution refers to the auditory system's ability to detect temporal changes in acoustic signals, including

brief silent intervals or gaps within or between sounds (Lister et al., 2006; Musiek et al., 2005). The measure of temporal resolution ability offers valuable insights into an individual's capacity to distinguish rapidly changing sounds, such as speech sounds (Iliadou et al., 2014). The ability to perceive and react to changes in speech sounds is a fundamental aspect of language learning; therefore, temporal resolution plays a significant role in normal language development, including pre-reading and reading skills (Hautus et al., 2003; Walker et al., 2002), speech perception (Phillips, 1999), and production (Tallal et al., 1993).

One commonly used method for evaluating auditory temporal resolution involves gap detection (Keith, 2000; Musiek et al., 2005). A silent interval (the “gap”) is inserted either into a continuous sound (Musiek et al., 2005) or a brief-duration sound (Keith, 2000). The shortest detectable gap duration defines the gap detection threshold (GDT). Several studies have shown higher GDTs in children with APDs compared to their typically developing (TD) peers (Cameron et al., 2005; Dias et al., 2012; Jain et al., 2025; McWeeny & Norton, 2024; Omidvar et al., 2023; Phillips et al., 2010), although this difference diminishes with increasing age (Dias et al., 2012). For example, Dias et al. (2012) used the Random Gap Detection Test (RGDT) in a large cohort of participants aged 5 to 25 years, with and without CAPD, and found poorer performance in the CAPD group, especially in younger children. They argued that age-related improvement likely reflects auditory maturation and noted that behavioural gap-detection results in young children may also be influenced by non-auditory factors. However, these findings are not consistent across all gap-detection paradigms. Phillips et al. (2010) found no significant group difference for within-channel gap detection, although children with APD showed poorer performance in the between-channel condition. Similarly, Jain et al. (2025) found no significant differences between children with APD and TD children for broadband-noise or within-channel gap-detection measures, with a significant group difference observed only in the across-channel condition. These findings suggest that poorer gap-detection performance is observed in many children with APD, but the presence and magnitude of group differences may depend on the specific task and stimulus conditions.

Gap detection is important for speech comprehension (Buss et al., 2017; Gay et al., 2020). For example, the silent period between the release of a stop consonant and the onset of voicing of the following speech sound is the Voice Onset Time (VOT). Adequate gap detection skills can support differential discrimination of phonemes with different VOTs, such as /ba/ and /pa/ (Elangovan & Stuart, 2008). However, while the VOT or other silent gaps in phonemes occur

within milliseconds of the sound, the placement of gaps in clinical behavioural gap detection tests is much longer (Phillips, 1998). In the Behavioural Gap in Noise (GIN) test, for instance, the time interval between the stimulus onset and gap onset is at least 500 ms (Musiek et al., 2005), and in the RGDT, a silent interval consistently occurs in the middle of stimulus pairs (Keith, 2000).

Other limitations of behavioural assessments of gap detection include the need for active engagement (Silman et al., 2000) and sustained attention (Gyldenkaerne et al., 2014) throughout the testing period. This is difficult for most children, but particularly for those with APDs. This challenge is exacerbated when the gap position is close to the stimulus onset or offset, rather than at the stimulus midpoint (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999). Given that children with APD may also experience attentional difficulties (Dillon & Cameron, 2021), poor behavioural gap detection scores might not reflect any true deficit in temporal processing. Thus, an elevated GDT may reflect an actual bottom-up temporal processing disorder or a top-down listening disorder, an inability to sustain attention and actively listen throughout the testing period. In addition, behavioural methods require a perceptual decision, which can also be affected by other nonsensory factors such as memory and motivation, and attention (Duda-Milloy et al., 2019).

Scalp recordings of event-related potentials (ERPs) provide a non-invasive neurophysiology method to measure temporal resolution without having participants engage in the gap detection task (Bertoli et al., 2001; Bertoli et al., 2002; Desjardins et al., 1999; Duda-Milloy et al., 2019; Duda et al., 2025; Duda et al., 2020; Heinrich et al., 2004; Ross et al., 2010; Tamakoshi et al., 2016; Todd et al., 2011; Uther et al., 2003; Yabe et al., 2005). An ERP component labelled as the mismatch negativity (MMN) is often used in gap-detection studies (Näätänen & Alho, 1997). The MMN is usually studied using a so-called oddball paradigm. Participants are presented with a sequence of frequently occurring "standard" stimuli. All auditory stimuli elicit a series of obligatory ERPs, labelled as P1, N1, and P2. The P1 occurring between 50 and 100 ms is particularly large in children and is much reduced, occurring earlier (around 50 ms) in adults (Sharma et al., 1997; Čeponienė et al., 2002). At rare, unpredictable times, a feature of the standard stimulus is altered to form a "deviant." The deviant stimulus elicits, in addition to the P1-N1-P2, another negativity, the MMN (Näätänen et al., 2007). It peaks between 100 and 200 ms after the onset of the deviant. Its amplitude is maximum over the frontal and central scalp areas (Garrido et al., 2009; Näätänen et al., 2007). When a nose reference is used, it inverts in polarity (it is recorded as positive potential) at the mastoids. (Näätänen et al., 1978). In gap-detection studies, a short-

duration gap is inserted some time after the onset of the long-duration deviant. Thus, the deviant is physically identical to the standard stimulus until the occurrence of the gap. Although many studies classify the negativity elicited by an infrequent gap deviant as an MMN, this response may not be a “pure” MMN. The gap onset also elicits an N1, a component elicited by abrupt stimulus onsets or offsets (Näätänen & Picton, 1987). Because the onset periods of the standard and deviant are physically identical, they will elicit the same N1. The onset of the gap will also elicit an N1. The occurrence of the gap deviant, however, signals an acoustic change from the standard and thus elicits an MMN. Therefore, the gap elicits a composite negativity, the N1+MMN (Duda-Millooy et al., 2019). This composite negativity has been labelled as a deviant-related negativity (DRN) (Woldorff et al., 1998).

While several studies have used the MMN to probe auditory deficits in APD or CAPD children, they have generally used speech or pure tone deviants (Bauer et al., 2009; Hestvik et al., 2023; Koravand et al., 2017; Liasis et al., 2003; Mohideen & Thangaraj, 2023; Pinto et al., 2024; Rocha-Muniz et al., 2015; Roggia & Colares, 2008; Sharma et al., 2025). Sharma et al. (2025) recorded an MMN to a /da/–/ga/ contrast in 85 children aged 7 to 12 years old with LiDs (clustered by cognitive profiles). They reported that an MMN was difficult to detect in APD children. Mohideen and Thangaraj (2023) presented 1000 Hz pure-tone stimuli in which the deviant had a shorter duration than the standard (250 ms vs. 500 ms). They found that APD/dyslexic children had a smaller MMN amplitude than controls. In contrast, Pinto et al. (2024) compared CAPD vs TD children (aged 9–12 years) using a standard /da/ and a deviant /ta/ speech stimulus. They found no group differences in MMN amplitude. Koravand et al. (2017) also recorded MMN responses to both verbal and non-verbal deviants with APD vs TD children aged 9–12 years. The verbal contrast consisted of the syllables /ba/ and /da/. The non-verbal contrasts included simple pure tones at 1 and 2 kHz, as well as synthesized versions of /ba/ and /da/. These synthesized stimuli were constructed from two sine waves corresponding to the central frequencies of the second and third formants of the synthetic /ba/ and /da/. In different conditions, the standard stimulus was either speech /ba/, synthesized /ba/, or a 1kHz pure tone, while the deviant stimulus was either speech /da/, synthesized /da/, or a 2 kHz pure tone, respectively. Their results revealed no significant MMN differences between the two groups. Thus, electrophysiological evidence of auditory change detection deficits in APD remains mixed, and very little is known about electrophysiological responses to gap deviants in this population. This is an important limitation,

because unlike many conventional speech or tone deviants, a gap deviant is defined not only by the presence of an acoustic change, but also by when that change occurs within the stimulus. The temporal position of the gap may therefore be critical for understanding how auditory change detection operates in APD.

The importance of gap onset time for auditory change detection can be understood in relation to temporal integration. Because acoustic information unfolds over time, successive sound features must be extracted and integrated with preceding and subsequent information to form a coherent auditory event. This process is called temporal integration (Näätänen and Winkler, 1999; see review by Weise et al., 2023). The extracted information is stored in a very brief-lasting sensory memory where integration takes place. The storage capacity of sensory memory, however, is very limited. As acoustic information accumulates over time, this capacity may be exceeded (Arnott & Alain, 2002; Bizley & Cohen, 2013; Poeppel, 2003). The time span over which this integration occurs is referred to as the temporal window of integration (TWI). The duration of the TWI depends on the spectrotemporal content of the stimulus and the temporal scale of the auditory information being processed (Weise et al., 2023). The auditory system may use different integration windows depending on whether the relevant information is carried by rapid acoustic changes or by slower temporal patterns. For example, phonemic cues in speech are associated with relatively short integration windows of approximately 20–40 ms, whereas syllabic-level patterns unfold over longer time scales and are associated with longer windows of approximately 125–250 ms. For non-speech sounds with more slowly varying acoustic structure, auditory information may be integrated over a longer window, with an upper limit often estimated around 300–350 ms (Poeppel, 2003; Weise et al., 2023). When acoustic features occur within the TWI, they are integrated and perceived as part of a single auditory event; when they occur outside this window, they are more likely to be perceived as separate auditory events (Näätänen, 1992; Winkler et al., 2009).

The concept of the TWI is particularly relevant to the study of gap detection using ERPs. In most MMN studies, the acoustic change defining the deviant occurs at stimulus onset and persists throughout the stimulus (Hestvik et al., 2023; Liasis et al., 2003; Pinto et al., 2024; Rocha-Muniz et al., 2015). This is not the case with gap deviants. Obviously, the gap cannot occur at stimulus onset. Rather, the gap occurs at some time after the onset of the deviant. Thus, prior to the onset of the gap, the deviant is physically identical to the standard stimulus. This means that

neural detection of the deviant depends not only on the presence of the gap itself, but also on when the gap occurs relative to the temporal limits of auditory integration. In most MMN studies, the gap is inserted in the centre of the standard stimulus (Duda-Milloy et al., 2019; Linnavalli et al., 2018; Partanen et al., 2013). However, this is not representative of many natural sounds, including speech, in which brief silent intervals or other acoustic changes may occur early or late within the unfolding stimulus. Acoustic changes can occur at any time after stimulus onset, ranging from shortly after to very late.

Several studies have examined the effects of inserting a brief change into the standard stimulus at different temporal positions within the deviant. This has been most commonly studied with frequency modulations (Alain et al., 1994; Grimm et al., 2006; Grimm & Schröger, 2005; Hoonhorst et al., 2012; Näätänen et al., 2004; Schröger et al., 1992; Tamakoshi et al., 2016; Timm et al., 2011; Weise et al., 2010; Weise et al., 2007; Weise et al., 2011; Yabe et al., 2001; Yabe et al., 2005). For example, Weise et al. (2011) evaluated 13 healthy adults (aged 19–32 years) by presenting a 900-ms, 440-Hz pure tone in which the deviant was created by inserting a brief 50-ms frequency change at different temporal positions within the stimulus. A large amplitude MMN was elicited when the frequency modulation occurred prior to 300 ms and was much reduced when it occurred at 300 ms. The MMN was absent when the frequency modulation occurred at 400 ms or later. This temporal-distance effect demonstrates that automatic detection of deviance is strongly constrained when a brief acoustic change occurs within a sound and supports the idea that deviance detection is limited by the duration of the TWI.

This issue is especially relevant to the study of gap detection. Brief silent intervals can occur at different temporal positions in speech and other natural sounds, yet the commonly used behavioural gap-detection tests do not examine the effect of gap onset time. In the present study, the gap occurred from 15 to 400 ms after the onset of the deviant. Evaluating such effects electrophysiologically with a conventional oddball design is not ideal, because only a single deviant is presented among the frequently occurring standards. To test the effect of several different gaps occurring at different times after the deviant onset would require several representations of the oddball sequence, once for each deviant. This would be very time-consuming and difficult for young children to tolerate. To address this limitation, we introduced a novel multi-deviant paradigm to investigate the effect of gap-onset timing in young adults (Omidvar et al., 2026). In this multi-deviant paradigm, standards and deviants alternated. Six different gap onset

times were examined (15, 50, 100, 200, 300, or 400 ms), using two different gap durations (10 and 30 ms). The duration of the gap was identical for each of the six deviants, so that the six deviants within a condition differed only in the temporal position of the silent gap. The results of the multi-deviant paradigm were compared to those of an oddball paradigm in which only a single deviant was presented. In the oddball paradigms, only 10-ms-gaps occurring at 100 or 300 ms were run in separate sequences. The DRN amplitude did not significantly differ between the single-deviant and multi-deviant paradigms for the matched conditions. These findings showed that measurable DRNs could be elicited using the multi-deviant paradigm while allowing several gap onset times to be examined within the same sequence.

A follow-up study (Omidvar et al., 2026) then applied this paradigm to children and adults to examine developmental differences in the effect of gap onset time. The results showed that DRN amplitude decreased substantially when the gap occurred 200 ms after the deviant onset in children and 400 ms after the deviant onset in adults. While the DRN amplitude from baseline to peak was smaller in children than in adults, when measured as peak-to-peak (from the negative DRN deflection to the subsequent positivity), it was comparable in both groups.

Whether a similar temporal-position effect is present in children with APD remains unknown. This question is important for two reasons. First, behavioural studies have shown elevated GDTs in children with APD compared with TD children (Cameron et al., 2005; Dias et al., 2012; Jain et al., 2025; McWeeny & Norton, 2024; Omidvar et al., 2023; Phillips et al., 2010), although findings vary depending on the gap-detection measure and stimulus condition (Phillips et al., 2010; Jain et al., 2025). Behavioural gap-detection measures can also be affected by attention, task engagement, and other non-sensory factors. Therefore, poorer behavioural performance may not necessarily reflect only a deficit in bottom-up auditory temporal processing. Passive electrophysiological measures reduce some of these task demands, although they do not provide a direct measure of auditory processing during active listening. Second, if difficulties in forming sensory representations in children with APD (Hestvik et al., 2023) also affect the temporal integration of auditory information, the effective TWI may differ between APD and TD children. However, this has not been directly examined. Hestvik et al. (2023) suggested that children with APD may have difficulty forming a stable sensory memory representation of the acoustic input. If so, children with APD may show a reduction in DRN amplitude at earlier gap onset times than TD children, reflecting a shorter effective window for integrating and comparing

acoustic information. The key unresolved issue in the literature is that electrophysiological responses to gap deviants in children with APD have received very little direct study, as previous APD MMN studies have primarily used speech, frequency, or duration deviants (Liu et al., 2021), and no study has examined whether the temporal position of a gap within an ongoing stimulus influences the DRN differently in APD and TD children. Addressing this issue is important because it can help clarify whether the elevated behavioural gap-detection thresholds often observed in APD (Cameron et al., 2005; Dias et al., 2012; Jain et al., 2025; McWeeny & Norton, 2024; Omidvar et al., 2023; Phillips et al., 2010) are associated with altered early auditory processing and temporal integration, or whether they are more likely explained by non-sensory task demands.

Accordingly, the objective of the present pilot study was to compare DRN amplitudes in children with APD and TD children using a multi-deviant paradigm in which a gap was inserted at different temporal positions after stimulus onset. We first asked whether children with APD would show reduced DRN amplitudes relative to TD children, which would be consistent with a bottom-up auditory processing deficit. If the poorer behavioural gap detection in APD is partly due to a deficit in automatic auditory processing, then reduced DRN amplitudes would also be expected in children with APD. However, our previous studies showed no significant correlations between behavioural sensitivity and DRN amplitude, indicating that the two measures do not necessarily vary together. Therefore, it was also possible that children with APD would show poorer behavioural performance without a consistent reduction in DRN amplitude. Such a pattern would suggest that the poorer behavioural performance may be influenced by the greater task demands of active gap detection, while not providing evidence for a group difference in automatic gap processing as measured by the DRN. If difficulties in forming sensory representations in children with APD also affect temporal integration, the effective TWI may differ between APD and TD children. In this case, the DRN amplitude may decline at earlier gap onset times in children with APD than in TD children.

5.2 Methods

5.2.1 *Participants*

The study included two groups: 7 children (4 males) with APD and 7 TD children aged 8–12 years, matched by age and gender (4 males per group). The TD comparison group was selected

from the TD children who had participated in Study 2 (Chapter 4) to provide age- and gender-matched controls for the children with APD. Seven children with APD and seven matched TD children were initially included. Following the electroencephalography (EEG) recording, the data from one child in the APD group was found to be excessively noisy, resulting in a final sample of six children in each group. Consequently, this participant and her matched TD counterpart were excluded from the analysis. None of the participants were diagnosed with neurological, attention, or cognitive disorders, and none had a history of seizures or head trauma. Normal hearing levels were verified using standard audiological procedures. Their hearing threshold did not exceed 15 dB HL at octave frequencies from 250 to 8000 Hz (American National Standards Institute [ANSI], 2010). Middle ear function was also assessed to ensure normal tympanometric peak pressure (between -100 and +50 daPa) in both ears (Gelfand, 2016). The University of Ottawa Research Ethics Board approved the study (file number # H-07-24-10412). Written informed consent was obtained from the parents or guardians of all child participants prior to the experiment. Children received a \$20 gift card as a token of appreciation for their participation.

Children in the APD group were recruited from a private clinic (APD Ottawa Clinic). Children had already received a clinical diagnosis of APD based on a comprehensive assessment conducted by a qualified audiologist before enrolling in the study (Table 5.1). That diagnosis was carried out separately from the current study. Children were classified as having APD if they showed abnormal performance (at least 2 standard deviations [SDs] below the mean) on two or more auditory processing tests, defined according to test-specific cut-off criteria (Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology, 2012).

Children with a history of attentional or cognitive difficulties, as reported by a psychologist, were excluded from the study. No family history of language or learning impairments was reported for children in the TD group. Parents of children in the control group also completed the Auditory Processing Domains Questionnaire (APDQ; O'Hara & Mealings, 2018) to screen for potential difficulties in auditory processing, language, or attention. Children were considered to pass the APDQ screening if they met the criteria for normal listening, with scores above the 15th percentile for the auditory-processing and language domains and above the 20th percentile for the attention domain (O'Hara & Mealings, 2018). In addition, the Auditory Continuous Performance Test (ACPT; Keith, 1994) was administered to screen sustained auditory attention in children aged 8 to 11 years. The ACPT Total Error Score was calculated as the sum of

omission and commission errors, and performance was classified as pass or fail according to the age-specific criterion provided in the test manual. The ACPT was not used to classify the four 12-year-old participants because they were outside the normative age range of the test (6;0–11;11 years). For the two 12-year-old TD children, no attentional difficulties were reported by the parent, and the attention domain of the APDQ was within the normal range. For the two 12-year-old children with APD, no attentional difficulties were reported by the parent, and they had passed their ACPT in their previous assessment at earlier ages.

Table 5.1 Results of participants with Auditory Processing Disorder (APD) on auditory-processing and attention screening tests.

Participant	Age (yr;mo)	ACPT	RGDT	Phonemic Synthesis	Filtered Words	Dichotic Digits	Competing Sentences	BKB-SIN
APD01	12;06	NT at current assessment Previous ACPT: N	N 2 ms	AN 20/25; criterion ≥ 23	RE: N, 88% LE: AN, 72% criterion $\geq 78\%$	RE: N, 100% LE: AN, 77.5% criterion $\geq 90\%$	RE: N, 96% LE: AN, 56% criterion $\geq 90\%$	BE: N, -0.3 dB RE: N, 1.7 dB LE: AN, 3.2 dB criterion ≤ 3 dB
APD02	12;07	NT at current assessment Previous ACPT: N	N 2 ms	N 23/25; criterion ≥ 23	RE: AN, 76% LE: AN, 68% criterion $\geq 78\%$	RE: N, 92.5% LE: N, 97.5% criterion $\geq 90\%$	RE: N, 98% LE: AN, 16% criterion $\geq 90\%$	BE: N, 2.15 dB RE: AN, 3.4 dB LE: AN, 4.15 dB criterion ≤ 3 dB
APD03	8;03	N 9 errors; criterion ≤ 25	N 2 ms	AN 9/25; criterion ≥ 18	RE: N, 76% LE: N, 88% criterion $\geq 70\%$	RE: N, 85% LE: N, 87.5% criteria RE $\geq 75\%$, LE $\geq 65\%$	RE: AN, 76% LE: N, 43% criteria RE $\geq 82\%$, LE $\geq 39\%$	BE: N, 0.7 dB RE: B, 3.2 dB LE: N, 1.7 dB criterion ≤ 3 dB
APD04	11;06	N 4 errors; criterion ≤ 16	N 5 ms;	N 21/25; criterion ≥ 21	RE: N, 84%; LE: N, 88%; criterion =75%	RE: N, 92.5%; LE: AN, 87.5%; criteria RE $\geq 90\%$, LE $\geq 88\%$	RE: N, 98%; LE: AN, 84%; criterion $\geq 90\%$	BE: AN, 4.4 dB; RE: AN, 3.9 dB; LE: N, 2.4 dB; criterion ≤ 3 dB
APD05	9;07	N 4 errors; criterion ≤ 19	NT	N 21/25; criterion ≥ 18	RE: N, 84% LE: AN, 52% criterion $\geq 68\%$	RE: N, 87.5% LE: AN, 55% criteria RE $\geq 80\%$, LE $\geq 75\%$	RE: N, 95% LE: AN, 4% criteria RE $\geq 90\%$, LE $\geq 74\%$	BE: N, 1.7 dB RE: N, 2.7 dB LE: N, -0.3 dB
APD06	11;04	N 6 errors; criterion ≤ 16	NT	AN 15/25; criterion ≥ 21	RE: N, 76% LE: N, 88% criterion =75%	RE: N, 97.5% LE: N, 92.5% criteria RE $\geq 90\%$, LE $\geq 88\%$	RE: AN, 84% LE: N, 96% criterion $\geq 90\%$	BE: AN, 4.7 dB RE: AN, 4.2 dB LE: AN, 3.9.7 dB criterion ≤ 3 dB

N = normal; AN = abnormal; B = borderline/slightly outside the expected range; NT = not tested; RE = right ear; LE = left ear; BE = both ears; ACPT = Auditory Continuous Performance Test; RGDT = Random Gap Detection Test; BKB-SIN = Bamford-Kowal-Bench Speech-in-Noise Test.

5.2.2 Procedure and stimuli

Participants in both groups sat in a sound-attenuated and electrically shielded booth. They were instructed to watch a silent, subtitled video of their choice and to ignore the auditory stimuli. A multi-deviant paradigm was used to present the standard and deviant stimuli. The standard stimulus was a 500 ms broadband white noise burst presented at a peak intensity of 80 dB SPL, with an instantaneous rise- and fall-time. The broadband noise ranged in frequency from near 0 Hz to 22 kHz. A wide bandwidth was used because stimuli with broader frequency bandwidths have gap detection thresholds that are similar in children and adults (Buss et al., 2014; Buss et al., 2017). Moreover, the use of broadband noise reduced the likelihood of detecting spectral splatter caused by the abrupt onset and offset of the stimulus (Brown & Nicholls, 1997). The to-be-ignored irrelevant auditory stimuli were delivered binaurally via insert-earphones (E-A-RTONE 3A). Sound intensity was calibrated using a Brüel & Kjær Type 2235 sound level meter and a 2 cm³ coupler (GRAS RA0045-S1).

Six different deviants were created by inserting a silent interval (a “gap”) at different positions in the standard. The six gaps occurred at 15, 50, 100, 200, 300, or 400 ms after the onset of the deviant. The gap had an instantaneous onset and offset. The standards and deviants alternated; the probability of occurrence of each stimulus type was therefore 50%. However, the probability of each of the six gap deviants was .083. The deviants were ordered pseudo-randomly such that in a sequence of 12 alternating standards and deviants, each of the six deviants was presented. Their order of presentation within the sequence was randomized. The last deviant in the sequence and the first in the subsequent sequence could not be the same. Moreover, the same random order of presentation of deviants within a sequence could not occur a second time. The inter-stimulus interval (offset-to-onset) was 400 ms. The block of trials lasted about 14 minutes, with 472 standards (including 10 initially presented) and 77 of each deviant. Gap duration was either 10 or 30 ms, in separate conditions. The two multi-deviant conditions were each repeated three times in random order. A brief rest period was provided between sequences. The timing and presentation of all stimuli were controlled by a computer running E-Prime® (Psychology Software Tools, version 2.0).

5.2.3 Electrophysiological recording and analyses

The EEG was recorded from 30 active silver-silver chloride electrodes placed according to the International 10-10 system (Pivik et al., 1993) and attached to an electrode cap. Electrodes were positioned on the midline and lateral areas of the frontal, central, parietal, temporal, and occipital regions. The reference electrode was located on the tip of the nose. Vertical eye movement and blink artifacts were recorded from an electrode placed on the infra-orbital ridge of the left eye. Inter-electrode impedance was below 25 k Ω . The continuous EEG activity was sampled at 500 Hz and stored on disk.

The data were subsequently analysed using Brain Products' Analyzer 2.3 software. The EEG and electrooculography (EOG) data were digitally filtered with a band-pass filter from 0.3 to 20 Hz. The continuous data were visually inspected to identify channels contaminated by high-amplitude noise. These channels were replaced by interpolating the data of surrounding electrode sites (Perrin et al., 1989). A maximum of four channels was replaced by interpolation. Independent Component Analysis (ICA; Makeig et al., 1996), was used to identify eye movement and blink artifacts statistically independent of the EEG activity. To correct for these artifacts occurring within the EEG signals, vertical and horizontal EOG activity needed to be computed. A vertical EOG channel was computed by subtracting activity recorded at FP1 from that at the infra-orbital ridge. A horizontal EOG channel was computed by subtracting FT9 activity from that of FT10. These statistically independent components were then subtracted from the raw EEG to minimize contamination from eye movements.

The continuous EEG was partitioned into 1000-ms epochs, beginning 100 ms before stimulus onset. The average of the 100-ms pre-stimulus period served as a zero-voltage baseline, from which ERP components were measured. Each single trial epoch was then baseline-corrected to remove slow voltage "drifts." For each of the standard and deviant single trials, the mean amplitude of all data points within the baseline period was subtracted from that of all subsequent data points in the post-stimulus period. Any epoch containing activity exceeding ± 100 μ V was then rejected from further analysis. Rejections based on this criterion were relatively low because the most common type of artifact, eye blinks and movements, had already been corrected. The single epochs were then sorted by electrode site, stimulus type (standard, six deviants), and condition (10, 30 ms duration), and averaged.

5.2.4 Behavioural detection

A separate condition was run after the ERP session, in which the participant was asked to actively detect the deviant gap. In this condition, the standards (500-ms broadband white noise bursts) and the 10- and 30-ms duration deviants were presented in a single sequence. A silent gap was again inserted at 15, 50, 100, 200, 300, or 400 ms after the onset of the deviant. The sequence thus included standards and 12 different deviants (2 gap durations x 6 different onset times). A total of 50 standards and 120 deviants (10 for each type of gap deviant) were presented in random order. The stimuli were presented with a fixed 1500 ms ISI to allow participants time to respond. Participants were asked to press the right mouse button if they judged the stimulus to contain a gap (i.e., the deviant stimulus) and the left mouse button if they judged it not to contain a gap (i.e., the standard stimulus). Ten practice trials were presented to ensure the participant understood the instructions.

Hits (correctly detecting the gap deviant), correct rejections (correctly detecting the standard), false positives (detecting the standard as a gap deviant), and misses (detecting the gap deviant as a standard) were computed. The hit and false-positive rates were used to compute an index of sensitivity (d').

5.2.5 ERP Quantification and Analysis

The gap within the deviant elicited a DRN in both groups. The DRN is best observed in a difference wave, computed by subtracting the ERP response to the standard from the response to the deviant. Processing common to both the standard and deviant (the P1-N1-P2) should have been removed in the subtraction routine, leaving only processing unique to the deviant (the DRN). The subtraction procedure for children with APD and TD children, at onset times of 15, 100, and 300 ms, is illustrated in Figure 5.1.

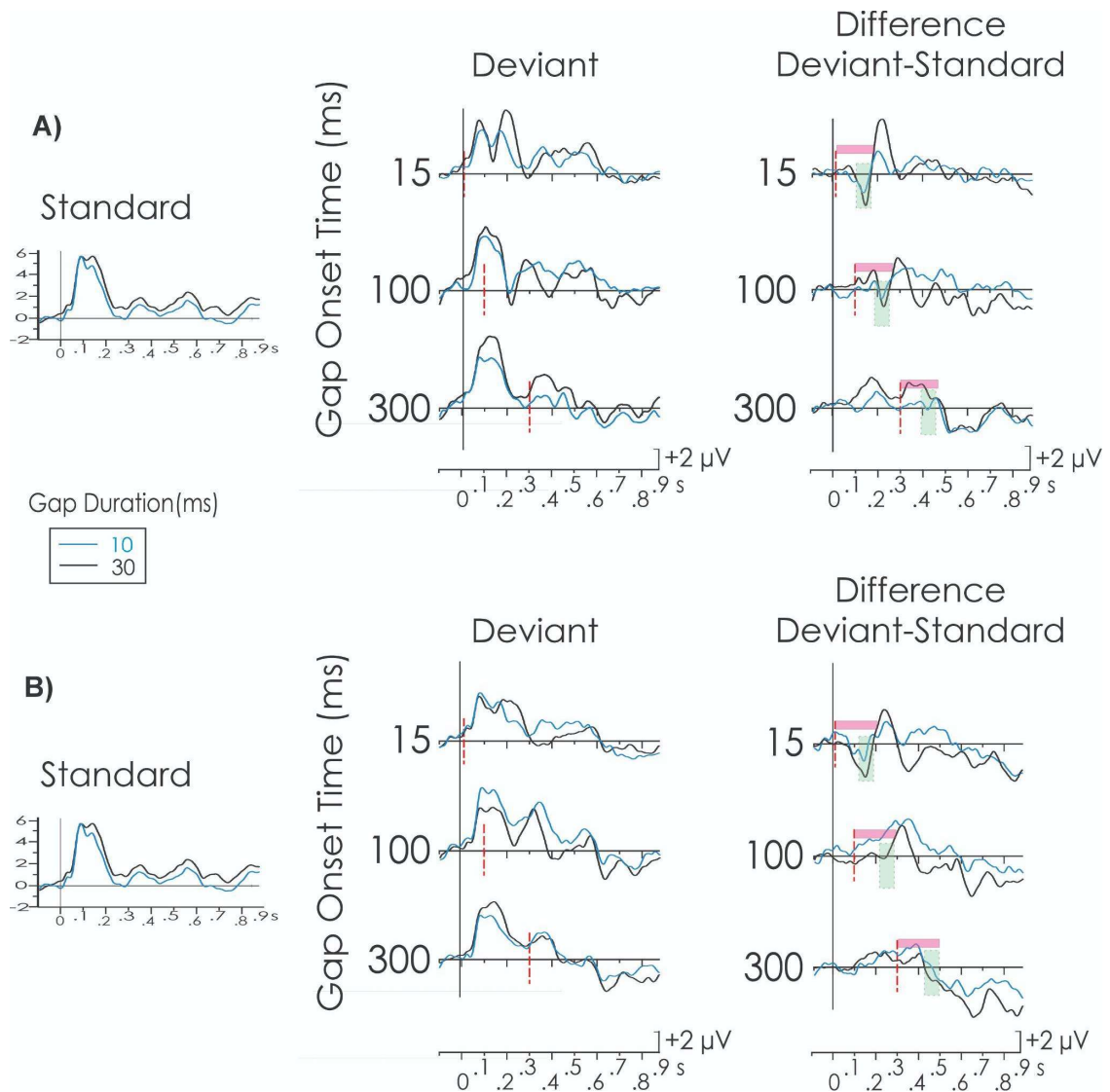


Figure 5.1 “Raw” standard (left column), deviant (middle column), and the deviant-standard difference wave (right column) in TD children (A), and children with APD (B) for the gap onset times of 15, 100, and 300 ms at Fz. The standard and deviant were physically identical until the time of onset of the gap (indicated by the red vertical lines). In the left portion of the Figure, the ERPs to the standard stimulus are illustrated. The onset of the standard elicited a large positivity. The middle portion illustrates the ERPs elicited by the different deviant stimuli. The onset of the deviant also elicited a large positivity. The right portion of the Figure illustrates the deviant-standard difference wave. Processing common to the standard and deviant should thus have been removed during the subtraction process. Thus, the large positive wave common to both the standard and deviant is removed in the difference wave, leaving processing unique to the gap deviant.

The DRN was quantified for each individual using the mean amplitude of all data points within ± 25 ms of the maximum peak identified in the grand average difference wave for each of the six deviants in the 10- and 30-ms gap duration conditions. Their amplitudes were measured relative to the zero-voltage pre-stimulus baseline. Another common approach for quantifying the

DRN is to measure the most negative peak within a predefined latency window. This approach, however, is sensitive to noise because the identified peak reflects the combined contribution of the true DRN and random fluctuations in the waveform. This becomes particularly problematic when the underlying DRN is small, as would be expected for brief gaps. The mean-amplitude measure used in the present study is less affected by this issue because positive- and negative-going drifts within the measurement window tend to cancel each other out (Luck, 2014). A limitation of the mean-amplitude approach is that it does not readily provide a clear estimate of peak latency. Table 5.2 summarizes the latency ranges used to compute amplitude for each group.

Table 5.2 Latency windows used to compute DRN amplitude for each condition in children with APD (n = 6) and TD children (n = 6).

Gap onset time (ms)	Gap duration: 10ms		Gap duration: 30 ms	
	APD	TD	APD	TD
15	120-170	115-165	125-175	120-170
50	165-215	145-195	160-210	155-205
100	215-265	200-250	210-260	205-255
200	310-360	290-340	330-380	320-370
300	405-455	400-450	435-485	405-455
400	535-585	500-550	550-600	510-560

Significant differences in the amplitude of the DRN as a function of deviant onset time would usually be determined using an analysis of variance (ANOVA) statistical procedure. It was, however, expected that only gaps occurring prior to 300 ms would elicit a DRN, while for deviants occurring later, a DRN might be much reduced or absent. The ANOVA procedure is used to determine if the amplitude of the DRN was significantly smaller or larger among the various deviants. The ANOVA cannot be used to determine if the DRN was absent (i.e., not elicited). To

determine whether a deviant elicited a DRN, confidence intervals (CIs) were computed. The CIs were calculated using data only at the Fz and Cz sites, where the DRN is largest. DRN responses were considered to be present only if both the upper and lower CI limits were more than the zero-voltage pre-baseline level (i.e., a positive value). Because the DRN was expected to have a negative value, a one-tailed test of significance ($p < .05$) was used for the CI testing.

Wilcoxon signed-rank tests were used to examine the effects of gap duration on the processing of the standard stimulus within each group, and Mann–Whitney U tests were used to compare differences in standard-stimulus processing between the two groups at each gap duration. Separate ANOVAs were then conducted at frontal (F3, Fz, F4) and central (C3, Cz, C4) regions of interest (ROIs) for the baseline-to-peak DRN data. A 3-way ANOVA with one between-group factor (TD, APD) and two repeated-measure factors, gap onset time (15, 50, 100, 200, 300, 400 ms) and gap duration (10, 30 ms) was computed. Because this was a small pilot study intended to identify potentially meaningful patterns for future investigation, the statistical analyses were considered exploratory, and a liberal significance criterion of $p \leq .10$ was adopted. This criterion increased sensitivity to possible effects but also increased the risk of Type I error; therefore, findings meeting this criterion were interpreted cautiously.

Given the pilot nature of the study and the small sample size, analyses conducted separately for the peak-to-peak DRN by gap duration and at Fz, as well as the subsequent condition-specific one-tailed comparisons, were considered exploratory. These analyses were intended to identify patterns that could inform future adequately powered studies rather than to provide confirmatory evidence of group differences. No correction for multiple comparisons was applied to these exploratory analyses; therefore, the resulting condition-specific findings should be interpreted cautiously. When a significant interaction was found, the source of the interaction was identified using simple main-effect tests. The power analysis, however, was conducted using the conventional $\alpha = .05$ threshold, because it was intended to estimate the sample size required for a future adequately powered confirmatory study. As this was a pilot study, a non-parametric Spearman's rank-order correlation was used to evaluate the relationship between the DRN and behavioural data. All statistical analyses were conducted using JASP software, version 0.96.0 (JASP Team, 2026).

5.3 Results

5.3.1 Behavioural data

Behavioural performance was measured using the signal detection measure, d' . This measure compares both the probability of correctly detecting a deviant containing a gap (i.e., a hit) and the probability of incorrectly detecting a standard as containing a gap (i.e., a false positive).

Figure 5.2 presents the mean d' for the 10- and 30-ms duration gaps as a function of gap onset time. As may be observed, d' values were higher in TD children than in APD children. A 3-way ANOVA was run with repeated measures on gap duration and gap onset time and one between-group measure. The group difference was significant, $F(1, 10) = 4.94$, $p = .05$, $\eta^2_p = .33$. TD children performed significantly better than APD children across both gap durations and regardless of gap onset time. The main effect of gap duration was not significant, $F < 1$. The main effect of gap onset time was significant, $F(5, 50) = 2.19$, $p = .07$, $\eta^2_p = .18$. The interactions between group and gap onset time, as well as group and gap duration, were not significant, $F < 1$. Correlations were computed between mean d' values and DRN amplitudes (baseline-to-peak and peak-to-peak) for each condition within each group. No significant correlations were found at either Fz or Cz for the two gap durations ($p > .1$ for all comparisons).

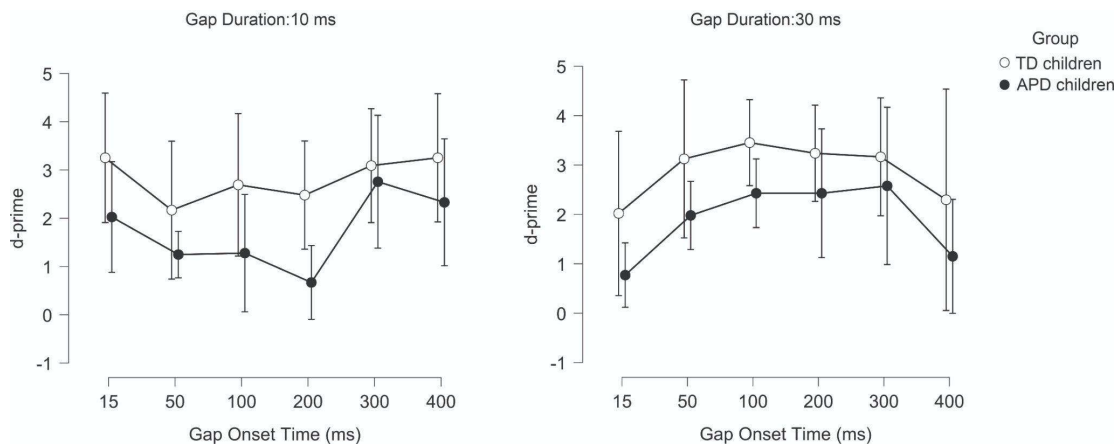


Figure 5.2 Mean d' for the 10- and 30-ms duration gaps as a function of gap onset time in TD children and children with APD. Error bars represent the 95% SE of the mean. As may be observed, d' was quite high overall, regardless of gap duration and the onset time of the gap. Performance, however, was significantly poorer in children with APD.

5.3.2 Electrophysiological data

5.3.2.1 Standard ERP

Figure 5.3 illustrates the standard ERP waveforms at Fz and Cz in the 10- and 30-ms conditions in the two groups. In both groups, a large-amplitude positivity (P1) was apparent around 95 ms. The N2 component peaks around 235 ms in TD children and around 250 ms in children with APD. Separate Wilcoxon tests were conducted for the two groups to compare the effects of gap duration on processing the standard stimulus. The comparisons revealed no significant differences in the amplitudes between the conditions (10 ms vs. 30 ms) at Fz, for either group, $p > .1$. The Mann–Whitney U tests revealed no significant differences in the amplitudes of P1 and N2 between the two groups ($p > .1$).

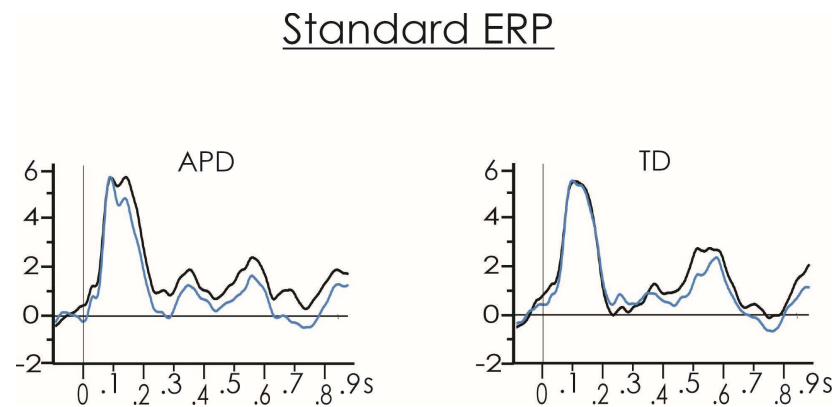


Figure 5.3 Standard ERP waveforms for the 10-ms and 30-ms gap duration conditions in TD and APD children at Fz. The amplitudes of the negative and positive deflections did not differ significantly within each condition.

5.3.2.2 Deviant-Standard ERPs

5.3.2.2.1 Deviant-Related Negativity (DRN)

The grand averages of the deviant-standard difference waves for 10- and 30-ms gaps in the two groups are shown in Figure 5.4. A fronto-central maximum DRN was elicited approximately 100–200 ms after gap onset.

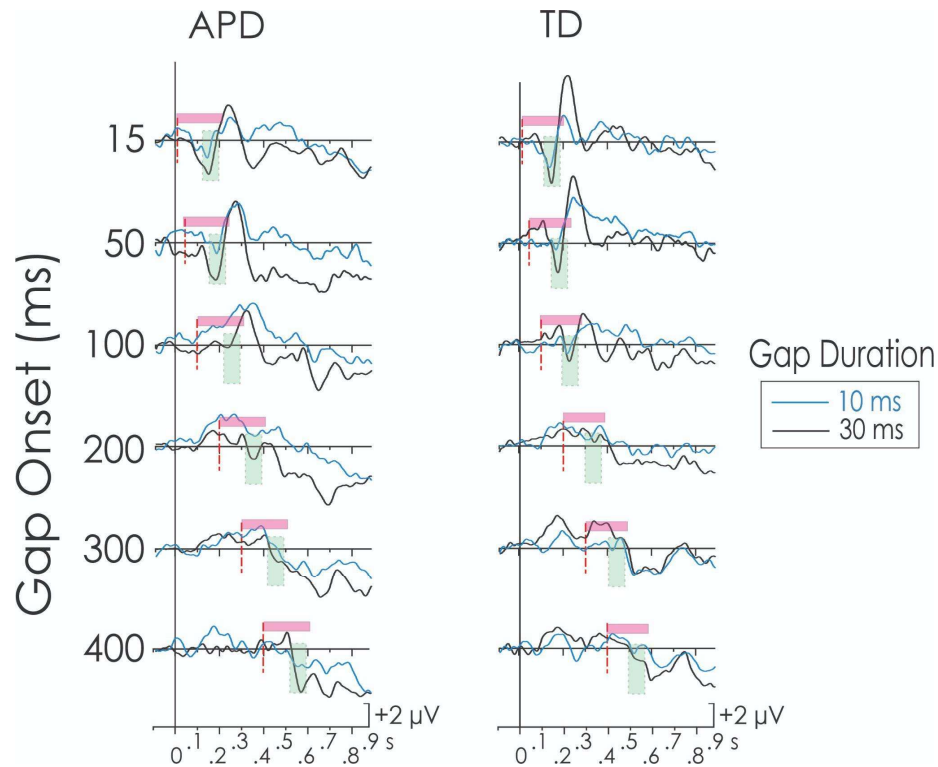


Figure 5.4 Deviant–standard ERPs in the multi-deviant condition for APD and TD children at Fz as a function of gap duration and gap onset time. The red dashed line indicates the onset of the gap. A clear DRN (green shaded regions) is visible around 100–150 ms following gaps occurring 15 to 100 ms after the onset of the deviant. For TD children, a clear DRN is visible for gaps occurring from 15 to 100 ms after the onset of the deviant. The DRN was not as clear as in TD children but appeared to be present at 15- and 50-ms onset times for the 30-ms gap duration.

Baseline-to-peak DRN.

The DRN was initially measured relative to the zero-voltage pre-stimulus baseline (i.e., baseline-to-peak). The ANOVA showed that the overall amplitude of the DRN did not differ between groups at either the frontal or central ROIs, $F < 1$ in both cases. The main effect of gap duration ($p \leq .06$) approached significance at both frontal and central ROIs. The DRN was larger for the 30-ms than for the 10-ms gap deviant. As expected, the main effect of gap onset time was significant, $p \leq .002$. However, the interactions between group and gap duration, as well as group and gap onset time, were not significant at either the frontal or central ROI ($p > .1$ in all cases).

Peak-to-peak DRN.

The DRN was measured relative to the zero-voltage pre-stimulus baseline. In Figure 5.4, it is apparent that in some conditions, the EEG prior to the onset of the gap was not always stable.

In some instances, it drifted in a negative direction; in others, in a positive direction. A negative drift will make the DRN larger (more negative-going), while a positive drift will make the DRN smaller (less negative-going). The DRN was followed by a positivity, the P2. The negative and positive drifts will “pull” this later positivity in the same direction as the DRN. When deflections are affected by baseline drift, ERPs are often measured as peak-to-peak rather than a baseline-to-peak measure (Picton et al., 2000). As shown in Figure 5.4, the amplitude of the DRN was often smaller than the subsequent positivity (P2). The data were, therefore, re-analyzed, measuring the DRN peak-to-peak amplitude relative to P2.

Figure 5.5 shows group mean amplitude as a function of gap onset time. The peak-to-peak amplitudes were much reduced, especially for gaps occurring 200 ms and later after the onset of the deviant. This was confirmed by the CI testing. The CI testing in TD children revealed significant peak-to-peak DRN responses at Fz when the gap occurred at 15, 50, or 100 ms for both 10- and 30-ms gap durations. A significant response was also observed for the 10-ms gap duration when the gap occurred at 200 ms. In APD children, a significant DRN was elicited by 30-ms gap-duration deviants at 15, 50, or 100 ms at Fz, and by 10-ms gap-duration deviants only at 50 ms.

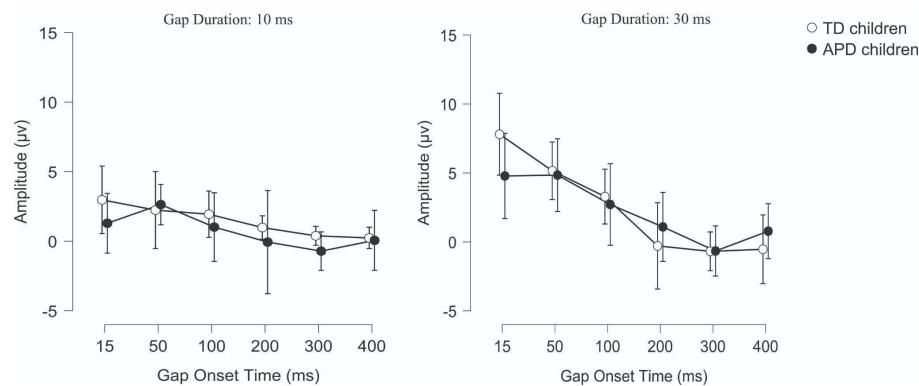


Figure 5.5 Mean peak-to-peak DRN amplitude in TD and APD children for the 10- and 30-ms duration gaps as a function of gap onset time at Fz. Error bars represent the standard error (SE) of the mean.

It is apparent in the grand-averaged waveforms that the amplitudes of the DRN and P2 were smaller following presentation of the 10-ms gap than the 30-ms gap. Although the overall ANOVA included gap duration as a factor and could therefore test interactions involving duration, effects that were collapsed across duration may have been influenced by the lower-amplitude 10-ms condition. Similarly, averaging across left, midline, and right scalp regions may have reduced

sensitivity to effects that were most clearly expressed at the midline electrode. Therefore, to maximize the likelihood of detecting group differences in this pilot study, separate ANOVAs were conducted on the 10- and 30-ms duration conditions. These ANOVAs were run only at Fz, where the DRN was largest.

The main effect of gap onset time was again significant in both conditions ($p < .1$). Group differences were still not significant for either duration ($F < 1$ in both cases), despite efforts to maximize differences. The group $\times$ gap onset interaction met the exploratory significance criterion when the gap duration was 30 ms, $F(5, 50) = 1.97$, $p = .10$. Liberal 1-tailed t-tests were employed to determine the possible source of the interaction, with the expectation that the DRN would be reduced in the APD group. For both the 10 and 30 ms duration conditions, no significant amplitude differences were observed between the TD and APD groups for gap deviants occurring between 50 and 400 ms ($t < 1$ in most cases). The amplitude of the DRN was significantly larger in the TD group when the gap occurred at 15 ms for the 10 ms ($p = .09$) and 30 ms ($p = .05$) duration gaps. Also, when the 10-ms gap was presented at a 300-ms onset, the difference was significant ($p = .05$).

5.3.3 Power analysis

A few statistically significant differences were observed that involved group comparisons. This may be because the small sample size lacked sufficient statistical power to reject the null hypothesis (i.e., to identify true group differences). A G*Power analysis (Faul et al., 2007) was conducted across the 12 conditions (2 gap durations $\times$ 12 onset times) to determine the sample size required to achieve statistical significance. The effect size within each of these 12 conditions was computed using the means and SDs of the 6 participants in each group and condition. These results are presented in Table 5.2. As may be observed, when existing group differences were relatively large, for example, when the 30-ms duration gap occurred 15 ms after the onset of the deviant, only 13 participants would need to be tested to attain significance at $p < .05$ with a power of 0.80. In most cases, however, group differences were very small. As such, hundreds, if not thousands, of participants would be required to achieve statistically significant differences when differences were very small.

Table 5.3 Total and per-group estimated sample sizes for each of the 12 conditions to detect a statistically significant difference (power = 0.80, α = 0.05) based on the means and standard deviations for each group.

Gap duration (ms)	Total (and per-group) Sample Size					
	Gap Onset Time					
	15	50	100	200	300	400
10	44 (22)	704 (352)	120 (60)	150 (75)	24 (12)	1902 (951)
30	26 (13)	1326 (663)	436 (218)	98 (49)	81308 (40654)	68 (34)

5.4 Discussion

The aim of the current investigation was to determine whether the poorer behavioural gap-detection performance often reported in children is accompanied by reduced DRN amplitudes, which would provide evidence of a difference in automatic auditory gap processing, or whether behavioural difficulties occur without a consistent reduction in the DRN. A number of studies have indicated that the gap-detection threshold is higher in children with APD than in TD children (Cameron et al., 2005; Dias et al., 2012; Mamatha & Yathiraj, 2020; McWeeny & Norton, 2023; Omidvar et al., 2023). At first glance, this could be interpreted as evidence of impaired bottom-up auditory processing, because successful gap detection depends on the auditory system's ability to encode brief temporal discontinuities in sound. However, these higher gap thresholds are derived from behavioural tasks, and performance on such tasks can also be influenced by non-auditory factors such as attention and motivation. Indeed, several studies have suggested that APD children may have difficulty in sustaining attention or maintaining engagement during demanding listening tasks (Chermak et al., 1998; Dawes & Bishop, 2009; Gyldenkerne et al., 2014; Moore et al., 2010; Riccio et al., 1994; Stavrinos et al., 2018). For this reason, it has also been hypothesized that APD children have a top-down cognitive deficit rather than a bottom-up auditory disorder. To reduce the potential influence of sustained-attention difficulties in the present study, auditory attention was screened using the ACPT and APDQ tests when participants were within the normative age range of the test. In the present study, a DRN was recorded for gaps inserted in white noise, because

this response can be elicited while the auditory stimuli are processed passively and is therefore relatively independent of active attention. Thus, if APD children have a bottom-up disorder, then it would be expected that DRN amplitude would be reduced compared to that of TD children. On the other hand, if the dysfunction reflects a top-down, non-sensory disorder, then the amplitude of the DRN would not be expected to differ between the two groups.

Children with APD had significantly poorer performance as measured by d' , regardless of the temporal position of the gap or its duration. As mentioned above, the poorer performance in APD children might reflect impaired bottom-up auditory processing. It may, however, also reflect top-down, nonsensory influences, such as attention. Children with APD have been reported to be less able or willing to sustain active attention throughout a gap-detection task (Dhamani et al., 2013; Ferguson et al., 2011; Moore et al., 2010; Moore & Hunter, 2013). For example, Dhamani et al. (2013) reported that children with listening difficulties were slower when they had to shift their attention from one sound source to another. Moore et al. (2010) found that children with listening difficulties showed greater variability in their responses across auditory-processing trials, which the authors suggested may reflect fluctuations in attention. In the present study, sustained auditory attention was screened using the ACPT for children within the normative age range of the test, and additional information regarding attention was considered for older participants. Therefore, sustained attention difficulties are less likely to fully explain the poorer behavioural performance of the APD group. However, attention during the gap-detection task itself was not directly measured and may still have varied during the task. Fatigue may also have contributed to behavioural performance because the behavioural task was administered after the physiological testing session. For both groups, gap detection was most challenging when gaps occurred near the beginning or end of the stimulus in the 30-ms gap condition. Performance patterns for the 10-ms gap condition were less stable and varied across participants. These findings align with previous research showing that temporal gap detection is more difficult near stimulus boundaries than in the middle of the stimulus (Gay et al., 2020; Phillips et al., 1997; Snell & Hu, 1999), likely because gaps occurring near the onset or offset of a sound provide less surrounding acoustic context for detecting a brief temporal discontinuity.

During the electrophysiological recording, participants were asked to watch a video while ignoring the auditory sequence. What the participant was actually “doing,” whether they were actively watching the video or not, should have had a minimal effect on the attention-independent

DRN. The usual gap effects were observed. The baseline-to-peak DRN amplitude was affected by gap duration. It was larger for the 30 than for the 10-ms gap. Also, as the gap onset time was delayed, the amplitude of the DRN decreased. The mean amplitude of the DRN, however, did not differ significantly between APD and TD children. There was, however, evidence of a slow drift in the EEG prior to the onset of the gap. For this reason, the DRN data were reanalyzed using a peak-to-peak amplitude measure, which is less affected by prestimulus baseline shifts. Peak-to-peak amplitude was smaller in the APD group than the TD group for both gap durations at the 15-ms onset time. In addition, as shown in Figure 5.4, children with APD showed greater variability and noisier DRN morphology than TD children, especially for the 10-ms gap duration. These condition-specific differences should be interpreted cautiously. The statistical approach in this pilot study was exploratory, with a liberal significance criterion of $p \leq .10$, and the subsequent condition-specific analyses included one-tailed comparisons without correction for multiple comparisons. Therefore, the lower DRN amplitudes observed in the APD group in three conditions should be considered preliminary and hypothesis-generating rather than confirmatory evidence of a bottom-up auditory processing deficit. Although reduced MMN amplitudes have been reported in previous studies of children with APD (Hestvik et al., 2023; Mohideen & Thangaraj, 2023; Rocha-Muniz et al., 2015), the present findings were limited to a small number of conditions and were not accompanied by an overall group difference in DRN amplitude. A power analysis was conducted to determine the sample size required to detect significant group differences, based on the effect sizes observed in this study. The power analyses were run for each of the six onset times and the two gap durations. The estimated sample size required to detect significant differences between the two groups varied widely, from about 25 to almost 100,000 participants, depending on gap duration and its temporal position. This variability indicates that some condition-specific group differences may be relatively large, whereas others are very small. Therefore, the absence of significant group differences in many conditions should be interpreted cautiously, because the small sample size limits the power to detect reliable effects.

The behavioural results showed a more consistent group difference, with poorer gap detection in children with APD across gap durations and onset times. In contrast, the electrophysiological differences were limited and condition specific. This discrepancy indicates that poorer behavioural gap detection in APD cannot be explained by the DRN findings alone. Other factors associated with the active behavioural task, such as attention, task demands, or

fatigue, may also contribute, although these factors were not directly examined in the present study. Given the small and heterogeneous sample, the present findings do not allow firm conclusions about the relative contributions of bottom-up and top-down factors to poorer behavioural gap detection in children with APD.

One aim of the present study was to compare the influence of the temporal position of the gap deviant on the DRN amplitude between TD and APD children. Based on the CI analysis, a DRN in TD children was detectable at gap onset times of 15, 50, and 100 ms for both gap durations, and it remained detectable at 200 ms with a 10-ms gap duration but was not detectable at 300 ms and later onsets. In contrast, in the APD children, a reliable DRN was present for the 30-ms gap at onset times of 15, 50, and 100 ms, but not for later gap onsets. This pattern may reflect a reduced auditory sensory memory capacity in children with APD compared with TD children (Hestvik et al., 2023). However, this interpretation should be tempered by the fact that, in the 10-ms gap condition, DRN amplitudes at 50, 100, and 200 ms did not differ significantly between groups. Therefore, the reduced detectability was not accompanied by consistent amplitude reductions across all conditions. Rather, group differences in DRN amplitude were observed only at specific gap-onset times. These condition-specific effects suggest that bottom-up auditory processing differences may contribute to gap-detection difficulties in APD, although the present pilot data did not support a generalized reduction in DRN amplitude across gap durations and onset times. Collectively, the present findings do not support a simple interpretation in which poorer behavioural gap detection in APD is explained entirely by either bottom-up or top-down factors. The absence of a group difference in overall DRN amplitude suggests that children with APD did not show a generalized reduction in automatic neural responses to gap deviants. However, reduced DRN amplitudes in some conditions indicate that top-down factors alone cannot fully account for the behavioural differences between groups. These condition-specific DRN differences are consistent with the possibility that bottom-up auditory processing limitations contribute to gap-detection difficulties in APD. At the same time, the absence of DRN amplitude differences at all gap-onset times where behavioural differences were present suggests that the relationship between neural gap processing and behavioural detectability is not direct or one-to-one. Behavioural performance may also have been influenced by attention, task engagement, or other non-sensory factors, and the small sample size may have limited the ability to detect additional group differences in the cortical responses. Thus, the present results are best interpreted as preliminary

evidence for both condition-specific bottom-up differences and possible top-down contributions to poorer behavioural gap detection in children with APD. In addition to the small sample size, a further limitation of the present study was that the electrophysiological paradigm included only a within-channel broadband-noise gap stimulus. Previous behavioural studies suggest that this type of gap stimulus may be less sensitive to APD-related group differences than gap-detection tasks between or across channels (Jain et al., 2025; Phillips et al., 2010). Phillips et al. (2010) reported that within-channel gap thresholds were very similar across control, APD+, and APD– groups, whereas between-channel thresholds differed significantly, with the largest difference observed between controls and children diagnosed with APD. More recently, Jain et al. (2025) likewise found significant group differences only for across-channel gap-detection measures, whereas within-channel and broadband-noise gap-detection measures did not significantly distinguish children with APD from TD children. It is therefore possible that the absence of clear group differences in DRN amplitude in the present study was, at least in part, due to the use of a within-channel broadband-noise gap paradigm. Future studies should examine electrophysiological responses to between-channel gap deviants in children with APD, ideally in direct comparison with within-channel conditions, to determine whether between-channel paradigms are more sensitive to group differences in neural temporal processing. It would also be useful to include a larger sample, examine narrower age ranges or developmental subgroups, and include speech-like or spectrally changing stimuli to determine whether APD-related group differences become more evident when the temporal task places greater demands on central auditory processing. Future studies should also characterize participants in greater detail and consider the heterogeneity of APD. Increasing the sample size alone may not be sufficient if children with different auditory-processing profiles are grouped together. More clearly defined participant subgroups, based on their clinical auditory-processing profiles and relevant co-occurring difficulties, may help identify whether particular patterns of neural temporal processing are associated with specific APD profiles.

5.5 Conclusions

This study examined whether children with APD showed evidence of impaired bottom-up temporal processing by comparing behavioural gap detection with an attention-independent electrophysiological response (DRN) to silent gaps in noise. Consistent with prior behavioural

research, children with APD showed significantly poorer gap detection performance (lower d') for both the 10- and 30 ms duration gaps, regardless of gap onset time. In contrast, when the auditory stimuli were processed passively (i.e., the auditory sequence was to be ignored), the analyses did not show a generalized reduction in DRN amplitude in children with APD. Exploratory follow-up analyses identified group differences in peak-to-peak DRN amplitude in only a limited number of conditions. The APD group also showed greater waveform variability and reduced DRN detectability in some conditions. These findings suggest the possibility of condition-specific differences in automatic gap processing between APD and TD children, but they should be considered preliminary and do not demonstrate a general deficit in bottom-up auditory temporal processing.

The discrepancy between the consistent behavioural group difference and the more limited electrophysiological differences indicates that poorer behavioural gap detection in APD is unlikely to be explained by the DRN findings alone. Factors related to the active behavioural task, such as attention or fatigue, may also contribute, although these were not directly measured. Given the small and heterogeneous sample, these findings should be considered preliminary. Future studies should include larger and more clearly characterized APD groups and examine whether different gap paradigms, including between-channel conditions, are more sensitive to differences in neural temporal processing.

Conflict of interest

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

Acknowledgements

The authors thank Dr. Caryn Bursch for her assistance with the recruitment of children with APD. We are also grateful to the children and their parents/guardians for their time and contribution to this study.

References

- Alain, C., Woods, D. L., & Ogawa, K. H. (1994). Brain indices of automatic pattern processing. *Neuroreport*, 6(1), 140-144.
- Alanazi, A. A. (2023). Understanding auditory processing disorder: A narrative review. *Saudi Journal of Medicine & Medical Sciences*, 11(4), 275-282.
- American-Academy-of-Audiology. (2010). Guidelines for the diagnosis, treatment, and management of children and adults with central auditory processing disorder. *Working Group on Auditory Processing Disorders*.
- American National Standards Institute [ANSI]. (2010). American national standard specification for audiometers. *American National Standards Institute, New York*.
- Arnott, S. R., & Alain, C. (2002). Effects of perceptual context on event-related brain potentials during auditory spatial attention. *Psychophysiology*, 39(5), 625-632.
- ASHA. (2005). Central auditory processing disorders [Technical Report]. In: Working Group on Auditory Processing Disorders.
- ASHA. (n.d.). *Central auditory processing disorder [Practice Portal]*. <https://www.asha.org/Practice-Portal/Clinical-Topics/Central-Auditory-Processing-Disorder/>
- Bauer, P., Burger, M., Kummer, P., Lohscheller, J., Eysholdt, U., & Doellinger, M. (2009). Correlation between psychometric tests and mismatch negativity in preschool children. *Folia Phoniatica et Logopaedica*, 61(4), 206-216.
- Bertoli, S., Heimberg, S., Smurzynski, J., & Probst, R. (2001). Mismatch negativity and psychoacoustic measures of gap detection in normally hearing subjects. *Psychophysiology*, 38(2), 334-342.
- Bertoli, S., Smurzynski, J., & Probst, R. (2002). Temporal resolution in young and elderly subjects as measured by mismatch negativity and a psychoacoustic gap detection task. *Clinical Neurophysiology*, 113(3), 396-406.
- Bizley, J. K., & Cohen, Y. E. (2013). The what, where and how of auditory-object perception. *Nature reviews neuroscience*, 14(10), 693-707.
- Brown, S., & Nicholls, M. E. (1997). Hemispheric asymmetries for the temporal resolution of brief auditory stimuli. *Perception & Psychophysics*, 59, 442-447.
- Buss, E., Hall III, J. W., Porter, H., & Grose, J. H. (2014). Gap detection in school-age children and adults: Effects of inherent envelope modulation and the availability of cues across frequency. *Journal of Speech, Language, and Hearing Research*, 57(3), 1098-1107.
- Buss, E., Porter, H. L., Hall III, J. W., & Grose, J. H. (2017). Gap detection in school-age children and adults: Center frequency and ramp duration. *Journal of Speech, Language, and Hearing Research*, 60(1), 172-181.
- Cameron, S., Dillon, H., & Newall, P. (2005). Three case studies of children with suspected auditory processing disorder. *Australian and New Zealand Journal of Audiology*, The, 27(2), 97-111.

- Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology. (2012). Canadian guidelines on auditory processing disorder in children and adults: Assessment and intervention.
- Čeponien, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870-882.
- Chermak, G. D., Somers, E. K., & Seikel, J. A. (1998). Behavioral signs of central auditory processing disorder and attention deficit hyperactivity disorder. *Journal of the American Academy of Audiology*, 9(1).
- Dawes, P., & Bishop, D. (2009). Auditory processing disorder in relation to developmental disorders of language, communication and attention: a review and critique. *International journal of language & communication disorders*, 44(4), 440-465.
- Desjardins, R. N., Trainor, L. J., Hevenor, S. J., & Polak, C. P. (1999). Using mismatch negativity to measure auditory temporal resolution thresholds. *Neuroreport*, 10(10), 2079-2082.
- Dhamani, I., Leung, J., Carlile, S., & Sharma, M. (2013). Switch attention to listen. *Scientific reports*, 3(1), 1297.
- Dias, K. Z., Jutras, B., Acrani, I. O., & Pereira, L. D. (2012). Random Gap Detection Test (RGDT) performance of individuals with central auditory processing disorders from 5 to 25 years of age. *International journal of pediatric otorhinolaryngology*, 76(2), 174-178.
- Diedler, J., Pietz, J., Bast, T., & Rupp, A. (2007). Auditory temporal resolution in children assessed by magnetoencephalography. *Neuroreport*, 18(16), 1691-1695.
- Dillon, H., & Cameron, S. (2021). Separating the causes of listening difficulties in children. *Ear and hearing*, 42(5), 1097.
- Duda-Milloy, V., Tavakoli, P., Campbell, K., Benoit, D. L., & Koravand, A. (2019). A time-efficient multi-deviant paradigm to determine the effects of gap duration on the mismatch negativity. *Hearing Research*, 377, 34-43.
- Duda, V., Augereau, T. M., Campbell, K., & Koravand, A. (2025). Does masking alter the mismatch negativity response to gaps? *Brain research*, 149651.
- Duda, V., Campbell, K., & Koravand, A. (2020). Event-related potentials following gaps in noise: The effects of the intensity of preceding noise. *Brain research*, 1748, 147078.
- Eggermont, J. (1995). Neural correlates of gap detection and auditory fusion in cat auditory cortex. *Neuroreport*, 6(12), 1645-1648.
- Elangovan, S., & Stuart, A. (2008). Natural boundaries in gap detection are related to categorical perception of stop consonants. *Ear and hearing*, 29(5), 761-774.
- Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G* Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior research methods*, 39(2), 175-191.
- Ferguson, M. A., Hall, R. L., Riley, A., & Moore, D. R. (2011). Communication, listening, cognitive and speech perception skills in children with auditory processing disorder (APD) or specific language impairment (SLI). *Journal of Speech, Language, and Hearing Research*, 54(1), 211-227.

- Forrest, T., & Green, D. M. (1987). Detection of partially filled gaps in noise and the temporal modulation transfer function. *The Journal of the Acoustical Society of America*, 82(6), 1933-1943.
- Garrido, M. I., Kilner, J. M., Stephan, K. E., & Friston, K. J. (2009). The mismatch negativity: a review of underlying mechanisms. *Clinical neurophysiology*, 120(3), 453-463.
- Gay, J. D., Rosen, M. J., & Huyck, J. J. (2020). Effects of Gap Position on Perceptual Gap Detection Across Late Childhood and Adolescence. *Journal of the Association for Research in Otolaryngology*, 21, 243-258.
- Gelfand, S. A. (2016). *Essentials of audiology* (Fourth ed.). Thieme.
- Grimm, S., Roeber, U., Trujillo-Barreto, N. J., & Schröger, E. (2006). Mechanisms for detecting auditory temporal and spectral deviations operate over similar time windows but are divided differently between the two hemispheres. *Neuroimage*, 32(1), 275-282.
- Grimm, S., & Schröger, E. (2005). Pre-attentive and attentive processing of temporal and frequency characteristics within long sounds. *Cognitive Brain Research*, 25(3), 711-721.
- Gyldenkerne, P., Dillon, H., Sharma, M., & Purdy, S. C. (2014). Attend to this: The relationship between auditory processing disorders and attention deficits. *Journal of the American Academy of Audiology*, 25(07), 676-687.
- Hautus, M. J., Setchell, G. J., Waldie, K. E., & Kirk, I. J. (2003). Age-related improvements in auditory temporal resolution in reading-impaired children. *Dyslexia*, 9(1), 37-45.
- Heinrich, A., Alain, C., & Schneider, B. A. (2004). Within-and between-channel gap detection in the human auditory cortex. *Neuroreport*, 15(13), 2051-2056.
- Hestvik, A., Morlet, T., Nagao, K., & Han, C. (2023). Auditory Processing Disorder Targets Phonetics, Not Phonology. Proceedings of the Annual Boston University Conference on Language Development. Boston University Conference on Language Development, Boston. (Vol. 2023, No. 1, p. 356).
- Hoonhorst, I., Deltenre, P., Markessis, E., Collet, G., Martin, X. P., & Colin, C. (2012). Evidence for a dual versus single origin of the MMNs evoked by cued versus cueless deviants. *Clinical Neurophysiology*, 123(8), 1561-1567.
- Iliadou, V., Bamiou, D.-E., Chermak, G. D., & Nimatoudis, I. (2014). Comparison of two tests of auditory temporal resolution in children with central auditory processing disorder, adults with psychosis, and adult professional musicians. *International Journal of Audiology*, 53(8), 507-513.
- Jain, C., Tanniru, K., & Kalarikkal, G. (2025). Performance of Children with and without Auditory Processing Disorders in Adaptive Temporal Gap Detection Measures. *International Archives of Otorhinolaryngology*, 29(04), 001-007.
- Keith, R. W. (1994). *ACPT: Auditory continuous performance test*. Psychological Corporation.
- Keith, R. W. (2000). *Random gap detection test*. St. Louis, MO: Auditec.
- Koravand, A., Jutras, B., & Lassonde, M. (2017). Abnormalities in cortical auditory responses in children with central auditory processing disorder. *Neuroscience*, 346, 135-148.

- Liasis, A., Bamliou, D.-E., Campbell, P., Sirimanna, T., Boyd, S., & Towell, A. (2003). Auditory event-related potentials in the assessment of auditory processing disorders: a pilot study. *Neuropediatrics*, 34(01), 23-29.
- Linnavalli, T., Putkinen, V., Huotilainen, M., & Tervaniemi, M. (2018). Maturation of speech-sound ERPs in 5–6-year-old children: a longitudinal study. *Frontiers in neuroscience*, 12, 814.
- Lister, J. J., Roberts, R. A., Shackelford, J., & Rogers, C. L. (2006). An adaptive clinical test of temporal resolution. *American Journal of Audiology*, 15, 133–140.
- Liu, P., Zhu, H., Chen, M., Hong, Q., & Chi, X. (2021). Electrophysiological screening for children with suspected auditory processing disorder: a systematic review. *Frontiers in Neurology*, 12, 692840.
- Luck, S. J. (2014). *An introduction to the event-related potential technique* (2nd ed.). MIT press.
- Makeig, S., Jung, T.-P., Ghahremani, D., & Sejnowski, T. J. (1996). Independent component analysis of simulated ERP data. *Institute for Neural Computation, University of California: technical report INC-9606*.
- Mamatha, N. M., & Yathiraj, A. (2020). Comparison of diagnostic auditory processing test scores measured in clinical and school settings. *Language, Speech, and Hearing Services in Schools*, 51(4), 1071-1080.
- McWeeny, S., & Norton, E. S. (2024). Auditory Processing and Reading Disability: A Systematic Review and Meta-Analysis. *Scientific Studies of Reading*, 1-23. <https://doi.org/10.1080/10888438.2023.2252118>
- Mohideen, R. S. P. M., & Thangaraj, M. (2023). Relationship Between Temporal Pattern Perception Test and Mismatch Negativity in Children With Auditory Processing Disorder and Dyslexia. *Journal of Audiology & Otology*, 27(1), 16.
- Moore, D., Campbell, N., Rosen, S., Bamliou, D., Sirimanna, T., Grant, P., & Wakeham, K. (2018). Position statement and practice guidance auditory processing disorder (APD) British Society of Audiology 19pp. In.
- Moore, D. R., Ferguson, M. A., Edmondson-Jones, A. M., Ratib, S., & Riley, A. (2010). Nature of auditory processing disorder in children. *Pediatrics*, 126(2), e382-e390.
- Moore, D. R., & Hunter, L. L. (2013). Auditory processing disorder (APD) in children: a marker of neurodevelopmental syndrome. *Hearing, Balance and Communication*, 11(3), 160-167.
- Musiek, F. E., Shinn, J. B., Jirsa, R., Bamliou, D.-E., Baran, J. A., & Zaida, E. (2005). GIN (Gaps-In-Noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear and hearing*, 26(6), 608-618.
- Näätänen, R. (1992). Event-related potentials and automatic information processing. *Attention and brain function*, 102-210.
- Näätänen, R., & Alho, K. (1997). Mismatch negativity—the measure for central sound representation accuracy. *Audiology and Neurotology*, 2(5), 341-353.
- Näätänen, R., Gaillard, A. W., & Mäntysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. *Acta psychologica*, 42(4), 313-329.

- Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: a review. *Clinical neurophysiology*, 118(12), 2544-2590.
- Näätänen, R., Pakarinen, S., Rinne, T., & Takegata, R. (2004). The mismatch negativity (MMN): towards the optimal paradigm. *Clinical Neurophysiology*, 115(1), 140-144.
- Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology*, 24(4), 375-425.
- Nagao, K., Riegner, T., Padilla, J., Greenwood, L. A., Loson, J., Zavala, S., & Morlet, T. (2016). Prevalence of auditory processing disorder in school-aged children in the Mid-Atlantic region. *Journal of the American Academy of Audiology*, 27(09), 691-700.
- O'Hara, B., & Mealings, K. (2018). Developing the auditory processing domains questionnaire (APDQ): a differential screening tool for auditory processing disorder. *International Journal of Audiology*, 57(10), 764-775.
- Omidvar, S., Campbell, K., Alain, C., Aiken, S., & Koravand, A. (2026). The Effect of the Temporal Position of a Gap on Its Automatic Detection. *European Journal of Neuroscience*, 63(12), e70585.
- Omidvar, S., Duquette-Laplante, F., Bursch, C., Jutras, B., & Koravand, A. (2023). Assessing Auditory Processing in Children with Listening Difficulties: A Pilot Study. *Journal of Clinical Medicine*, 12(3), 897.
- Partanen, E., Torppa, R., Pykäläinen, J., Kujala, T., & Huotilainen, M. (2013). Children's brain responses to sound changes in pseudo words in a multifeature paradigm. *Clinical Neurophysiology*, 124(6), 1132-1138.
- Penner, M. (1977). Detection of temporal gaps in noise as a measure of the decay of auditory sensation. *The Journal of the Acoustical Society of America*, 61(2), 552-557.
- Perrin, F., Pernier, J., Bertrand, O., & Echallier, J. F. (1989). Spherical splines for scalp potential and current density mapping. *Electroencephalography and clinical neurophysiology*, 72(2), 184-187.
- Phillips, D., Taylor, T., Hall, S., Carr, M., & Mossop, J. (1997). Detection of silent intervals between noises activating different perceptual channels: some properties of "central" auditory gap detection. *The Journal of the Acoustical Society of America*, 101(6), 3694-3705.
- Phillips, D. P. (1998). Sensory representations, the auditory cortex, and speech perception. *Seminars in Hearing*, 19(04), 319-332.
- Phillips, D. P. (1999). Auditory gap detection, perceptual channels, and temporal resolution in speech perception. *Journal of the American Academy of Audiology*, 10(06), 343-354.
- Phillips, D. P., Comeau, M., & Andrus, J. N. (2010). Auditory temporal gap detection in children with and without auditory processing disorder. *Journal of the American Academy of Audiology*, 21(06), 404-408.
- Picton, T. W., Bentin, S., Berg, P., Donchin, E., Hillyard, S., Johnson, R., Miller, G., Ritter, W., Ruchkin, D., & Rugg, M. (2000). Guidelines for using human event-related potentials to study cognition: recording standards and publication criteria. *Psychophysiology*, 37(2), 127-152.

- Pinto, J. D., Temp, D. A., Ferreira, L., de Souza, A. E. H., Garcia, M. V., de Andrade, A. N., & Biaggio, E. P. V. (2024). Mismatch Negativity in Children with Deficits in Auditory Abilities. *International Archives of Otorhinolaryngology*, 28(04), e561-e567.
- Pivik, R. T., Broughton, R. J., Coppola, R., Davidson, R. J., Fox, N., & Nuwer, M. R. (1993). Guidelines for the recording and quantitative analysis of electroencephalographic activity in research contexts. *Psychophysiology*, 30(6), 547-558.
- Poeppel, D. (2003). The analysis of speech in different temporal integration windows: cerebral lateralization as ‘asymmetric sampling in time’. *Speech communication*, 41(1), 245-255.
- Riccio, C. A., Hynd, G. W., Cohen, M. J., Hall, J., & Molt, L. (1994). Comorbidity of central auditory processing disorder and attention-deficit hyperactivity disorder. *Journal of the American Academy of Child & Adolescent Psychiatry*, 33(6), 849-857.
- Rocha-Muniz, C. N., Befi-Lopes, D. M., & Schochat, E. (2015). Mismatch negativity in children with specific language impairment and auditory processing disorder. *Brazilian journal of Otorhinolaryngology*, 81(4), 408-415.
- Roggia, S. M., & Colares, N. T. (2008). Mismatch negativity in patients with (central) auditory processing disorders. *Revista Brasileira de Otorrinolaringologia*, 74, 705-711.
- Ross, B., Schneider, B., Snyder, J. S., & Alain, C. (2010). Biological markers of auditory gap detection in young, middle-aged, and older adults. *PloS one*, 5(4), e10101.
- Schröger, E., Näätänen, R., & Paavilainen, P. (1992). Event-related potentials reveal how non-attended complex sound patterns are represented by the human brain. *Neuroscience letters*, 146(2), 183-186.
- Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 104(6), 540-545.
- Sharma, M., Peter, V., Dennis, D., Raman, G., & Purdy, S. C. (2025). Test–Retest Reliability of Mismatch Negativity and Late Discriminative Negativity Response in Children With Listening Difficulties. *European journal of neuroscience*, 61(5), e70065.
- Shreyas, S., & Jain, C. (2024). Two-year prevalence of central auditory processing disorders in children. *Indian Journal of Otolaryngology and Head & Neck Surgery*, 76(4), 3283-3288.
- Silman, S., Silverman, C. A., & Emmer, M. B. (2000). Central auditory processing disorders and reduced motivation: Three case studies. *Journal of the American Academy of Audiology*, 11(02), 57-63.
- Stavrinos, G., Iliadou, V.-M., Edwards, L., Sirimanna, T., & Bamiou, D.-E. (2018). The relationship between types of attention and auditory processing skills: reconsidering auditory processing disorder diagnosis. *Frontiers in psychology*, 9, 34.
- Tallal, P., Miller, S., & Fitch, R. H. (1993). Neurobiological basis of speech: a case for the preeminence of temporal processing. *Annals-New York Academy of Sciences*, 682, 27-47.
- Tamakoshi, S., Minoura, N., Katayama, J. i., & Yagi, A. (2016). Entire sound representations are time-compressed in sensory memory: evidence from MMN. *Frontiers in neuroscience*, 10, Article 347.

- JASP Team. (2026). JASP (Version 0.96.0)[Computer software]. <https://jasp-stats.org/>
- Timm, J., Weise, A., Grimm, S., & Schröger, E. (2011). An asymmetry in the automatic detection of the presence or absence of a frequency modulation within a tone: a mismatch negativity study. *Frontiers in psychology*, 2, 189.
- Todd, J., Finch, B., Smith, E., Budd, T. W., & Schall, U. (2011). Temporal processing ability is related to ear-asymmetry for detecting time cues in sound: A mismatch negativity (MMN) study. *Neuropsychologia*, 49(1), 69-82.
- Uther, M., Jansen, D., Huotilainen, M., Ilmoniemi, R., & Näätänen, R. (2003). Mismatch negativity indexes auditory temporal resolution: evidence from event-related potential (ERP) and event-related field (ERF) recordings. *Cognitive Brain Research*, 17(3), 685-691.
- Walker, M. M., Shinn, J. B., Cranford, J. L., Givens, G. D., & Holbert, D. (2002). Auditory temporal processing performance of young adults with reading disorders. *Journal of Speech, Language, and Hearing Research*, 45(3), 598-605.
- Weise, A., Grimm, S., Müller, D., & Schröger, E. (2010). A temporal constraint for automatic deviance detection and object formation: a mismatch negativity study. *Brain research*, 1331, 88-95.
- Weise, A., Grimm, S., Rimmele, J. M., & Schröger, E. (2023). Auditory representations for long lasting sounds: Insights from event-related brain potentials and neural oscillations. *Brain and language*, 237, 105221.
- Weise, A., Müller, D., Grimm, S., Rübsamen, R., & Schröger, E. (2007). Differential processing of terminal tone parts within structured and non-structured tones. *Neuroscience letters*, 421(2), 163-167.
- Weise, A., Ritter, W., & Schröger, E. (2011). The representation of unattended, segmented sounds: A mismatch negativity (MMN) study. *International journal of psychophysiology*, 81(2), 121-126.
- Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: predictive regularity representations and perceptual objects. *Trends in cognitive sciences*, 13(12), 532-540.
- Woldorff, M. G., Hillyard, S. A., Gallen, C. C., Hampson, S. R., & Bloom, F. E. (1998). Magnetoencephalographic recordings demonstrate attentional modulation of mismatch-related neural activity in human auditory cortex. *Psychophysiology*, 35(3), 283-292.
- Yabe, H., Koyama, S., Kakigi, R., Gunji, A., Tervaniemi, M., Sato, Y., & Kaneko, S. (2001). Automatic discriminative sensitivity inside temporal window of sensory memory as a function of time. *Cognitive Brain Research*, 12(1), 39-48.
- Yabe, H., Matsuoka, T., Sato, Y., Hiruma, T., Sutoh, T., Koyama, S., Gunji, A., Kakigi, R., & Kaneko, S. (2005). Time may be compressed in sound representation as replicated in sensory memory. *Neuroreport*, 16(2), 95-98.

Chapter 6: General discussion

6.1 Overview of the main findings

This thesis examined automatic auditory temporal resolution using ERPs elicited by silent gaps embedded within a long-duration stimulus. The main purposes of this thesis were to develop a novel time-efficient multi-deviant paradigm to examine the effects of gap duration and temporal position, and to determine how these two factors influence the automatic detection of the gap by the change detection system. The present thesis was designed to address these questions in adults, TD children, and children with APD, using both behavioural and electrophysiological measures. Across the three studies, the findings were generally consistent. Longer duration gaps elicited larger DRNs than shorter duration gaps, and the amplitude of the DRN decreased as the gap occurred later in the stimulus. The same general pattern was present in adults and TD children, although the temporal constraint was more pronounced in children. In children with APD, behavioural gap detection was poorer than in TD children. However, overall DRN amplitude did not differ significantly between groups, although some condition-specific reductions and reduced response detectability were observed in the APD group. Together, these findings suggest that automatic neural detection of a gap is influenced not only by gap duration, but also by when the gap occurs within the ongoing sound.

A further important finding of the thesis was methodological. The first study in young adults did establish that a time-efficient multi-deviant paradigm could be used, even though each of the several deviants was physically identical. It also showed that the effect of the temporal position of the gap was consistent in both the single deviant oddball and the multi-deviant paradigms. This was important because if an oddball paradigm had been used for all temporal positions, total testing time would have been exceedingly long. This issue is particularly important in children and in clinical populations, who may not be able to tolerate such a long testing session.

6.2 Behavioural measures

Behavioural gap-detection tasks were included in all three studies to determine whether the timing of the gap within the stimulus affected active detection, and whether this pattern differed across development and in APD. An important limitation of behavioural gap-detection tasks is that performance may reflect not only the ability of the auditory system to detect a brief temporal

discontinuity, but also non-auditory processes required to perform the task, including attention, memory, and response selection (Cacace & McFarland, 2013). This issue is particularly important in children and in clinical groups such as those with APD, because LiDs are often multifactorial and may reflect overlapping deficits in hearing, cognition, language, and auditory processing rather than a purely sensory impairment (Ahmmed, 2021; Bigras et al., 2024; Dillon & Cameron, 2021; Gitto et al., 2025).

6.2.1 Effect of gap duration

Across the three studies, behavioural performance was generally good, especially in adults and for the longer-duration gaps. In the adult study, d' values were very high overall, regardless of gap onset time, and the main effect of gap duration was not significant. Thus, adults were able to detect both the 10- and 30-ms gaps with relative ease. In the study comparing adults and TD children, the same general pattern was observed. Again, the main effect of gap duration was not significant, although adults performed significantly better than children overall. These findings indicate that, at the behavioural level, increasing gap duration from 10 to 30 ms did not strongly affect performance.

The APD findings were different. Children with APD had significantly poorer behavioural performance than TD children across both gap durations and regardless of gap onset time. The poorer gap detection is consistent with previous behavioural studies in children with APD (Cameron et al., 2005; Dias et al., 2012; Mamatha & Yathiraj, 2020; Omidvar et al., 2023). At first glance, this could be interpreted as evidence of impaired bottom-up auditory processing, because successful gap detection depends on the auditory system's ability to encode brief temporal discontinuities in sound. However, these higher gap thresholds are derived from behavioural tasks, and performance on such tasks can also be influenced by non-auditory factors such as attention and motivation. For example, children with listening difficulties have been reported to show slower shifting of auditory attention and greater variability in their responses across auditory-processing tasks (Dhamani et al., 2013; Moore et al., 2010). More broadly, attention and other cognitive processes have been suggested to contribute to the listening difficulties associated with APD (Ferguson et al., 2011; Moore & Hunter, 2013). Thus, poorer behavioural gap-detection performance in children with APD may reflect differences in bottom-up auditory processing but may also be influenced by top-down cognitive factors.

Taken together, the behavioural findings suggest that gap duration had less influence on active performance than might have been expected. Adults and TD children could detect both the 10- and 30-ms gaps quite well, although children performed more poorly overall than adults. In APD, however, behavioural performance was poorer across both gap durations. This pattern suggests that behavioural gap detection is sensitive to group differences, but these differences cannot automatically be interpreted as reflecting a pure temporal-resolution deficit.

6.2.2 Effect of gap onset time

The temporal position of the gap influenced behavioural performance, but this effect differed across groups. In the adult study, the main effect of gap onset time was significant. For the 10-ms gap, there were no significant differences in d' across the different onset times. However, for the 30-ms gap, d' for the gap occurring at 400 ms was significantly reduced compared to all other onset times except for that occurring at 15 ms. Thus, although adults could behaviourally detect the gaps with relative ease, late-occurring gaps were somewhat more difficult to detect.

In the child study, behavioural sensitivity was high overall, though lower in children than in adults. For the 30-ms gap, children performed more poorly when the gap occurred very early or very late after stimulus onset. This is broadly consistent with previous behavioural studies showing that gap position can affect detectability, and that gaps near stimulus boundaries can be more difficult to perceive than those occurring in more central positions (Diedler et al., 2007; Eggermont, 1995; Forrest & Green, 1987; Gay et al., 2020; Penner, 1977; Phillips et al., 1997).

In children with APD, the main effect of gap onset time was also significant, but the interaction between group and gap onset time was not. Children with APD had poorer performance than TD children, regardless of the time of occurrence of the gap. This finding is important. It suggests that the behavioural disadvantage in APD was not restricted to late-occurring gaps but was present across the task more generally. Again, this pattern is consistent with the idea that behavioural gap detection in APD may be influenced by broader task demands, such as sustained attention, rather than reflecting only a deficit in encoding the timing of the gap itself (Chermak et al., 1998; Dawes & Bishop, 2009; Gyldenkerne et al., 2014; Moore et al., 2010; Stavrinos et al., 2018).

Taken together, the behavioural findings indicate that the timing of the gap affects active detection, but this effect is modest in adults, more evident in children, and accompanied by a general reduction in performance in children with APD.

6.3 Electrophysiological measures

The electrophysiological findings were generally consistent across the three studies. Two effects were observed repeatedly. First, longer-duration gaps elicited larger DRNs than shorter-duration gaps. Second, the amplitude of the DRN decreased as the gap onset time was delayed. These two findings were present in adults, in TD children, and, in general form, also in children with APD.

6.3.1 Effect of gap duration

The manipulation of gap duration was successful. In the adult study, DRN amplitude was significantly larger for the 30-ms gap than for the 10-ms gap. This replicates a large number of previous studies showing a larger DRN for longer duration gaps (Alain et al., 2004; Bertoli et al., 2001; Duda-Milloy et al., 2019; Duda et al., 2025; Duda et al., 2020).

A similar effect of gap duration was observed in the study comparing adults and TD children. The DRN amplitude was significantly larger following the presentation of the 30-ms duration gap than following the presentation of the 10-ms duration gap. Thus, the effect of gap duration was present in both children and adults. This indicates that the paradigm was also sensitive to the perceptibility of the gap in the child group. In other words, the child's auditory system showed the expected neural sensitivity to a more salient temporal discontinuity. This extends the adult gap-ERP literature to school-age children and suggests that the basic relationship between deviant and automatic neural response is already present by this age.

There was, however, a difference in DRN amplitude between children and adults when the response was measured relative to the pre-stimulus baseline, with amplitudes being significantly smaller in children. However, when the response was measured peak-to-peak, from the DRN peak to the peak of the subsequent positivity, the overall group difference was no longer significant, although the effects of gap duration and gap onset time remained. This suggests that the smaller baseline-to-peak DRN in children should be interpreted cautiously. It may reflect, at least in part, developmental differences in waveform morphology rather than a simple reduction in automatic

change detection. This interpretation is consistent with the broader developmental ERP literature, which shows that children often have larger positive obligatory responses, less adult-like N1 morphology, and greater overlap between obligatory and mismatch-related activity than adults (Čeponien et al., 2002; Lohvansuu et al., 2013; Mahajan & McArthur, 2015; Shafer et al., 2000; Sharma et al., 1997; Themás et al., 2023; Wunderlich & Cone-Wesson, 2006). In the present case, this issue is likely to be especially important because a gap deviant does not elicit a pure MMN, but rather a composite response that includes both exogenous and deviance-related activity.

The APD study showed the same general pattern. The baseline-to-peak DRN was larger for the 30-ms than for the 10-ms gap deviant, but this difference was not statistically significant. When the data were reanalyzed using a peak-to-peak measure, the amplitudes of the DRN and the following positivity were again much smaller following presentation of the 10-ms gap than following presentation of the 30-ms gap. Thus, the electrophysiological findings in the APD study were also generally consistent with those observed in the adult and TD child studies.

In the APD study, the overall DRN amplitude did not differ significantly between the APD and TD groups. However, the responses in the APD group were noisier and less stable, especially for the 10-ms gap duration. Thus, although the overall amplitude measures did not show a significant group difference, the APD group showed a narrower range of conditions in which a response could be identified, particularly for the shorter gap duration. This pattern may reflect greater response variability in the APD group and possibly a less stable neural representation of brief temporal deviants. Still, given the small sample size of the pilot study, this point should be interpreted cautiously.

6.3.2 Effect of gap onset time

The temporal position of the gap did influence the amplitude of the DRN. In the adult study, for the 30-ms gap, the amplitude of the DRN gradually decreased with increasingly delayed gap onset times. The effect was less consistent for the 10-ms gap, which could be explained by the poorer signal-to-noise ratio at shorter durations. Importantly, a large decrease in the amplitude of the DRN was apparent for gap onsets occurring at 300 and 400 ms. These findings are thus consistent with the temporal distance effect (Grimm et al., 2006; Grimm & Schröger, 2005; Grimm et al., 2004; Hoonhorst et al., 2012; Timm et al., 2011; Weise et al., 2010; Weise et al., 2023; Weise et al., 2007; Weise et al., 2011).

The effect has been explained by temporal integration theory. Sound features are initially extracted and subsequently integrated with previous and subsequent features into a single unit. This single perceptual unit forms the deviant representation in sensory memory. Integration theory, however, limits the integration window to a very short time period, estimated at no more than 300 ms. The DRN gradually decreased in amplitude from 15 to 400 ms. This is consistent with suggestions that stimulus changes that occur early contribute more to the representation in sensory memory than those that occur later. Stimulus change that occurs to the long-duration standard after about 300 ms after stimulus onset, and certainly after 400 ms, will contribute little to the representation of the deviant stimulus. As such, the DRN will be absent or much reduced in amplitude.

In the child study, the DRN also decreased in amplitude as the onset time of the gap deviant was delayed in both children and adults. In adults, a small-amplitude DRN was still apparent when the gap occurred at 400 ms. The TWI was different in children. Confidence interval procedures indicated that the DRN was also absent when the gap was inserted at 200 and 300 ms after stimulus onset. The child data, therefore, suggests that automatic detection of within-stimulus gaps is adult-like in school-age children, provided the deviance occurs relatively soon after stimulus onset. However, the TWI appears shorter in children than in adults, and as a result, the DRN will be much reduced or absent for gaps that occur as early as 200 ms after stimulus onset.

The APD study again showed that the temporal position of the gap affected the electrophysiological response. In both groups, the amplitude of the DRN decreased as the gap occurred later in the stimulus. Thus, the APD data were generally consistent with the findings in adults and TD children. There was no significant overall interaction between group and gap onset time. This means that the general temporal-position effect was present in both APD and TD children.

At the same time, the CI analysis suggested that the pattern of response detectability was somewhat more restricted in the APD group. In TD children, a reliable DRN was present for gap onsets of 15, 50, and 100 ms for both gap durations, and for the 10-ms gap at 200 ms. In contrast, in children with APD, a reliable DRN was present for the 30-ms duration gaps at 15, 50, and 100 ms, whereas for the 10-ms gap, it was clearly present only at 50 ms. Thus, although the overall amplitude measures did not show a significant group difference, the APD group showed a narrower

range of conditions in which a response could be identified, particularly for the shorter gap duration. This pattern may reflect greater response variability in the APD group and possibly a less stable neural representation of brief temporal deviants.

Taken together, the electrophysiological findings across the three studies were remarkably consistent. In adults, TD children, and children with APD, longer-duration gaps elicited larger DRNs, and the DRN amplitude decreased as the gap occurred later in the stimulus. The temporal position effect was therefore observed even when the deviant was a silent gap with abrupt boundaries. At the same time, the effect appeared to emerge earlier in children than in adults, and the APD group showed less stable and less detectable responses in a narrower range of conditions. Therefore, these findings support the view that automatic neural detection of a gap is constrained not only by gap duration but also by the timing of the gap within the ongoing sound.

6.4 Comparing electrophysiological with behavioural measures

A major finding of the present thesis was the dissociation between electrophysiological and behavioural measures of gap detection. In the adult study, the DRN was much reduced when the gap occurred at 300 or 400 ms after stimulus onset. This was not the case for the behavioural data. Overall, participants could readily detect the deviants, even for the gap occurring at 400 ms, when the DRN was very much reduced. Moreover, correlations between DRN amplitude and behavioural performance were not significant. Other studies have also reported that stimulus changes to the standard after 300 ms are easily detected, even though the MMN is much reduced or absent (Hoonhorst et al., 2012; Timm et al., 2011).

The dissociation between electrophysiological and behavioural findings was also present in the children's data. In children, a clear DRN was not visible for gap deviants occurring at 200 ms and later. In adults, the DRN was much reduced when the gap was inserted at 300 ms and absent when it was inserted at 400 ms. It would therefore be expected that when the gap was inserted 200 ms or later for children, and at 300 or 400 ms for adults, performance would be very poor or at chance levels when participants were asked to actively detect the gap. This was not the case. Both groups could easily detect the gap, even if it was inserted well after the onset of the deviant stimulus when the DRN was reduced or absent. Moreover, there were no significant correlations between the amplitude of the DRN and behavioural performance. Behavioural sensitivity was high overall, though lower in children than in adults.

The dissociation of the physiological and behavioural group differences was particularly important in the APD study. The physiological measure, the DRN, did not significantly differ between the two groups. This, however, was not the case for the behavioural data. The gap detection accuracy was significantly poorer in the APD group, regardless of the time of occurrence of the gap. Of course, the absence of a physiological group difference could be due to insufficient statistical power given the small sample size. Still, the sample size was large enough to detect significant differences in the performance measures. Moreover, power analyses were conducted on the physiological data to determine how large the sample needed to be for the physiological differences to attain statistical significance. The power analysis indicated that, in most comparisons, a very large sample size would be needed to achieve significance. However, some condition-specific DRN differences were observed, suggesting that APD-related physiological differences may be present in selected conditions rather than across the response as a whole.

The differences between electrophysiological and behavioural findings may be attributed to variations in task demands and stimulus presentation procedures. During ERP recordings, participants were instructed to ignore the auditory stimuli and attend to a silent video. Stimulus change was thus detected automatically without the need to direct attention to the auditory channel. In the Näätänen model, such automatic detection of change results from a mismatch between the features of the incoming stimulus and what is stored in sensory memory. In contrast, during the behavioural task, participants were required to actively attend to the auditory sequence to determine whether the current stimulus contained a gap. This type of comparison involves the working memory system. The capacity of working memory is much larger and longer lasting than that of sensory memory. Importantly, working memory requires active attention and rehearsal to prevent the fading of previous events (Hitch et al., 2025; Oberauer, 2019; Shevlin, 2020). Weise et al. (2023) suggest that while the integration and storage capacity of auditory sensory memory is very limited, this can be overcome through the involvement of working memory processes. This does come at a cost. The listener must actively attend to the auditory stream (Weise et al., 2023).

The DRN reflects the outcome of a change detection system that operates relatively independently of attention and task demands. Behavioural detection, in contrast, reflects the outcome of several additional processes, including attention, working memory, decision-making, and response selection (Baddeley, 2012; Cacace & McFarland, 2013). For this reason, behavioural measures are not a pure index of sensory temporal processing. This distinction is particularly

important in children and in clinical groups, where non-sensory factors may contribute substantially to performance.

Poorer behavioural performance in APD can be explained by a bottom-up auditory temporal-processing deficit (American Speech-Language-Hearing Association [ASHA], 2005; Dias et al., 2012; Phillips et al., 2010). If this were the only explanation, then a generalized reduction in passive DRN amplitude would also have been expected. This was not observed. The other view considers the poorer behavioural performance in children with APD as a reflection, at least in part, of a possible top-down or non-sensory influence, such as attention, motivation, or task engagement (Cacace & McFarland, 2013; Dhamani et al., 2013; Ferguson et al., 2011; Moore et al., 2010; Moore & Hunter, 2013). This hypothesis is also consistent with the present findings, because behavioural performance was consistently poorer in the APD group, whereas overall DRN amplitude did not differ significantly between groups. However, the condition-specific DRN reductions and reduced response detectability observed in the APD group suggest that top-down factors alone cannot fully explain the APD findings. Therefore, the findings are best interpreted as suggesting that poorer behavioural gap detection in APD may reflect both non-sensory task demands and condition-specific differences in automatic auditory temporal processing. However, given the small and heterogeneous sample size of study 3, the present findings do not allow firm conclusions about the relative contributions of bottom-up and top-down factors to poorer behavioural gap detection in children with APD. Taken together, these findings suggest that good behavioural gap detection can be maintained even when the passive DRN response is reduced or absent, particularly for gaps occurring later within the stimulus.

6.5 Limitations

Several limitations of the present thesis should be acknowledged.

First, the participant samples were not fully independent across the three studies. The adult comparison group in Study 2 was drawn from the adult sample in Study 1, and the TD comparison group in Study 3 was drawn from the TD child sample in Study 2. Therefore, similarities in the adult findings across Studies 1 and 2, and in the TD child findings across Studies 2 and 3, should not be interpreted as independent replications. Rather, these overlapping samples were used to provide consistent comparison groups across studies employing the same experimental paradigm. Second, it should also be noted that the final study was a small pilot study. Only 6 children with

APD and 6 TD children were tested. The power to reject the null hypothesis, to find significant differences between the two groups, was therefore limited. The overall DRN amplitude did not differ significantly between the two groups. Of course, this could be explained by a lack of statistical power because of the small sample size. Still, the sample size was large enough to detect significant differences in the performance measures. Moreover, power analyses were conducted on the physiological data to determine how large the sample needed to be for the physiological differences to attain statistical significance. The power analysis indicated that in most comparisons, a very large sample size would be needed to achieve significance. However, some condition-specific DRN differences were observed, suggesting that APD-related physiological differences may be present in selected conditions rather than across the response as a whole. Thus, the APD findings should still be interpreted cautiously, as the pilot nature of the study limits the strength of any conclusion about the absence of group differences.

Third, the electrophysiological studies used a broadband WC gap paradigm. Although this was appropriate for examining the effect of gap duration and temporal position, it may not be the most sensitive paradigm for revealing APD-related differences. Behavioural studies suggest that BC gap tasks may be more sensitive to group differences in APD (Jain et al., 2025; Phillips et al., 2010). Another point to consider is the nature of the stimulus used in the present study. Only gap deviants were used. It is not yet known whether the same pattern would be obtained with other types of deviants, such as frequency modulations or speech-like contrasts, because the temporal cues relevant for everyday listening often occur within more complex acoustic patterns than broadband noise.

Fourth, the present thesis focused mainly on the amplitude and detectability of the DRN. Other aspects of the response, such as latency or time-course, may also provide useful information about developmental or clinical differences in temporal processing. However, these measures were not examined in the present study. One reason is that their interpretation can be difficult, particularly when the DRN is very small or absent. Under such conditions, identifying a clear response peak becomes less reliable because the waveform may be strongly affected by residual noise. This was especially relevant in the late-gap conditions, where the DRN was often markedly reduced or absent. In addition, developmental differences in waveform morphology made interpretation more difficult in the child study, particularly when the response was measured relative to the pre-stimulus baseline.

Fifth, gap onset time was necessarily confounded with leading marker and trailing-marker duration and proximity to stimulus offset in the present design. Previous studies have shown that marker duration can influence behavioural gap detection. Phillips et al. (1997) found that shortening the leading marker increased gap thresholds primarily for between-channel gaps, whereas Smith et al. (2008) found that increasing the duration of the trailing marker from 10 to 300 ms increased gap-detection thresholds. Importantly, the direction of the trailing-marker effect reported by Smith et al. does not readily explain the reduction observed at later gap onset times in the present studies, because later gaps in the present paradigm had shorter, rather than longer, trailing segments. Nevertheless, because gap onset time, trailing-marker duration, and proximity to stimulus offset varied together, their individual contributions cannot be separated in the present design.

Sixth, the gap onset time was not independent of the temporal context surrounding the gap. Because the total stimulus duration was fixed at 500 ms, gaps occurring later in the stimulus were preceded by progressively longer leading segments and followed by progressively shorter trailing segments. In addition, later gaps occurred increasingly close to stimulus offset. This is important because gap detection can depend on the temporal position of the gap within a finite-duration stimulus. Snell and Hu (1999), for example, found that gap-detection thresholds were elevated when gaps occurred close to either the onset or offset of a noise burst compared with gaps occurring near its temporal centre. Marker duration can also affect gap detection. Smith et al. (2008) reported higher gap-detection thresholds with longer trailing markers, although the direction of this effect does not readily explain the present findings because later gaps in the present study had shorter, rather than longer, trailing segments. Therefore, the reduction in behavioural performance at later gap onset times may partly reflect proximity to stimulus offset or other temporal-context effects, and cannot be attributed solely to the temporal position of the gap.

Another limitation concerns the relatively high overall probability of gap occurrence in the multi-deviant paradigm. Although each specific gap onset time occurred on only 8.3% of trials, a gap was present on 50% of trials. This differs from a traditional oddball paradigm, in which the gap itself is a rare event. Previous work using a similar multi-gap paradigm has raised this concern and reported somewhat smaller DRNs in multi-deviant than in single-deviant conditions (Duda-Milloy et al., 2019). The relatively frequent occurrence of gaps may therefore have reduced the MMN contribution to the DRN. If the MMN contribution was reduced, the measured DRN would

be influenced more strongly by the obligatory N1 response. This is important because the N1 response may vary with the duration of the noise preceding the gap and could therefore contribute to the reduction in DRN amplitude at later gap onset times. However, in Study 1, DRN amplitudes did not differ significantly between matched multi-deviant and single-deviant conditions, and a reduction in DRN amplitude at the later gap position was also observed in the single-deviant paradigm. Therefore, the relatively high probability of gaps in the multi-deviant paradigm does not appear to fully explain the effect of gap onset time. Nevertheless, because the N1 and MMN contributions to the DRN could not be separated, the possible contribution of N1-related effects cannot be excluded.

A related limitation concerns the contribution of obligatory N1 activity to the DRN. Because the DRN elicited by a gap includes both obligatory responses to the acoustic change and MMN activity, changes in the N1 contribution across gap positions could influence the measured DRN. As the gap occurred progressively later in the stimulus, the duration of the noise preceding the gap also increased, potentially altering the state of adaptation of neural populations responding to the post-gap resumption of the noise. Lanting et al. (2013) showed that auditory cortical adaptation increases with increasing duration of preceding acoustic stimulation, indicating that adaptation can accumulate during an ongoing sound. However, when N1 and P2 were examined separately, the effect of adapter duration was significant for P2 but not for N1 amplitude, suggesting that increasing preceding-stimulus duration does not necessarily produce a corresponding monotonic reduction in N1 amplitude. Nevertheless, because the obligatory N1 and MMN contributions to the DRN could not be separated in the present study, a contribution of N1 adaptation to the reduction in DRN amplitude at later gap positions cannot be excluded. Consequently, the observed temporal-position effect cannot be attributed exclusively to changes in MMN associated with the temporal integration window. Finally, although the passive ERP paradigm reduced the role of active attention, it did not eliminate all sources of variability. Factors such as movement artifacts remain important in pediatric recordings (Brooker et al., 2020; Heise et al., 2022). Future studies may therefore benefit from combining ERP measures with independent measures of attention and other non-sensory factors that may influence performance.

6.6 Future directions

Future studies should compare electrophysiological responses across multi-deviant WC and BC gap-detection paradigms. The present thesis used a broadband WC gap stimulus. Although this was appropriate for examining the effects of gap duration and temporal position, behavioural studies suggest that WC or BC gap tasks may be more sensitive to group differences in APD (Jain et al., 2025; Phillips et al., 2010). It is also not yet known whether the same pattern would be obtained with other types of deviants, such as frequency modulations or more speech-like contrasts, because the temporal cues relevant for everyday listening often occur within more complex acoustic patterns than broadband noise.

Future research should also further examine developmental differences in TWI. The present findings suggest that automatic auditory gap detection is more affected by delayed gap position in children than in adults. Future research might therefore examine whether the temporal-position effect is even more pronounced in younger children, and how it changes across development. Such comparisons could yield valuable insights into the maturation of auditory sensory memory and its role in speech and language development (Cabrera & Lau, 2022; Wang et al., 2005). It would also be useful to examine narrower age ranges or developmental subgroups to determine whether maturational changes occur gradually or more abruptly throughout childhood.

A further direction concerns the relation between automatic change detection and task demands. The differences between electrophysiological and behavioural findings suggest that active and passive gap detection do not rely on exactly the same processes. Future studies should therefore examine how task demands and attentional engagement influence DRN amplitude and the temporal constraints of auditory integration. This could be done by directly comparing passive and active listening conditions within the same participants, or by combining ERP measures with independent measures of attention and other non-sensory factors that may influence performance. Such work would help clarify the extent to which later-occurring gaps can be processed automatically and under what conditions top-down factors alter this processing.

Future studies could also examine the EEG data using time-frequency analyses. The present studies focused on the amplitude of the gap-elicited DRN, whereas time-frequency analysis could provide additional information about changes in oscillatory power and phase consistency associated with gap processing. Previous studies have shown that successful gap detection is

associated with specific combinations of neural phase across delta, theta, and alpha frequency bands (Henry et al., 2016), and that pre-gap entrained delta phase and alpha amplitude can predict whether a near-threshold gap will be detected (Cabral-Calderin & Henry, 2022). Gamma activity may also be particularly relevant to auditory temporal resolution. Gap processing has been associated with transient 40-Hz and high-gamma cortical responses (Ross et al., 2010), and individual differences in the preferred gamma-range frequency of the auditory cortex have been associated with behavioural gap-detection thresholds (Baltus & Herrmann, 2015). Time-frequency analyses could therefore determine whether gap onset time and gap duration affect oscillatory activity and whether these effects differ between children and adults or between children with APD and TD children. Such analyses could also examine inter-trial phase consistency, which may help determine whether the greater variability observed in the APD group reflects less consistent neural timing across trials. Finally, the APD findings need to be followed up in a larger sample. The pilot study suggested that children with APD showed poorer behavioural performance, while overall DRN amplitude did not significantly differ from that of TD children. At the same time, the APD group showed greater variability and a narrower range of conditions in which a reliable DRN could be identified. Future studies should therefore include larger samples and determine whether these trends remain stable. It would also be useful to examine whether APD-related differences become more evident when the temporal task places greater demands on central auditory processing, for example, by using BC or speech-like stimuli. Such studies may help clarify whether the poorer behavioural gap detection often observed in APD reflects mainly non-sensory task demands, subtle instability in automatic auditory processing, or both.

6.7 General Conclusions

The present thesis examined automatic neural processing of silent gaps embedded at different temporal positions within a long-duration stimulus. Across the three studies, two findings were consistent. First, longer-duration gaps elicited larger DRNs than shorter gaps. Second, the amplitude of the DRN decreased as the gap occurred later in the stimulus. Together, these findings indicate that automatic acoustic change detection is influenced not only by the salience of the gap, but also by when the gap occurs within the ongoing sound.

The thesis also showed that these effects differ across development. In adults, a clear DRN was elicited across a broader range of gap onset times. In TD children, the same general pattern

was present, but the reduction in DRN amplitude at later gap positions was more pronounced. This pattern is consistent with a stronger temporal constraint on automatic auditory processing in children than in adults. In children with APD, behavioural gap detection was poorer than in TD children. Overall, DRN amplitude did not differ significantly between groups. This dissociation suggests that poorer behavioural performance in APD cannot be attributed simply to a deficit in the early, automatic detection of the gap. At the same time, the greater variability and reduced detectability of the response in some APD conditions suggest that automatic temporal processing may be less stable in this group, but the exploratory nature of the analyses and the small sample prevent firm conclusions. Taken together, the findings support a multifactorial interpretation of auditory temporal difficulties in APD, in which behavioural performance reflects not only auditory sensory processing, but also non-sensory influences.

6.7.26.7.1 *Novel findings of the thesis*

A multi-deviant paradigm can be used efficiently to examine DRN responses to gaps occurring at several temporal positions within a single recording sequence. In adults, DRN amplitude decreases as the gap is inserted later in the deviant stimulus. This finding does not support the hypothesis that stimuli that have abrupt onsets, such as a gap, would overcome the temporal position effect. School-age children also exhibit a clear DRN to silent gaps. The reduction in DRN amplitude at later gap positions is more pronounced in children than in adults, consistent with a stronger temporal constraint on automatic auditory processing.

In children with APD, no overall group difference in DRN amplitude was detected despite poorer behavioural gap detection relative to TD children. Exploratory analyses also indicated greater response variability, reduced detectability, and some condition-specific DRN differences in the APD group. These observations are preliminary and require confirmation in larger, more clearly characterized samples.

References (excluding articles)

American Academy of Audiology. (2010). Guidelines for the diagnosis, treatment, and management of children and adults with central auditory processing disorder. Working Group on Auditory Processing Disorders.

Ahmmed, A. U. (2021). Auditory processing disorder and its comorbidities: A need for consistency in test cutoff scores. *American Journal of Audiology*, 30(1), 128-144.

Alain, C., McDonald, K. L., Ostroff, J. M., & Schneider, B. (2004). Aging: A switch from automatic to controlled processing of sounds? *Psychology and Aging*, 19(1), 125.

Alain, C., Woods, D. L., & Ogawa, K. H. (1994). Brain indices of automatic pattern processing. *Neuroreport*, 6(1), 140-144.

Alanazi, A. A. (2023). Understanding auditory processing disorder: A narrative review. *Saudi Journal of Medicine & Medical Sciences*, 11(4), 275-282.

Amaral, M. I. R. d., & Colella-Santos, M. F. (2010). Temporal resolution: Performance of school-aged children in the GIN-Gaps-in-noise test. *Brazilian Journal of Otorhinolaryngology*, 76, 745-752.

American Speech-Language-Hearing Association. (2005). Central auditory processing disorders [Technical Report]. Working Group on Auditory Processing Disorders.

American Speech-Language-Hearing Association. (n.d.). Central auditory processing disorder [Practice Portal]. <https://www.asha.org/Practice-Portal/Clinical-Topics/Central-Auditory-Processing-Disorder/>

Arnott, S. R., & Alain, C. (2002). Effects of perceptual context on event-related brain potentials during auditory spatial attention. *Psychophysiology*, 39(5), 625-632.

Baddeley, A. (2012). Working memory: Theories, models, and controversies. *Annual Review of Psychology*, 63(1), 1-29.

Balen, S. A., Bretzke, L., Mottecy, C. M., Liebel, G., Boeno, M. R. M., & Gondim, L. M. A. (2009). Temporal resolution in children: Comparing normal hearing, conductive hearing loss and auditory processing disorder. *Revista Brasileira de Otorrinolaringologia*, 75, 123-129.

Baltus, A., & Herrmann, C. S. (2015). Auditory temporal resolution is linked to resonance frequency of the auditory cortex. *International Journal of Psychophysiology*, 98(1), 1-7.

Bamiou, D.-E., Musiek, F., & Luxon, L. (2001). Aetiology and clinical presentations of auditory processing disorders—A review. *Archives of Disease in Childhood*, 85(5), 361-365.

Bauer, P., Burger, M., Kummer, P., Lohscheller, J., Eysholdt, U., & Doellinger, M. (2009). Correlation between psychometric tests and mismatch negativity in preschool children. *Folia Phoniatrica et Logopaedica*, 61(4), 206-216.

Bernstein, J. G., & Grant, K. W. (2009). Auditory and auditory-visual intelligibility of speech in fluctuating maskers for normal-hearing and hearing-impaired listeners. *The Journal of the Acoustical Society of America*, 125(5), 3358-3372.

Bertoli, S., Heimberg, S., Smurzynski, J., & Probst, R. (2001). Mismatch negativity and psychoacoustic measures of gap detection in normally hearing subjects. *Psychophysiology*, 38(2), 334-342.

Bertoli, S., Smurzynski, J., & Probst, R. (2002). Temporal resolution in young and elderly subjects as measured by mismatch negativity and a psychoacoustic gap detection task. *Clinical Neurophysiology*, 113(3), 396-406.

Bigras, J., Lagacé, J., El Mawazini, A., & Lessard-Dostie, H. (2024). Interventions for school-aged children with auditory processing disorder: A scoping review. *Healthcare*, 12(12), 1161.

Bizley, J. K., & Cohen, Y. E. (2013). The what, where and how of auditory-object perception. *Nature Reviews Neuroscience*, 14(10), 693-707.

Boboshko, M. Y., Savenko, I. V., Garbaruk, E. S., Knyazeva, V. M., & Vasilyeva, M. J. (2023). Impact of prematurity on auditory processing in children. *Pathophysiology*, 30(4), 505-521.

Brooker, R. J., Bates, J. E., Buss, K. A., Canen, M. J., Dennis-Tiway, T. A., Gatzke-Kopp, L. M., Hoyniak, C., Klein, D. N., Kujawa, A., & Lahat, A. (2020). Conducting event-related potential (ERP) research with young children. *Journal of Psychophysiology*, 34(3), 137-158.

Buss, E., Hall III, J. W., Porter, H., & Grose, J. H. (2014). Gap detection in school-age children and adults: Effects of inherent envelope modulation and the availability of cues across frequency. *Journal of Speech, Language, and Hearing Research*, 57(3), 1098-1107.

Buss, E., Porter, H. L., Hall III, J. W., & Grose, J. H. (2017). Gap detection in school-age children and adults: Center frequency and ramp duration. *Journal of Speech, Language, and Hearing Research*, 60(1), 172-181.

Cabral-Calderin, Y., & Henry, M. J. (2022). Reliability of neural entrainment in the human auditory system. *The Journal of Neuroscience*, 42(5), 894-908.

Cabrera, L., & Lau, B. K. (2022). The development of auditory temporal processing during the first year of life. *Hearing, Balance and Communication*, 20(3), 155-165.

Cacace, A. T., & McFarland, D. J. (2013). Factors influencing tests of auditory processing: A perspective on current issues and relevant concerns. *Journal of the American Academy of Audiology*, 24(07), 572-589.

Cameron, S., Dillon, H., & Newall, P. (2005). Three case studies of children with suspected auditory processing disorder. *Australian and New Zealand Journal of Audiology*, 27(2), 97-111.

Campbell, K., & Macdonald, M. (2011). The effects of attention and conscious state on the detection of gaps in long duration auditory stimuli. *Clinical Neurophysiology*, 122(4), 738-747. <https://doi.org/10.1016/j.clinph.2010.10.036>

Canadian Interorganizational Steering Group for Speech-Language Pathology and Audiology. (2012). Canadian guidelines on auditory processing disorder in children and adults: Assessment and intervention.

Čeponien, R., Rinne, T., & Näätänen, R. (2002). Maturation of cortical sound processing as indexed by event-related potentials. *Clinical Neurophysiology*, 113(6), 870-882.

Chermak, G. D., & Musiek, F. E. (1997). *Central auditory processing disorders: New perspectives*. Singular Publishing Group.

Chermak, G. D., Somers, E. K., & Seikel, J. A. (1998). Behavioral signs of central auditory processing disorder and attention deficit hyperactivity disorder. *Journal of the American Academy of Audiology*, 9(1).

Chowsilpa, S., Bamiou, D.-E., & Koohi, N. (2021). Effectiveness of the auditory temporal ordering and resolution tests to detect central auditory processing disorder in adults with evidence of brain pathology: A systematic review and meta-analysis. *Frontiers in Neurology*, 12, 656117.

Colin, C., Hoonhorst, I., Markessis, E., Radeau, M., De Tourtchaninoff, M., Foucher, A., Collet, G., & Deltenre, P. (2009). Mismatch negativity (MMN) evoked by sound duration contrasts: An unexpected major effect of deviance direction on amplitudes. *Clinical Neurophysiology*, 120(1), 51-59.

Cowan, N. (1984). On short and long auditory stores. *Psychological Bulletin*, 96(2), 341-370.

Dawes, P., & Bishop, D. (2009). Auditory processing disorder in relation to developmental disorders of language, communication and attention: A review and critique. *International Journal of Language & Communication Disorders*, 44(4), 440-465.

Dawes, P., Bishop, D. V., Sirimanna, T., & Bamiou, D.-E. (2008). Profile and aetiology of children diagnosed with auditory processing disorder (APD). *International Journal of Pediatric Otorhinolaryngology*, 72(4), 483-489.

Desjardins, R. N., Trainor, L. J., Hevenor, S. J., & Polak, C. P. (1999). Using mismatch negativity to measure auditory temporal resolution thresholds. *Neuroreport*, 10(10), 2079-2082.

Dhamani, I., Leung, J., Carlile, S., & Sharma, M. (2013). Switch attention to listen. *Scientific Reports*, 3(1), 1297.

Dias, K. Z., Jutras, B., Acrani, I. O., & Pereira, L. D. (2012). Random Gap Detection Test (RGDT) performance of individuals with central auditory processing disorders from 5 to 25 years of age. *International Journal of Pediatric Otorhinolaryngology*, 76(2), 174-178.

Diedler, J., Pietz, J., Bast, T., & Rupp, A. (2007). Auditory temporal resolution in children assessed by magnetoencephalography. *Neuroreport*, 18(16), 1691-1695.

Dillon, H., & Cameron, S. (2021). Separating the causes of listening difficulties in children. *Ear and Hearing*, 42(5), 1097.

Drosos, K., Papanicolaou, A., Voniati, L., Panayidou, K., & Thodi, C. (2024). Auditory processing and speech-sound disorders. *Brain Sciences*, 14(3), 291.

Duda-Milloy, V., Tavakoli, P., Campbell, K., Benoit, D. L., & Koravand, A. (2019). A time-efficient multi-deviant paradigm to determine the effects of gap duration on the mismatch negativity. *Hearing Research*, 377, 34-43.

Duda, V., Augereau, T. M., Campbell, K., & Koravand, A. (2025). Does masking alter the mismatch negativity response to gaps? *Brain Research*, 149651. <https://doi.org/10.1016/j.brainres.2025.149651>

Duda, V., Campbell, K., & Koravand, A. (2020). Event-related potentials following gaps in noise: The effects of the intensity of preceding noise. *Brain Research*, 1748, 147078. <https://doi.org/10.1016/j.brainres.2020.147078>

Eggermont, J. (1995). Neural correlates of gap detection and auditory fusion in cat auditory cortex. *Neuroreport*, 6(12), 1645-1648.

Elangovan, S., & Stuart, A. (2008). Natural boundaries in gap detection are related to categorical perception of stop consonants. *Ear and Hearing*, 29(5), 761-774.

Ferguson, M. A., Hall, R. L., Riley, A., & Moore, D. R. (2011). Communication, listening, cognitive and speech perception skills in children with auditory processing disorder (APD) or specific language impairment (SLI). *Journal of Speech, Language, and Hearing Research*, 54(1), 211-227.

Forrest, T., & Green, D. M. (1987). Detection of partially filled gaps in noise and the temporal modulation transfer function. *The Journal of the Acoustical Society of America*, 82(6), 1933-1943.

Gay, J. D., Rosen, M. J., & Huyck, J. J. (2020). Effects of gap position on perceptual gap detection across late childhood and adolescence. *Journal of the Association for Research in Otolaryngology*, 21(3), 243-258.

Gitto, M., Motta, N., Aldè, M., Zanetti, D., & Di Berardino, F. (2025). Auditory processing disorders: Navigating the diagnostic maze of central hearing losses. *Journal of Clinical Medicine*, 14(7), 2256.

Gomes, H., Bernstein, R., Ritter, W., Vaughan, H. G., & Miller, J. (1997). Storage of feature conjunction in transient auditory memory. *Psychophysiology*, 34(6), 712-716.

Grimm, S., Roeber, U., Trujillo-Barreto, N. J., & Schröger, E. (2006). Mechanisms for detecting auditory temporal and spectral deviations operate over similar time windows but are divided differently between the two hemispheres. *Neuroimage*, 32(1), 275-282.

Grimm, S., & Schröger, E. (2005). Pre-attentive and attentive processing of temporal and frequency characteristics within long sounds. *Cognitive Brain Research*, 25(3), 711-721.

Grimm, S., Widmann, A., & Schröger, E. (2004). Differential processing of duration changes within short and long sounds in humans. *Neuroscience Letters*, 356(2), 83-86.

Gudkar, A. I., Dillon, H., Heinrich, A., Nixon, G., Gurteen, H., Burgoyne, K., Cameron, S., Zhou, X., & Tomlin, D. (2026). Establishing normative data for five new temporal auditory processing tests in primary-school aged children. *International Journal of Audiology*, 1-15. <https://doi.org/10.1080/14992027.2026.2614523>

Gyldenkerne, P., Dillon, H., Sharma, M., & Purdy, S. C. (2014). Attend to this: The relationship between auditory processing disorders and attention deficits. *Journal of the American Academy of Audiology*, 25(07), 676-687.

Hautus, M. J., Setchell, G. J., Waldie, K. E., & Kirk, I. J. (2003). Age-related improvements in auditory temporal resolution in reading-impaired children. *Dyslexia*, 9(1), 37-45.

He, S., Grose, J. H., Teagle, H. F., Woodard, J., Park, L. R., Hatch, D. R., & Buchman, C. A. (2013). Gap detection measured with electrically evoked auditory event-related potentials and speech-perception abilities in children with auditory neuropathy spectrum disorder. *Ear and Hearing*, 34(6), 733-744.

Heinrich, A., Alain, C., & Schneider, B. A. (2004). Within- and between-channel gap detection in the human auditory cortex. *Neuroreport*, 15(13), 2051-2056.

Heise, M. J., Mon, S. K., & Bowman, L. C. (2022). Utility of linear mixed effects models for event-related potential research with infants and children. *Developmental Cognitive Neuroscience*, 54, 101070.

Henry, M. J., Herrmann, B., & Obleser, J. (2016). Neural microstates govern perception of auditory input without rhythmic structure. *The Journal of Neuroscience*, 36(3), 860-871.

Hestvik, A., Morlet, T., Nagao, K., & Han, C. (2023). Auditory processing disorder targets phonetics, not phonology. *Proceedings of the Annual Boston University Conference on Language Development*, 2023(1), 356.

Hind, S. E., Haines-Bazrafshan, R., Benton, C. L., Brassington, W., Towle, B., & Moore, D. R. (2011). Prevalence of clinical referrals having hearing thresholds within normal limits. *International Journal of Audiology*, 50(10), 708-716.

Hitch, G. J., Allen, R. J., & Baddeley, A. D. (2025). The multicomponent model of working memory fifty years on. *Quarterly Journal of Experimental Psychology*, 78(2), 222-239.

Hoonhorst, I., Deltenre, P., Markessis, E., Collet, G., Martin, X. P., & Colin, C. (2012). Evidence for a dual versus single origin of the MMNs evoked by cued versus cueless deviants. *Clinical Neurophysiology*, 123(8), 1561-1567.

Hoover, E., Pasquesi, L., & Souza, P. (2015). Comparison of clinical and traditional gap detection tests. *Journal of the American Academy of Audiology*, 26(06), 540-546.

Iliadou, V., Bamiou, D.-E., Chermak, G. D., & Nimatoudis, I. (2014). Comparison of two tests of auditory temporal resolution in children with central auditory processing disorder, adults with psychosis, and adult professional musicians. *International Journal of Audiology*, 53(8), 507-513.

Ismaail, N. M., Shalaby, A. A., & Ibraheem, O. A. (2019). Effect of age on gaps-in-noise test in pediatric population. *International Journal of Pediatric Otorhinolaryngology*, 122, 155-160.

Jain, C., Tanniru, K., & Kalarikkal, G. (2025). Performance of children with and without auditory processing disorders in adaptive temporal gap detection measures. *International Archives of Otorhinolaryngology*, 29(04), 001-007.

Jaramillo, M., Paavilainen, P., & Näätänen, R. (2000). Mismatch negativity and behavioural discrimination in humans as a function of the magnitude of change in sound duration. *Neuroscience Letters*, 290(2), 101-104.

Keith, R. W. (2000). *Random gap detection test*. Auditec.

Koravand, A., Jutras, B., & Lassonde, M. (2017). Abnormalities in cortical auditory responses in children with central auditory processing disorder. *Neuroscience*, 346, 135-148.

Lister, J. J., & Roberts, R. A. (2005). Effects of age and hearing loss on gap detection and the precedence effect. *Journal of Speech, Language, and Hearing Research*, 48, 482-493.

Lister, J. J., Roberts, R. A., Shackelford, J., & Rogers, C. L. (2006). An adaptive clinical test of temporal resolution. *American Journal of Audiology*, 15, 133-140.

Lohvansuu, K., Hämäläinen, J. A., Tanskanen, A., Bartling, J., Bruder, J., Honbolygó, F., Schulte-Körne, G., Démonet, J. F., Csépe, V., & Leppänen, P. H. (2013). Separating mismatch negativity (MMN) response from auditory obligatory brain responses in school-aged children. *Psychophysiology*, 50(7), 640-652.

Lyytinen, H., Blomberg, A. P., & Näätänen, R. (1992). Event-related potentials and autonomic responses to a change in unattended auditory stimuli. *Psychophysiology*, 29(5), 523-534.

Mahajan, Y., & McArthur, G. (2015). Maturation of mismatch negativity and P3a response across adolescence. *Neuroscience Letters*, 587, 102-106.

Mamatha, N. M., & Yathiraj, A. (2020). Comparison of diagnostic auditory processing test scores measured in clinical and school settings. *Language, Speech, and Hearing Services in Schools*, 51(4), 1071-1080.

Marculino, C. F., Rabelo, C. M., & Schochat, E. (2011). Gaps-in-Noise test: Gap detection thresholds in 9-year-old normal-hearing children. *Jornal da Sociedade Brasileira de Fonoaudiologia*, 23, 364-367.

McWeeny, S., & Norton, E. S. (2023). Auditory processing and reading disability: A systematic review and meta-analysis. *Scientific Studies of Reading*, 1-23. <https://doi.org/10.1080/10888438.2023.2252118>

Mohideen, R. S. P. M., & Thangaraj, M. (2023). Relationship between temporal pattern perception test and mismatch negativity in children with auditory processing disorder and dyslexia. *Journal of Audiology & Otology*, 27(1), 16.

Moore, D. R., Campbell, N., Rosen, S., Bamiau, D. E., Sirimanna, T., Grant, P., & Wakeham, K. (2018). Position statement and practice guidance auditory processing disorder (APD). British Society of Audiology.

Moore, D. R., Ferguson, M. A., Edmondson-Jones, A. M., Ratib, S., & Riley, A. (2010). Nature of auditory processing disorder in children. *Pediatrics*, 126(2), e382-e390.

Moore, D. R., & Hunter, L. L. (2013). Auditory processing disorder (APD) in children: A marker of neurodevelopmental syndrome. *Hearing, Balance and Communication*, 11(3), 160-167.

Moore, D. R., Lin, L., Bhalerao, R., Caldwell-Kurtzman, J., & Hunter, L. L. (2025). Multidisciplinary clinical assessment and interventions for childhood listening difficulty and auditory processing disorder: Relation between research findings and clinical practice. *Journal of Speech, Language, and Hearing Research*, 68(6), 2978-2991.

Mulle, J. L. (2012). In search of an electrophysiological correlate to between-channel modulation gap detection (Doctoral dissertation, Dalhousie University).

Muller-Gass, A., Stelmack, R. M., & Campbell, K. B. (2005). "... and were instructed to read a self-selected book while ignoring the auditory stimuli": The effects of task demands on the mismatch negativity. *Clinical Neurophysiology*, 116(9), 2142-2152.

Muller-Gass, A., Stelmack, R. M., & Campbell, K. B. (2006). The effect of visual task difficulty and attentional direction on the detection of acoustic change as indexed by the mismatch negativity. *Brain Research*, 1078(1), 112-130.

Musiek, F. E. (1994). Frequency (pitch) and duration pattern tests. *Journal of the American Academy of Audiology*, 5(4), 265-268.

Musiek, F. E., Shinn, J. B., Jirsa, R., Bamiau, D.-E., Baran, J. A., & Zaida, E. (2005). GIN (Gaps-In-Noise) test performance in subjects with confirmed central auditory nervous system involvement. *Ear and Hearing*, 26(6), 608-618.

Näätänen, R. (1990). The role of attention in auditory information processing as revealed by event-related potentials and other brain measures of cognitive function. *Behavioral and Brain Sciences*, 13(2), 201-233.

Näätänen, R. (1992). Event-related potentials and automatic information processing. *Attention and Brain Function*, 102-210.

Näätänen, R., Gaillard, A. W., & Mäntysalo, S. (1978). Early selective-attention effect on evoked potential reinterpreted. *Acta Psychologica*, 42(4), 313-329.

Näätänen, R., Jacobsen, T., & Winkler, I. (2005). Memory-based or afferent processes in mismatch negativity (MMN): A review of the evidence. *Psychophysiology*, 42(1), 25-32.

Näätänen, R., Jiang, D., Lavikainen, J., Reinikainen, K., & Paavilainen, P. (1993). Event-related potentials reveal a memory trace for temporal features. *Neuroreport*, 5(3), 310-312.

Näätänen, R., Kujala, T., & Winkler, I. (2011). Auditory processing that leads to conscious perception: A unique window to central auditory processing opened by the mismatch negativity and related responses. *Psychophysiology*, 48(1), 4-22.

Näätänen, R., Paavilainen, P., Rinne, T., & Alho, K. (2007). The mismatch negativity (MMN) in basic research of central auditory processing: A review. *Clinical Neurophysiology*, 118(12), 2544-2590.

Näätänen, R., Pakarinen, S., Rinne, T., & Takegata, R. (2004). The mismatch negativity (MMN): Towards the optimal paradigm. *Clinical Neurophysiology*, 115(1), 140-144.

Näätänen, R., & Picton, T. (1987). The N1 wave of the human electric and magnetic response to sound: A review and an analysis of the component structure. *Psychophysiology*, 24(4), 375-425.

Näätänen, R., & Winkler, I. (1999). The concept of auditory stimulus representation in cognitive neuroscience. *Psychological Bulletin*, 125(6), 826-859.

Nagao, K., Riegner, T., Padilla, J., Greenwood, L. A., Loson, J., Zavala, S., & Morlet, T. (2016). Prevalence of auditory processing disorder in school-aged children in the Mid-Atlantic region. *Journal of the American Academy of Audiology*, 27(09), 691-700.

Negri, L., Oliver, P., Mitchell, R., Sinha, L., Kearney, J., Saad, D., Nodal, F. R., & Bajo, V. M. (2024). Optimization of the operant silent gap-in-noise detection paradigm in humans. *Journal of Integrative Neuroscience*, 23(10), 183.

Nordby, H., Roth, W. T., & Pfefferbaum, A. (1988). Event-related potentials to breaks in sequences of alternating pitches or interstimulus intervals. *Psychophysiology*, 25(3), 262-268.

Oberauer, K. (2019). Is rehearsal an effective maintenance strategy for working memory? *Trends in Cognitive Sciences*, 23(9), 798-809.

Okazaki, S., Kanoh, S. i., Takaura, K., Tsukada, M., & Oka, K. (2006). Change detection and difference detection of tone duration discrimination. *Neuroreport*, 17(4), 395-399.

Omidvar, S., Duquette-Laplane, F., Bursch, C., Jutras, B., & Koravand, A. (2023). Assessing auditory processing in children with listening difficulties: A pilot study. *Journal of Clinical Medicine*, 12(3), 897.

Palmer, S. B., & Musiek, F. E. (2013). N1-P2 recordings to gaps in broadband noise. *Journal of the American Academy of Audiology*, 24(1), 37-45. <https://doi.org/10.3766/jaaa.24.1.5>

Partanen, E., Torppa, R., Pykäläinen, J., Kujala, T., & Huotilainen, M. (2013). Children's brain responses to sound changes in pseudo words in a multifeature paradigm. *Clinical Neurophysiology*, 124(6), 1132-1138.

Penner, M. (1977). Detection of temporal gaps in noise as a measure of the decay of auditory sensation. *The Journal of the Acoustical Society of America*, 61(2), 552-557.

Perez, A. P., & Pereira, L. D. (2010). The gap in noise test in 11 and 12-year-old children. *Pró-Fono Revista de Atualização Científica*, 22, 7-12.

Petermann, M., Kummer, P., Burger, M., Lohscheller, J., Eysholdt, U., & Döllinger, M. (2009). Statistical detection and analysis of mismatch negativity derived by a multi-deviant design from normal hearing children. *Hearing Research*, 247(2), 128-136.

Petley, L., Hunter, L. L., Zadeh, L. M., Stewart, H. J., Sloat, N. T., Perdew, A., Lin, L., & Moore, D. R. (2021). Listening difficulties in children with normal audiograms: Relation to hearing and cognition. *Ear and Hearing*, 42(6), 1640-1655.

Phillips, D., Taylor, T., Hall, S., Carr, M., & Mossop, J. (1997). Detection of silent intervals between noises activating different perceptual channels: Some properties of "central" auditory gap detection. *The Journal of the Acoustical Society of America*, 101(6), 3694-3705.

Phillips, D. P. (1998, November). Sensory representations, the auditory cortex, and speech perception. *Seminars in Hearing*, 19(04), 319-332.

Phillips, D. P. (1999). Auditory gap detection, perceptual channels, and temporal resolution in speech perception. *Journal of the American Academy of Audiology*, 10(06), 343-354.

Phillips, D. P., Comeau, M., & Andrus, J. N. (2010). Auditory temporal gap detection in children with and without auditory processing disorder. *Journal of the American Academy of Audiology*, 21(06), 404-408.

Phillips, D. P., & Smith, J. C. (2004). Correlations among within-channel and between-channel auditory gap-detection thresholds in normal listeners. *Perception*, 33(3), 371-378.

Pihko, E., Leppäsaari, T., Leppänen, P., Richardson, U., & Lyytinen, H. (1997). Auditory event-related potentials (ERP) reflect temporal changes in speech stimuli. *Neuroreport*, 8(4), 911-914.

Pinto, J. D., Temp, D. A., Ferreira, L., de Souza, A. E. H., Garcia, M. V., de Andrade, A. N., & Biaggio, E. P. V. (2024). Mismatch negativity in children with deficits in auditory abilities. *International Archives of Otorhinolaryngology*, 28(04), e561-e567.

Pratt, H., Starr, A., Michalewski, H. J., Bleich, N., & Mittelman, N. (2007). The N1 complex to gaps in noise: Effects of preceding noise duration and intensity. *Clinical Neurophysiology*, 118(5), 1078-1087. <https://doi.org/10.1016/j.clinph.2007.01.005>

Putkinen, V., Tervaniemi, M., Saarikivi, K., Ojala, P., & Huotilainen, M. (2014). Enhanced development of auditory change detection in musically trained school-aged children: A longitudinal event-related potential study. *Developmental Science*, 17(2), 282-297.

Rapaport, H., Seymour, R. A., Benikos, N., He, W., Pellicano, E., Brock, J., & Sowman, P. F. (2023). Investigating predictive coding in younger and older children using MEG and a multi-feature auditory oddball paradigm. *Cerebral Cortex*, 33(12), 7489-7499.

Rocha-Muniz, C. N., Befi-Lopes, D. M., & Schochat, E. (2015). Mismatch negativity in children with specific language impairment and auditory processing disorder. *Brazilian Journal of Otorhinolaryngology*, 81(4), 408-415.

Roggia, S. M., & Colares, N. T. (2008). Mismatch negativity in patients with (central) auditory processing disorders. *Revista Brasileira de Otorrinolaringologia*, 74, 705-711.

Ross, B., Schneider, B., Snyder, J. S., & Alain, C. (2010). Biological markers of auditory gap detection in young, middle-aged, and older adults. *PloS one*, 5(4), e10101.

Sabri, M., De Lugt, D. R., & Campbell, K. B. (2000). The mismatch negativity to frequency deviants during the transition from wakefulness to sleep. *Canadian Journal of Experimental Psychology/Revue Canadienne de Psychologie Expérimentale*, 54(4), 230.

Schröger, E., Näätänen, R., & Paavilainen, P. (1992). Event-related potentials reveal how non-attended complex sound patterns are represented by the human brain. *Neuroscience Letters*, 146(2), 183-186.

Shafer, V. L., Morr, M. L., Kreuzer, J. A., & Kurtzberg, D. (2000). Maturation of mismatch negativity in school-age children. *Ear and Hearing*, 21(3), 242-251.

Sharma, A., Kraus, N., McGee, T. J., & Nicol, T. G. (1997). Developmental changes in P1 and N1 central auditory responses elicited by consonant-vowel syllables. *Electroencephalography and Clinical Neurophysiology/Evoked Potentials Section*, 104(6), 540-545.

Sharma, M., Peter, V., Dennis, D., Raman, G., & Purdy, S. C. (2025). Test-retest reliability of mismatch negativity and late discriminative negativity response in children with listening difficulties. *European Journal of Neuroscience*, 61(5), e70065.

Shevlin, H. (2020). Current controversies in the cognitive science of short-term memory. *Current Opinion in Behavioral Sciences*, 32, 148-154.

Shinn, J. B., Chermak, G. D., & Musiek, F. E. (2009). GIN (Gaps-In-Noise) performance in the pediatric population. *Journal of the American Academy of Audiology*, 20(04), 229-238.

Shreyas, S. R., & Jain, C. (2024). Two-year prevalence of central auditory processing disorders in children. *Indian Journal of Otolaryngology and Head & Neck Surgery*, 76(4), 3283-3288.

Stavrinos, G., Iliadou, V.-M., Edwards, L., Sirimanna, T., & Bamio, D.-E. (2018). The relationship between types of attention and auditory processing skills: Reconsidering auditory processing disorder diagnosis. *Frontiers in Psychology*, 9, 34.

Takegata, R., Heikkilä, R., & Näätänen, R. (2009). Neural process underlying gap detection for spectrally rich and asymmetrical markers. *Neuroreport*, 20(12), 1120-1124.

Tallal, P., Miller, S., & Fitch, R. H. (1993). Neurobiological basis of speech: A case for the preeminence of temporal processing. *Annals of the New York Academy of Sciences*, 682, 27-47.

Tamakoshi, S., Minoura, N., Katayama, J. i., & Yagi, A. (2016). Entire sound representations are time-compressed in sensory memory: Evidence from MMN. *Frontiers in Neuroscience*, 10, Article 347.

Tervaniemi, M., Saarinen, J., Paavilainen, P., Danilova, N., & Näätänen, R. (1994). Temporal integration of auditory information in sensory memory as reflected by the mismatch negativity. *Biological Psychology*, 38(2-3), 157-167.

Themas, L., Lippus, P., Padrik, M., Kask, L., & Kreegipuu, K. (2023). Maturation of the mismatch response in pre-school children: Systematic literature review and meta-analysis. *Neuroscience & Biobehavioral Reviews*, 153, 105366.

Timm, J., Weise, A., Grimm, S., & Schröger, E. (2011). An asymmetry in the automatic detection of the presence or absence of a frequency modulation within a tone: A mismatch negativity study. *Frontiers in Psychology*, 2, 189.

Todd, J., Finch, B., Smith, E., Budd, T. W., & Schall, U. (2011). Temporal processing ability is related to ear-asymmetry for detecting time cues in sound: A mismatch negativity (MMN) study. *Neuropsychologia*, 49(1), 69-82.

Tomlin, D., Dillon, H., Sharma, M., & Rance, G. (2015). The impact of auditory processing and cognitive abilities in children. *Ear and hearing*, 36(5), 527-542.

Trainor, L., McFadden, M., Hodgson, L., Darragh, L., Barlow, J., Matsos, L., & Sonnadara, R. (2003). Changes in auditory cortex and the development of mismatch negativity between 2 and 6 months of age. *International Journal of Psychophysiology*, 51(1), 5-15.

Trainor, L. J., Samuel, S. S., Desjardins, R. N., & Sonnadara, R. R. (2001). Measuring temporal resolution in infants using mismatch negativity. *Neuroreport*, 12(11), 2443-2448.

Uhlén, I., Engström, E., Kallioinen, P., Nakeva von Mentzer, C., Lyxell, B., Sahlén, B., Lindgren, M., & Ors, M. (2017). Using a multi-feature paradigm to measure mismatch responses

to minimal sound contrasts in children with cochlear implants and hearing aids. *Scandinavian Journal of Psychology*, 58(5), 409-421.

Uther, M., Jansen, D., Huotilainen, M., Ilmoniemi, R., & Näätänen, R. (2003). Mismatch negativity indexes auditory temporal resolution: Evidence from event-related potential (ERP) and event-related field (ERF) recordings. *Cognitive Brain Research*, 17(3), 685-691.

Walker, M. M., Shinn, J. B., Cranford, J. L., Givens, G. D., & Holbert, D. (2002). Auditory temporal processing performance of young adults with reading disorders. *Journal of Speech, Language, and Hearing Research*, 45(3), 598-605.

Wang, W., Datta, H., & Sussman, E. (2005). The development of the length of the temporal window of integration for rapidly presented auditory information as indexed by MMN. *Clinical Neurophysiology*, 116(7), 1695-1706.

Weise, A., Grimm, S., Müller, D., & Schröger, E. (2010). A temporal constraint for automatic deviance detection and object formation: A mismatch negativity study. *Brain Research*, 1331, 88-95.

Weise, A., Grimm, S., Rimmele, J. M., & Schröger, E. (2023). Auditory representations for long lasting sounds: Insights from event-related brain potentials and neural oscillations. *Brain and Language*, 237, 105221.

Weise, A., Müller, D., Grimm, S., Rübsamen, R., & Schröger, E. (2007). Differential processing of terminal tone parts within structured and non-structured tones. *Neuroscience Letters*, 421(2), 163-167.

Weise, A., Ritter, W., & Schröger, E. (2011). The representation of unattended, segmented sounds: A mismatch negativity (MMN) study. *International Journal of Psychophysiology*, 81(2), 121-126.

Wightman, F., Allen, P., Dolan, T., Kistler, D., & Jamieson, D. (1989). Temporal resolution in children. *Child Development*, 60, 611-624.

Wilson, W. J. (2013). *Screening for central auditory processing disorder*. In F. E. Musiek & G. D. Chermak (Eds.), *Handbook of central auditory processing disorder* (Vol. 1, pp. 265-290). Plural Publishing.

Winkler, I., Denham, S. L., & Nelken, I. (2009). Modeling the auditory scene: Predictive regularity representations and perceptual objects. *Trends in Cognitive Sciences*, 13(12), 532-540.

Woldorff, M. G., Hillyard, S. A., Gallen, C. C., Hampson, S. R., & Bloom, F. E. (1998). Magnetoencephalographic recordings demonstrate attentional modulation of mismatch-related neural activity in human auditory cortex. *Psychophysiology*, 35(3), 283-292.

Wunderlich, J. L., & Cone-Wesson, B. K. (2006). Maturation of CAEP in infants and children: A review. *Hearing Research*, 212(1-2), 212-223.

Yabe, H., Koyama, S., Kakigi, R., Gunji, A., Tervaniemi, M., Sato, Y., & Kaneko, S. (2001). Automatic discriminative sensitivity inside temporal window of sensory memory as a function of time. *Cognitive Brain Research*, 12(1), 39-48.

Yabe, H., Matsuoka, T., Sato, Y., Hiruma, T., Sutoh, T., Koyama, S., Gunji, A., Kakigi, R., & Kaneko, S. (2005). Time may be compressed in sound representation as replicated in sensory memory. *Neuroreport*, 16(2), 95-98.

Appendices

Appendix 1. Ethics approval

Université d'Ottawa

Bureau d'éthique et d'intégrité de la recherche

University of Ottawa

Office of Research Ethics and Integrity

CERTIFICAT D'APPROBATION ÉTHIQUE | CERTIFICATE OF ETHICS APPROVAL

Numéro du dossier / Ethics File Number

H-07-24-10412

Titre du projet / Project Title

Investigating Temporal
Resolution in Children with
Listening Difficulties Using a
Novel Multi-Deviant Paradigm

Type de projet / Project Type

Thèse de doctorat / Doctoral
thesis

Statut du projet / Project Status

Renouvelé / Renewed

Date d'approbation (jj/mm/aaaa) / Approval Date (dd/mm/yyyy)

20/09/2024

Date d'expiration (jj/mm/aaaa) / Expiry Date (dd/mm/yyyy)

19/09/2026

Équipe de recherche / Research Team

Chercheur /

Affiliation

Role

Researcher

Shaghayegh OMIDVAR

École des sciences de la réadaptation / School of
Rehabilitation Sciences
École des sciences de la réadaptation / School of

Chercheur Principal / Principal
Investigator

Amineh KORAVAND

Rehabilitation Sciences
Département de biologie / Department of Biology

Superviseur / Supervisor

William KHAWAM

Autre / Other

Conditions spéciales ou commentaires / Special conditions or comments

550, rue Cumberland, pièce 154 550 Cumberland Street, Room 154 Ottawa (Ontario) K1N 6N5 Canada Ottawa,
Ontario K1N 6N5 Canada 613-562-5387 • 613-562-5338 • ethique@uOttawa.ca / ethics@uOttawa.ca
www.recherche.uottawa.ca/deontologie | www.recherche.uottawa.ca/ethics 22/08/2025

Le Comité d'éthique de la recherche (CÉR) de l'Université d'Ottawa, opérant conformément à l'*Énoncé de politique des Trois conseils* (2014) et toutes autres lois et tous règlements applicables, a examiné et approuvé la demande d'éthique du projet de recherche ci-nommé.

L'approbation est valide pour la durée indiquée plus haut et est sujette aux conditions énumérées dans la section intitulée "Conditions Spéciales ou Commentaires". Le formulaire « Renouvellement ou Fermeture de Projet » doit être complété quatre semaines avant la date d'échéance indiquée ci-haut afin de demander un renouvellement de cette approbation éthique ou afin de fermer le dossier.

Toutes modifications apportées au projet doivent être approuvées par le CÉR avant leur mise en place, sauf si le participant doit être retiré en raison d'un danger immédiat ou s'il s'agit d'un changement ayant trait à des éléments administratifs ou logistiques du projet. Les chercheurs doivent aviser le CÉR dans les plus brefs délais de tout changement pouvant augmenter le niveau de risque aux participants ou pouvant affecter considérablement le déroulement du projet, rapporter tout événement imprévu ou indésirable et soumettre toute nouvelle information pouvant nuire à la conduite du projet ou à la sécurité des participants.

The University of Ottawa Research Ethics Board, which operates in accordance with the *Tri-Council Policy Statement* (2014) and other applicable laws and regulations, has examined and approved the ethics application for the above-named research project.

Ethics approval is valid for the period indicated above and is subject to the conditions listed in the section entitled "Special Conditions or Comments". The "Renewal/Project Closure" form must be completed four weeks before the above-referenced expiry date to request a renewal of this ethics approval or closure of the file.

Any changes made to the project must be approved by the REB before being implemented, except when necessary to remove participants from immediate endangerment or when the modification(s) only pertain to administrative or logistical components of the project. Investigators must also promptly alert the REB of any changes that increase the risk to participant(s), any changes that considerably affect the conduct of the project, all unanticipated and harmful events that occur, and new information that may negatively affect the conduct of the project or the safety of the participant(s).

Coordonnateur / COORDINATOR

Coordonnateur de l'éthique / Ethics Coordinator

Pour/For **Daniel LAGAREC** Président(e) du/ Chair of the **Comité d'éthique de la recherche en sciences de la santé et**

550, rue Cumberland, pièce 154 550 Cumberland Street, Room 154 Ottawa (Ontario) K1N 6N5 Canada Ottawa,
Ontario K1N 6N5 Canada 613-562-5387 • 613-562-5338 • ethique@uOttawa.ca / ethics@uOttawa.ca
www.recherche.uottawa.ca/deontologie | www.recherche.uottawa.ca/ethics 22/08/2025

Appendix 2. Consent form: Adults.



uOttawa

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

Consent Form

Title of the project: Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm

Thesis Supervisor

Amineh Koravand, Ph.D.

Associate Professor and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Principal Investigator

Shaghayegh Omidvar, M.Sc.

Ph.D. candidate and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Other

William Khawam

Undergraduate student in biomedical sciences

Invitation to participate: You are invited to participate in a research project organized by Dr. Amineh Koravand (thesis supervisor), Shaghayegh Omidvar (principal investigator), and William Khawam. The study is conducted by Shaghayegh Omidvar for her doctoral thesis project under the supervision of Dr. Amineh Koravand.

This form explains the reasons for conducting the study and describes any potential risks or benefits for you. A member of the team will be responsible for discussing this project with you and answering any questions you may have.

Purpose of Study



uOttawa

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

The purpose of this study is to use auditory stimulation to explore brainwaves called the Mismatch Negativity. These brainwaves can be measured in reaction to simple or more complex sounds. The sounds of interest for this project are silent gaps inserted in the noise. We are interested in collecting cortical wave data from adults with the age of 20 to 40 years old and then children between 8 and 12 years old.

Why conduct this study?

This study will be conducted to further our knowledge of the way the central auditory system responds to gapped stimuli. The findings will add to the body of knowledge of the central auditory system of adults as well as explain their capacity to communicate in non-favorable listening conditions. In addition, the results collected by this research will be used by a PhD student in rehabilitation sciences for her thesis project.

How many people will take part in this study?

We will invite a total of 15 adults aged between 20 and 40 years to participate in our study.

What will happen during the appointment?

Firstly, we will evaluate your audition capacities, which will take approximately 10 minutes.

This step consists of:

1) Filling out a questionnaire

We will ask you to fill out a six-page questionnaire about your demographic, hearing, and language-related information, and your musical experience. It will take around 5 minutes to fill out this questionnaire.

2) Audiological screening

This screening includes measures that will allow us to determine the level of your hearing ability. The whole screening test takes around 8 minutes. It includes these steps:

1. Otoscopy. With the help of a special lamp, we will look inside the ears 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.
2. Screening audiometry. We will also verify how well you hear 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.
3. Tympanometry. We will conduct tympanometry, which is a test that assesses your middle ear function by measuring eardrum movements. The test involves placing a small, soft probe in your ear to gently change the air pressure and see how the eardrum reacts.

The sounds and pressure used while doing the screening are safe and will not damage your ears. The analysis of the results will be done by an Audiologist.

If we discover anything abnormal with your hearing during the screening, we will let you know and suggest you consult your physician.

Eligibility criteria are verified on the phone or by email before the first evaluation session as well as during this session. You can only participate in the second part of the appointment if the results for these different tests indicate that you fulfill the different criteria. You will be made aware of these results at the electrophysiological testing. If the results show that you are no longer eligible during the first session or before that time, you might not be able to continue the study. You might also be unable to participate in the rest of the research, depending on the



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

results obtained during this appointment.

3) Experimental Measures

You will sit quietly for approximately 3 hours and will watch a video. A small earphone (like an earbud) is placed in your ears, and you will hear various sounds (gapped noises). A cap will be placed on the head with 32 electrodes to measure the very small shifts in electrical activity or EEG that are normally present on the scalp. Preparing the cap, including head measurements and applying gel to ensure a good connection between the electrodes and your scalp, will take around 30 minutes. We will examine how the brain's responses change when there are different sounds. A computer will analyze the response and display the result. Testing takes place at the Faculty of Health Sciences at the University of Ottawa located at 200 Lees Ave. The testing session is scheduled depending on your availability (during weekends, during the work week, etc.) and it lasts around 4 hours. During the testing, you can take breaks if you feel tired.

For these measures, we will not be able to communicate the results because the analysis will only take place when all the participants have been tested.

How will the name and results stay confidential?

An identification number will be assigned to each participant on the forms collected. All information will be kept in a locked file in the office of the head researcher. None of the personal information, such as the name, will be published or mentioned. All information will stay confidential, and the name will not appear on any document used for the research.

What are the risks?

There are no risks to health or self-esteem associated with your participation in this project. All the procedures are documented and have been commonly used for years in many laboratories. However, your participation could cause a low level of stress or fatigue. Short breaks are planned to prevent tiredness. Additional breaks are always an option.

Storing results and data

The results will be kept securely for 5 years after the publication of the study in a locked folder or a computer protected with a password. This procedure applies to all participants, whether the research was completed or not. This information will be kept in the office of the main researcher. All data will be destroyed after 5 years.

Advantages

While taking part in the study, you can benefit from a free hearing screening test, as well as contribute to the development of general knowledge in the field of audiology. When the study is complete, you will receive a summary of the results of the group tested by email. You do not have to pay for anything while participating.

Participating voluntarily

Your participation is fully voluntary. We will respect your choice if you refuse to participate even though your consent was already given. If you choose to withdraw from the study, any data collected from you up to that point will be securely deleted and permanently destroyed. Your signature does not mean that you have given up any of your legal rights. If we discover any abnormality, you will be told, and we will suggest you consult your physician.

Declaring responsibility



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

The signature of this form tells us that you understand the information concerning this research project.

Conflicts of interest

There are no conflicts of interest between the main researcher and the assistants, as nobody is part of a company or other organization. This research project is not financed.

DECLARATION

Please read the instructions carefully and check the box below to show that you have understood. If you have any questions regarding this project, please talk to a team member. You can contact Dr. Aminch Koravand (thesis supervisor, University of Ottawa, (514) 243-9518).

For any additional information about the ethical aspects of this research, please contact the Protocol Officer for Research Ethics, University of Ottawa, Tabaret Hall, 550 Cumberland Street, room 154, (613) 562-5387, or ethics@uottawa.ca.

Confidentiality:

- ☐ I understand that all information will be kept confidential.
- ☐ I understand that the information and data collected will be kept confidential. I understand that neither my name nor any other information will be included in the publication of this research. My information will always be kept locked.
- ☐ I understand that the results of this study will be used for a PhD thesis project in Audiology at the University of Ottawa.

Dropping out of the study:

- ☐ I understand that I can choose not to participate in this study and that I will not be affected by this decision. I understand that my medical services provided by health professionals, such as my family doctor, will not be altered if I decide not to participate. These services could be appointments as well as referrals to other health specialists.
- ☐ I understand that I can drop out of this study at any moment by telling a member of the research team that I am no longer interested and asking to have my data removed from the study. This will not affect the medical service I receive.

Consent

I, _____, hereby consent to take part in this study. The purpose of this study has been explained to me, and I have been allowed to ask questions about the purpose and requirements of my participation. My signature indicates that I agree to participate in the study.

There are two copies of the consent form, one of which you may keep.

Name: _____

Signature: _____ Date: _____

Consent from the person in charge, the researcher, or their representative.

The research project, as well as the modalities of participation, has been described to the participant. A member of the research group has answered any questions that have been asked and has explained that participation is completely voluntary. The team will respect what has been agreed on in the 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

Name of the researcher (or representative):

Signature of the researcher (or representative):

Date: _____

Appendix 2. Consent form: Typically Developing Children.

Consent Form



Title of the project: Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm

Thesis Supervisor

Amineh Koravand, Ph.D.

Associate Professor and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Principal Investigator

Shaghayegh Omidvar, M.Sc.

Ph.D. candidate and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Other

William Khawam

Undergraduate student in biomedical sciences

Invitation to participate: My child is invited to participate in a research project organized by Dr. Amineh Koravand (thesis supervisor), Shaghayegh Omidvar (principal investigator), and William Khawam. The study is conducted by Shaghayegh Omidvar for her doctoral thesis project under the supervision of Dr. Amineh Koravand.

This form explains the reasons for conducting the study and describes any potential risks or benefits for you. A member of the team will be responsible for discussing this project with you and answering any questions you may have.

Purpose of Study



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

The purpose of this study is to use auditory stimulation to explore brainwaves called the Mismatch Negativity in children. These brainwaves can be measured in reaction to simple or more complex sounds. The sounds of interest for this project are silent gaps inserted in the noise. We are interested in collecting cortical wave data from children between 8 and 12 years old.

Why conduct this study?

This study will be conducted to further our knowledge of the way the central auditory system in children responds to gapped stimuli. The findings will add to the body of knowledge of the central auditory system of children, as well as explain their capacity to communicate in non-favorable listening conditions. In addition, the results collected by this research will be used by a PhD student in rehabilitation sciences for her thesis project.

How many people will take part in this study?

We will invite a total of 15 children aged between 8-12 years to participate in our study.

What will happen during the appointment?

Firstly, we will evaluate your audition capacities, which will take approximately 10 minutes.

This step consists of:

1) Filling out a questionnaire

We will ask your parents to fill out a five-page questionnaire about your demographic, hearing, and language-related information, and your musical experience. It will take around 5 minutes to fill out this questionnaire. Your parent will also be asked to fill out the “Auditory Processing Domains Questionnaire (APDQ)”, which includes 50 items to identify if there are any auditory processing, attention, and language problems. It will take around 10-15 minutes to complete this questionnaire.

2) Audiological screening

This screening includes measures that will allow us to determine the level of your hearing ability. The whole screening test takes around 8 minutes. It includes these steps:

3. Otoscopy. With the help of a special lamp, we will look inside your ears 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.

4. Screening audiometry. We will also verify how well you hear 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.

3. Tympanometry. We will conduct tympanometry, which is a test that assesses your middle ear function by measuring eardrum movements. The test involves placing a small, soft probe in your ear to gently change the air pressure and see how the eardrum reacts.

4) The Auditory Continuous Performance Test (ACPT). You will take part in an attention test to ensure that there are no attentional issues. This assessment takes around 10 minutes. You will be asked to push the button when you hear the word “dog”.

The sounds and pressure used while doing the screening are safe and will not damage your ears. The analysis of the results will be done by an Audiologist.

If we discover anything abnormal with your hearing during the screening, we will let you know and suggest you consult your physician.



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

Eligibility criteria are verified on the phone or by email before the first evaluation session, as well as during this session. You can only participate in the second part of the appointment if the results for these different tests indicate that you fulfill the different criteria. You will be made aware of these results at the electrophysiological testing. If the results show that you are no longer eligible during the first session or before that time, you might not be able to continue the study. You might also be unable to participate in the rest of the research, depending on the results obtained during this appointment.

3) Experimental Measures

You will sit quietly for approximately 2-3 hours and will watch a video. A small earphone (like an earbud) is placed in your ears, and you will hear various sounds (gapped noises). A cap will be placed on the head with 32 electrodes to measure the very small shifts in electrical activity or EEG that are normally present on the scalp. Preparing the cap, including head measurements and applying gel to ensure a good connection between the electrodes and your scalp, will take around 30 minutes. We will examine how the brain's responses change when there are different sounds. A computer will analyze the response and display the result.

Testing takes place at the Faculty of Health Sciences at the University of Ottawa, located at 200 Lees Ave. The testing session is scheduled depending on your availability (during weekends, during the work week, etc.), and it lasts around 3.5-4 hours. During the testing, you can take breaks if you feel tired.

For these measures, we will not be able to communicate the results because the analysis will only take place when all the participants have been tested.

How will your name and results stay confidential?

An identification number will be assigned to each participant on the forms collected. All information will be kept in a locked file in the office of the head researcher. None of the personal information, such as the name, will be published or mentioned. All information will stay confidential, and the name will not be shown on any document used for the research.

What are the risks?

There are no risks to health or self-esteem associated with your participation in this project. All the procedures are documented and have been commonly used for years in many laboratories. However, your participation could cause a low level of stress or fatigue. Short breaks are planned to prevent tiredness. Additional breaks are always an option.

Storing results and data

The results will be kept secure for 5 years after the publication of the study in a locked folder or a computer protected with a password. This procedure applies to all participants, whether the research was completed or not. This information will be kept in the office of the main researcher. All data will be destroyed after 5 years.

Advantages

You will benefit from a free hearing screening test, as well as contribute to the development of general knowledge in the field of audiology. When the study is complete, your parent will receive a resume of the results of the group tested by email. You do not have to pay for anything while participating.

Compensation



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

While participating in the study, you will receive a 20-dollar Amazon gift card for your time. You receive the compensation even if you decide to withdraw from the study.

Participating voluntarily

Your participation is fully voluntary. We will respect your choice if you refuse to participate even though your consent was already given. If you choose to withdraw from the study, any data collected from you up to that point will be securely deleted and permanently destroyed. Your signature does not mean that you have given up any of your legal rights. If we discover any abnormality, you will be told, and we will suggest you consult your physician.

Declaring responsibility

The signature of this form tells us that you understand the information concerning this research project.

Conflicts of interest

There are no conflicts of interest between the main researcher and the assistants, as nobody is part of a company or other organization. This research project is not financed.

DECLARATION

Please read the instructions carefully and check the box below to show that you have understood. If you have any questions regarding this project, please talk to a team member. You can contact Dr. Amineh Koravand (thesis supervisor, University of Ottawa, (514) 243-9518).

For any additional information about the ethical aspects of this research, please contact the Protocol Officer for Research Ethics, University of Ottawa, Tabaret Hall, 550 Cumberland Street, room 154, (613) 562-5387 or ethics@uottawa.ca.

Confidentiality:

☐ I understand that the information and data collected will be kept confidential. I understand that neither my name nor any other information will be included in the publication of this research. My information will always be kept locked.

☐ I understand that the results of this study will be used for a PhD thesis project at the University of Ottawa.

Dropping out of the study:

☐ I understand that I can choose not to participate in this study and will not be affected by this decision. I understand that our medical services, provided by health professionals, such as my family doctor, will not be altered if I decide not to participate. These services could be appointments as well as references to other health specialists.

☐ I understand that I can drop out of this study at any moment by telling a member of the research team that I am no longer interested and asking to have my data removed from the study. This will not affect the medical service I receive.

Consent

I, _____, hereby consent to take part in this study. The purpose of this study has been explained to me, and I have been allowed to ask questions about the purpose and requirements of my participation. My signature indicates that I agree to participate in the study.

There are two copies of the consent form, one of which you may keep.



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

Name: _____

Signature: _____ Date: _____

Consent from the person in charge, the researcher, or their representative.

The research project, as well as the modalities of participation, have been described to the participant. A member of the research group has answered any questions that have been asked and has explained that participation is completely voluntary. The team will respect what has been agreed on in the consent form.

Name of the researcher (or representative):

Signature of the researcher (or representative):

Date: _____

Appendix 3. Consent form: Children with Auditory Processing Disorders.



Université d'Ottawa

Faculté des sciences
de la santé

Ecole des sciences de la
réadaptation

University of Ottawa

Faculty of Health
Sciences

School of Rehabilitation
Sciences

Consent Form

Title of the project: Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm

Thesis Supervisor

Amineh Koravand, Ph.D.

Associate Professor and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Principal Investigator

Shaghayegh Omidvar, M.Sc.

Ph.D. candidate and Audiologist

Speech Pathology and Audiology Program

School of Rehabilitation Sciences

Faculty of Health Science

Other

William Khawam

Undergraduate student in biomedical sciences

Invitation to participate: You are invited to participate in a research project organized by Dr. Amineh Koravand (thesis supervisor), Shaghayegh Omidvar (principal investigator), and William Khawam. The study is conducted by Shaghayegh Omidvar for her doctoral thesis project under the supervision of Dr. Amineh Koravand.

This form explains the reasons for conducting the study and describes any potential risks or benefits for you. A member of the team will be responsible for discussing this project with you and answering any questions you may have.



Université d'Ottawa
Faculté des sciences
de la santé

Ecole des sciences de la
réadaptation

University of Ottawa
Faculty of Health
Sciences

School of Rehabilitation
Sciences

Purpose of Study

The purpose of this study is to use auditory stimulation to explore brainwaves called the Mismatch Negativity in children. These brainwaves can be measured in reaction to simple or more complex sounds. The sounds of interest for this project are silent gaps inserted in the noise. We are interested in collecting cortical wave data from children between 8 and 12 years old.

Why conduct this study?

This study will be conducted to further our knowledge of the way the central auditory system in children responds to gapped stimuli. The findings will add to the body of knowledge of the central auditory system of children as well as explain their capacity to communicate in non-favorable listening conditions. In addition, the results collected by this research will be used by a PhD student in rehabilitation sciences for her research project.

How many people will take part in this study?

We will invite a total of 15 children aged between 8-12 years to participate in our study.

What will happen during the appointment?

Firstly, we will evaluate your audition capacities, which will take approximately 10 minutes.

This step consists of:

1) Filling out a questionnaire

We will ask your parents to fill out a five-page questionnaire about your demographic, hearing, and language-related information, and your musical experience. It will take around 5 minutes to fill out this questionnaire.

2) Audiological screening

This screening includes measures that will allow us to determine the level of your hearing ability. The whole screening test takes around 8 minutes. It includes these steps:

5. Otoscopy. With the help of a special lamp, we will look inside your ears 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.

6. Screening audiometry. We will also verify how well you hear 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.

3. Tympanometry. We will conduct tympanometry which is a test that assesses your middle ear function by measuring eardrum movements. The test involves placing a small, soft probe in your ear to gently change the air pressure and see how the eardrum reacts.

The sounds and pressure used while doing the screening are safe and will not damage your ears. The analysis of the results will be done by an Audiologist.

If we discover anything abnormal with your hearing during the screening, we will let you know and suggest you consult your physician.

Eligibility criteria are verified on the phone or by email before the first evaluation session as well as during this session. You can only participate in the second part of the appointment if the results for these different tests indicate that you fulfill the different criteria. You will be made aware of these results at the electrophysiological testing. If the results show that you are no longer eligible during the first session or before that time, you might not be able to continue the study. You might also be unable to participate in the rest of the research, depending on the results obtained during this appointment.



Université d'Ottawa

Faculté des sciences
de la santé

Ecole des sciences de la
réadaptation

University of Ottawa

Faculty of Health
Sciences

School of Rehabilitation
Sciences

3) Experimental Measures

You will sit quietly for approximately 2-3 hours and will watch a video. A small earphone (like an earbud) is placed in your ears, and you will hear various sounds (gapped noises). A cap will be placed on the head with 32 electrodes to measure the very small shifts in electrical activity or EEG that are normally present on the scalp. Preparing the cap, including head measurements and applying gel to ensure a good connection between the electrodes and your scalp, will take around 30 minutes. We will examine how the brain's responses change when there are different sounds. A computer will analyze the response and display the result.

Testing takes place at the Faculty of Health Sciences at the University of Ottawa located at 200 Lees Ave. The testing session is scheduled depending on your availability (during weekends, during the work week, etc.), and it lasts around 3.5-4 hours. During the testing, you can take breaks if you feel tired.

For these measures, we will not be able to communicate the results because the analysis will only take place when all the participants have been tested.

How will your name and results stay confidential?

An identification number will be assigned to each participant on the forms collected. All information will be kept in a locked file in the office of the head researcher. None of the personal information, such as the name, will be published or mentioned. All information will stay confidential, and the name will not be shown on any document used for the research.

What are the risks?

There are no risks to health or self-esteem associated with your participation in this project. All the procedures are documented and have been commonly used for years in many laboratories. However, your participation could cause a low level of stress or fatigue. Short breaks are planned to prevent tiredness. Additional breaks are always an option.

Storing results and data

The results will be kept secure for 5 years after the publication of the study in a locked folder or a computer protected with a password. This procedure applies to all participants, whether the research was completed or not. This information will be kept in the office of the main researcher. All data will be destroyed after 5 years.

Advantages

You will benefit from a free hearing screening test, as well as, contribute to the development of general knowledge in the field of audiology. When the study is complete, your parent will receive a resume of the results of the group tested by email. You do not have to pay for anything while participating.

Compensation

While participating in the study, you will receive a 20-dollar Amazon gift card for your time. You receive the compensation even if you decide to withdraw from the study.

Participating voluntarily

Your participation is fully voluntary. We will respect your choice if you refuse to participate even though your consent was already given. If you choose to withdraw from the study, any



Université d'Ottawa
Faculté des sciences
de la santé

Ecole des sciences de la
réadaptation

University of Ottawa
Faculty of Health
Sciences

School of Rehabilitation
Sciences

data collected from you up to that point will be securely deleted and permanently destroyed. Your signature does not mean that you have given up any of your legal rights. If we discover any abnormality, you will be told, and we will suggest you consult your physician.

Declaring responsibility

The signature of this form tells us that you understand the information concerning this research project.

Conflicts of interest

There are no conflicts of interest between the main researcher and the assistants as nobody is part of a company or other organization. This research project is not financed.

DECLARATION

Please read the instructions carefully and check the box below to show that you have understood. If you have any questions regarding this project, please talk to a team member. You can contact Dr. Amineh Koravand (thesis supervisor, University of Ottawa, (514) 243-9518).

For any additional information about the ethical aspects of this research, please contact the Protocol Officer for Research Ethics, University of Ottawa, Tabaret Hall, 550 Cumberland Street, room 154, (613) 562-5387 or ethics@uottawa.ca.

Confidentiality:

- ☐ I understand that the information and data collected will be kept confidential. I understand that neither my name nor any other information will be included in the publication of this research. My information will always be kept locked.
- ☐ I understand that the results of this study will be used for a PhD's thesis project at the University of Ottawa.

Dropping out of the study:

- ☐ I understand that I can choose not to participate in this study and will not be affected by this decision. I understand that our medical services provided by health professionals such as my family doctor will not be altered if I decide to not to participate. These services could be appointments as well as references to other health specialists.
- ☐ I understand that I can drop out of this study at any moment by telling a member of the research team that I am no longer interested and asking to have my data removed from the study. This will not affect the medical service I receive.

- **Consent**

I, _____, hereby consent to take part in this study. The purpose of this study has been explained to me, and I have been allowed to ask questions about the purpose and requirements of my participation. My signature indicates that I agree to participate in the study.

There are two copies of the consent form, one of which you may keep.

Name: _____

Signature: _____

Date: _____



Université d'Ottawa

Faculté des sciences
de la santé

Ecole des sciences de la
réadaptation

University of Ottawa

Faculty of Health
Sciences

School of Rehabilitation
Sciences

Consent from the person in charge, the researcher, or their representative.

The research project, as well as the modalities of participation, have been described to the participant. A member of the research group has answered any questions that have been asked and has explained that participation is completely voluntary. The team will respect what has been agreed on in the consent form.

Name of the researcher (or representative):

Signature of the researcher (or representative):

Date: _____

Appendix 4. Recruitment Poster for Adults.



RECRUITMENT FOR A STUDY

Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm

We are looking for participants for a research study.

You must:

- ▶ **Have good hearing** (auditory screening done during the evaluation session)
- ▶ Be between 20 and 40 years of age
- ▶ Be right-handed
- ▶ Have no known medical conditions related to your hearing
- ▶ Have no known attention or learning difficulties
- ▶ Have no experience of formal musical training

The testing session will take place at the Faculty of Health Sciences (200 Lees Avenue) of the University of Ottawa. During this session, a hearing-screening test will be performed as well as an electrophysiological test. The evaluation session will take approximately **4 hours**. Appointments can be made both within and outside of work hours.

Participants will be kept following the principle of first-come, first-served.

If interested in participating, please leave your contact information at the following number or email address, and one of the researchers involved in the study will contact you as soon as possible.

Name of researchers: Shaghayegh Omidvar, Ph.D. candidate (Principal investigator), and Amineh Koravand, Ph.D. (Thesis supervisor), University of Ottawa, Faculty of Health Sciences, School of Rehabilitation Sciences, 200 Lees Avenue, Ottawa, Ontario, K1S 5S9. Phone number: *****. E-mail address: *****

Appendix 5. Recruitment Poster for Typically Developing Children.



RECRUITMENT FOR A STUDY

Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm

We are looking for children to participate in a research study.

The child must:

- ▶ **Have good hearing** (auditory screening done during the evaluation session)
- ▶ Be between 8 and 12 years of age
- ▶ Be right-handed
- ▶ Have no known medical conditions related to your hearing
- ▶ Have no known attention or learning difficulties
- ▶ Have no experience of formal musical training

The testing session will take place at the Faculty of Health Sciences (200 Lees Avenue) of the University of Ottawa. During this session, a hearing-screening test will be performed as well as an electrophysiological test. The evaluation session will take approximately **2.5-3 hours**. Appointments can be made both within and outside of work hours.

Participants will be kept following the principle of first come first served.

If interested in participating, please leave your contact information at the following number or email address and one of the researchers involved in the study will contact you as soon as possible.

Name of researchers: Shaghayegh Omidvar Ph.D. candidate (Principal investigator), and Amineh Koravand Ph.D. (Thesis supervisor) University of Ottawa, Faculty of Health Sciences, School of Rehabilitation Sciences, 200 Lees Avenue, Ottawa, Ontario, K1S 5S9. Phone number: *****. E-mail address: *****

Appendix 6. Compensation form.

Compensation for participation

I hereby confirm that I have received the amount of a \$ 20 Amazon gift card as compensation for the participation of my child _____ in the research project entitled *Investigating Temporal Resolution in Children with Listening Difficulties Using a Novel Multi-Deviant Paradigm*.

Name of participant/parent: _____

Signature: _____

Date: _____

Name of researcher: _____

(or assistant)

Signature of researcher: _____

(or assistant)

Date: _____

Pour usage administrative seulement

Numéro du compte:

Approuvé par:

Appendix 7. Demographic and Music Experience Questionnaire



**Demographic and Music Experience
Questionnaire**

Hearing Sensitivity Among Young Adult Non-Musicians

**Piano Pedagogy Research Laboratory
Audiology and Speech-Language Pathology Program
University of Ottawa**

© 2019 Piano Pedagogy Research Laboratory

Demographic & Music Experience - CON Questionnaire

- General Information

Participant ID <i>(to be filled in by the Clinic)</i>	
Date	

- NOTE

Rest assured that this information will remain strictly confidential. Only the members of the research team will have access to this information. Only group data (e.g. group averages) will be made public when the results of this study are presented in scientific conferences or similar contexts.

PLEASE CIRCLE OR ENTER YOUR RESPONSES TO THE QUESTIONS BELOW

• SECTION 1: DEMOGRAPHIC AND HEARING-RELATED INFORMATION

QUESTIONS		YOUR RESPONSE
1	Gender	
2	Age	
3	If you are a university student, what is your current program?	
4	Do you have any concerns about your hearing?	<ul style="list-style-type: none"> • Yes: Specify: _____ • No
5	Do you ever have difficulty understanding speech?	<ul style="list-style-type: none"> • Yes • No
6	Do you have a history of ear infections?	<ul style="list-style-type: none"> • Yes • No
7	Do you have tinnitus (ringing, roaring, hissing, buzzing in your ears?)	<ul style="list-style-type: none"> • Yes • No
8	Do you have otalgia (ear pain)?	<ul style="list-style-type: none"> • Yes • No
9	Do you have vertigo/disequilibrium?	<ul style="list-style-type: none"> • Yes • No
10	Are you regularly exposed to loud sound? (Working in a bar, construction, etc.)	<ul style="list-style-type: none"> • Yes: Specify: _____ • No
11	Do you have a history of any noise exposure?	<ul style="list-style-type: none"> • Yes: Specify: _____ • No
12	If you answered "yes" to the previous question, did you wear hearing protection when exposed?	<ul style="list-style-type: none"> • Yes • No
13	Have you ever had your hearing tested?	<ul style="list-style-type: none"> • Yes • No
14	If you answered "yes" to the previous question, what were the results?	
15	Have you ever seen a doctor who specializes in ears?	<ul style="list-style-type: none"> • Yes • No
16	Are you currently taking any medications?	<ul style="list-style-type: none"> • Yes • No
17	If you answered "yes" to the previous question, please list:	

SECTION 2: LANGUAGE INFORMATION

	Language	Age that you first learned it	Place you learned it (at home, at school, in an office, from neighbours, etc)	Your current degree of mastery of the language					
					Highly fluent	Fluent	Some mastery	A little mastery	Not at all
First language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Second language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Third language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Fourth language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐

SECTION 3: MUSIC EXPERIENCE

1	Have you ever played an instrument? (If not, go to question #15 of this section).	<ul style="list-style-type: none"> Yes No
2	If you answered yes, what instrument do you play?	
3	For how many years have you played this instrument?	Years: _____ Age of start: _____ Age of end: _____
4	If you are still playing, how many hours do you practice per week?	
5	If you play more than one instrument, what is the secondary instrument you play?	
6	For how many years have you played this secondary instrument?	Years: _____ Age of start: _____ Age of end: _____
7	If you are still playing this instrument, how many hours do you practice per week?	

8	How many years of formal training on a musical instrument (including voice) have you had during your lifetime? (circle one option)	<ul style="list-style-type: none"> • 0 • 0.5 • 1 • 2 • 3-5 • 6-9 • 10 or more
9	For how many years have you engaged in regular, daily practice of a musical instrument (including voice)? (circle one option)	<ul style="list-style-type: none"> • 0 • 1 • 2 • 3 • 4-5 • 6-9 • 10 or more
10	At the peak of your interest, how many hours a day did you practice on your primary instrument? (circle one option)	<ul style="list-style-type: none"> • 0 • 0.5 • 1 • 1.5 • 2 • 3-4 • 5 or more
11	*For how many years have you played or sung in a group, band, choir or orchestra? (circle one option)	<ul style="list-style-type: none"> • 0 • 1 • 2 • 3 • 4-5 • 6-9 • 10 or more
12	If you are still playing an instrument, where do you usually practice?	<ul style="list-style-type: none"> • Practice room with sound absorption/dampening • Practice room without sound absorption/dampening • At home • Other: _____
13	Do you use any auditory protection when you practice?	<ul style="list-style-type: none"> • Yes • No
14	If so, what kind?	

15	How many live music events have you attended as an audience member in the past twelve months? (circle one option)	<ul style="list-style-type: none"> • 0 • 1 • 2 • 3 • 4-6 • 7-10 • 11 or more
16	How much time do you spend per day attentively listening to music? (circle one option)	<ul style="list-style-type: none"> • 0-15 min • 15-30 min • 30-60 min • 60-90 min • 2 hrs • 2-3 hrs • 4 hrs or more

SECTION 4: PERSONAL MUSIC SYSTEMS

1	Do you regularly listen to a personal music system (PMS) with headphones? (iPod, smartphone, MP3 player, computer etc).	<ul style="list-style-type: none"> • Yes • No
2	How many minutes or hours per day do you listen to your PMS per day?	
3	At what volume level do you usually listen to your PMS?	<ul style="list-style-type: none"> • Soft • Medium • Loud • Very loud
4	What kind of headphones do you use when listening to music on your PMS? (You can circle more than one)	<ul style="list-style-type: none"> • Earbuds (fit in outer ear) • In-ear headphones (extend into the ear canal) • Circumaural (full-size headphones that encompass the entire ear) • Supra-aural headphones (pads that press against the ear)

THANK YOU FOR TAKING THE TIME TO FILL OUT THIS FORM

Appendix 8. Demographic and Music Experience Questionnaire: Child's version.

Demographic and Music Experience Questionnaire

Hearing Sensitivity Among Child Non-Musicians

- General Information

Participant ID <i>(to be filled in by the Clinic)</i>	
Date	

- NOTE

Rest assured that this information will remain strictly confidential. Only the members of the research team will have access to this information. Only group data (e.g. group averages) will be made public when the results of this study are presented in scientific conferences or similar contexts.

PLEASE CIRCLE OR ENTER YOUR RESPONSES TO THE QUESTIONS BELOW

• SECTION 1: DEMOGRAPHIC AND HEARING-RELATED INFORMATION

QUESTIONS		YOUR RESPONSE
1	Gender	
2	Age	
3	Do you have any concerns about your child's hearing?	<ul style="list-style-type: none"> • Yes: Specify: _____ • No
4	Does your child ever have difficulty understanding speech?	<ul style="list-style-type: none"> • Yes • No
5	Does your child have a history of ear infections?	<ul style="list-style-type: none"> • Yes • No
6	Does your child have tinnitus (ringing, roaring, hissing, buzzing in your ears?)	<ul style="list-style-type: none"> • Yes • No
7	Does your child have otalgia (ear pain)?	<ul style="list-style-type: none"> • Yes • No
8	Does your child have vertigo/disequilibrium?	<ul style="list-style-type: none"> • Yes • No
9	Has your child ever had their hearing tested?	<ul style="list-style-type: none"> • Yes • No
10	If you answered "yes" to the previous question, what were the results?	<ul style="list-style-type: none"> • Yes • No
11	Has your child ever seen a doctor who specializes in ears?	<ul style="list-style-type: none"> • Yes • No
12	Is your child currently taking any medications?	<ul style="list-style-type: none"> • Yes • No
13	If you answered "yes" to the previous question, please list:	

• SECTION 2: LANGUAGE INFORMATION

	Language	Age your child first learned it	Place you learned it (at home, at school, in a language class, from neighbors, etc)	Your child's current degree of mastery of the language					
					Highly fluent	Fluent	Some mastery	A little mastery	Not at all
First language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Second language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Third language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐
Fourth language				Understand	☐	☐	☐	☐	☐
				Speak	☐	☐	☐	☐	☐
				Read	☐	☐	☐	☐	☐
				Write	☐	☐	☐	☐	☐

• SECTION 3: MUSIC EXPERIENCE

1	Has your child ever played an instrument? (If not, go to question #15 of this section).	<ul style="list-style-type: none"> • Yes • No
2	If you answered yes, what instrument does your child play?	
3	For how many years has your child played this instrument?	Years: _____ Age of start: _____ Age of end: _____
4	If your child is still playing, how many hours does your child practice per week?	
5	If your child plays more than one instrument, what is the secondary instrument your child plays?	
6	For how many years has your child played this secondary instrument?	Years: _____ Age of start: _____ Age of end: _____
7	If your child is still playing this instrument, how many hours does your child practice per week?	
8	How many years of formal training on a musical instrument (including voice) has your child had during their lifetime? (circle one option)	<ul style="list-style-type: none"> • 0 • 0.5 • 1 • 2 • 3-5 • 6-9 • 10 or more
9	For how many years has your child engaged in regular, daily practice of a musical instrument (including voice)? (circle one option)	<ul style="list-style-type: none"> • 0 • 1 • 2 • 3 • 4-5 • 6-9 • 10 or more
10	At the peak of your child's interest, how many hours a day did your child practice on their primary instrument? (circle one option)	<ul style="list-style-type: none"> • 0 • 0.5 • 1 • 1.5 • 2 • 3-4 • 5 or more

11	How much time does your child spend per day attentively listening to music? (circle one option)	<ul style="list-style-type: none"> • 0-15 min • 15-30 min • 30-60 min • 60-90 min • 2 hrs • 2-3 hrs • 4 hrs or more
----	---	--

• SECTION 4: PERSONAL MUSIC SYSTEMS

1	Does your child regularly listen to a personal music system (PMS) with headphones? (iPod, smartphone, MP3 player, computer etc).	<ul style="list-style-type: none"> • Yes • No
2	How many minutes or hours per day does your child listen to your PMS per day?	
3	At what volume level does your child usually listen to your PMS?	<ul style="list-style-type: none"> • Soft • Medium • Loud • Very loud
4	What kind of headphones does your child use when listening to music on your PMS? (You can circle more than one)	<ul style="list-style-type: none"> • Earbuds (fit in outer ear) • In-ear headphones (extend into the ear canal) • Circumaural (full-size headphones that encompass the entire ear) • Supra-aural headphones (pads that press against the ear)

THANK YOU FOR TAKING THE TIME TO FILL OUT THIS FORM

Appendix 9. Auditory Processing Questionnaire (APDQ)

The Auditory Processing Questionnaire



FOR PARENTS AND TEACHERS OF STUDENTS 7 TO 18 YEARS OLD- Version

The APDQ reviews a child's everyday listening experience. Language, attention, and hearing are all important.

Please rate your child or student's performance on each item below based on your recent observations. Keep in mind what is "expected" for his or her age.

"Noisy conditions" refers to background noise from such sources as TV, machinery, music, people talking etc.

"Listening accurately" means hearing words or statements right the first time, without saying "what?" or needing repetition (repeats).

Check (✓)	Column 1: if skill has been observed	Most times	over $\frac{3}{4}$ the time
	Column 2: if skill has been observed	Often	over $\frac{1}{2}$ the time
	Column 3: if skill has been observed	Sometimes	under $\frac{1}{2}$ the time
	Column 4: if skill has been observed	Rarely	under $\frac{1}{4}$ the time

"Most times" gets the highest (favorable) score for most items. However, the format is reversed for items 16, 17, 21, 22, 30 and 35 where "Rarely" gets the highest and "Most times" the lowest score. Items with reversed scoring are starred (*).

Please rate all items. Write "N/A" in the first column if you are unable to do so.

YOUR CHILD or STUDENT:	MOST TIMES > 3/4	OFTEN >1/2	SOMETIMES <1/2	RARELY <1/4
1. listens attentively when spoken to alone.				
2. listens attentively when listening to speakers in a <u>quiet</u> group (meetings and classrooms).				
3. listens attentively when listening to speakers in a <u>noisy</u> group (meetings and classrooms).				
4. hears your words accurately , without repeats, when paying attention in a <u>quiet</u> room.				
5. hears your words accurately , without repeats, when paying attention in a <u>noisy</u> room,				

YOUR CHILD or STUDENT:	MO ST TIM ES > 3/4	OFTEN >1/2	SO ME TIME S <1/2	RARELY <1/4
6. takes the time to listen more carefully to important information.				
7. understands your directions & requests when paying attention in a <u>quiet</u> place.				
8. understands your directions & requests when paying attention in a <u>noisy</u> place.				
9. understands what is said when there is "echo" noise (in gyms, cafeterias or where the sound is coming from "echoey" loudspeakers).				
10. understands your conversation while others are conversing nearby (parties & meals).				
11. can listen accurately to you while doing something else (games or chores).				
12. can listen accurately without visual aids (not seeing a speaker's face or gestures- not having diagrams or pictures).				
13. focuses well – avoids distractions when doing non-listening tasks (studies and chores).				
14. focuses well – avoids distractions , when listening to stories and presentations.				
15. understands written instructions (as expected for his or her age)				
* 16. tires easily when studying (yawns & fidgets).				
* 17. tires easily when listening (yawns & fidgets)				
18. explains things fairly well during conversations.				
19. concentrates on important tasks even when they are not fun or interesting.				
20. hears words right when a speaker's back is turned (or is spoken to from behind).				

YOUR CHILD or STUDENT:	MO ST TIM ES > 3/4	OFTEN >1/2	SO ME TIME S <1/2	RARELY <1/4
* 21. says “what?” or needs repeats , when conversing with interest in a <u>quiet</u> place.				
* 22. says “what?” or needs repeats , when conversing with interest in a <u>noisy</u> place.				
23. pays attention to details and avoids careless errors when doing tasks.				
24. understands and uses longer sentences (as expected for his or her age.)				
25. understands and answers your questions promptly in <u>quiet</u> places (when attentive.)				
26. understands and answers your questions promptly in <u>noisy</u> places (when attentive).				
27. follows spoken directions with steps or sequences (as expected for age).				
28. organizes tasks and activities to do them on time.				
29. understands and uses common slang words (“dudes” “cool” “copycat”).				
* 30. loses or forgets to do things (gets absent minded).				
31. understands speakers who say words less clearly (rapid or mumbled speech, foreign accents).				
32. understands soft spoken-high voiced speakers. (shy speakers-children's voices).				
33. listens accurately on the telephone (without needing information repeated).				
34. can listen accurately to speakers from 6-7 feet away (whether sitting or standing).				
* 35. mishears and confuses similar sounding words (“fifty” not fifteen”, “thirsty” not “Thursday”).				

YOUR CHILD or STUDENT:	MOST TIMES > 3/4	OFTEN >1/2	SOME TIMES <1/2	RARELY <1/4
36. remembers and uses new words correctly (as expected for age).				
37. pronounces new words correctly after hearing them a couple of times (including names of people and places).				
38. can sound out words (letter sounds) to help read and spell them correctly (phonics).				
39. reads happily and at an ok rate (as expected for age).				
40. controls impulses and activity levels to avoid annoying or unsafe actions.				
41. remembers details of spoken directions or requests (without repeats a short time later).				
42. learns well by listening (without a strong need for more visual or hands-on instruction).				
43. can follow pitch and beat patterns – responds to music.				
44. varies speaking voice for emphasis, clarity, and pleasantness.				
45. understands “how” things were said when interpreting comments & following directions (tones of voice and word emphasis).				
46. understands what people say without needing more simple words.				
47. hears ok without needing louder sounds (including voices and warning signals).				
48. talks easily and smoothly for his or her age (without pausing too much or forgetting words).				
49. understands conversations & instructions without major noise controls (turning off the TV, closing windows, moving closer).				
50. understands speakers without needing them to slow down and talk more clearly.				