Correlation between auditory brainstem responses, hyperacusis, and severity of autism spectrum disorder in young children with normal hearing at a tertiary referral center in Indonesia

Margaretta Simamora, MD[1], Semiramis Zizlavsky, MD[1], Tri Juda Airlangga Hardjoprawito, MD[1], Tjhin Wiguna, PhD[2], Bernie Endyarni Medise, MD[3], Retno Wibawanti, MD[4]

[1]Department of Otorhinolaryngology Head and Neck Surgery (ORL-HNS), Dr. Cipto Mangunkusumo National General Hospital, Faculty of Medicine Universitas Indonesia, Jakarta, Indonesia, [2]Department of Psychiatry, Dr. Cipto Mangunkusumo National General Hospital, Faculty of Medicine Universitas Indonesia, Jakarta, Indonesia, [3]Department of Pediatric, Dr. Cipto Mangunkusumo National General Hospital, Faculty of Medicine Universitas Indonesia, Jakarta, Indonesia, [4]Department of Community Medicine, Dr. Cipto Mangunkusumo National General Hospital, Faculty of Medicine Universitas Indonesia, Jakarta, Indonesia

ABSTRACT

Introduction: Autism spectrum disorder (ASD) is a complex condition impacting social communication, behavior, and interests. ASD affects 1 in 100 children globally, with a higher prevalence in boys. Auditory disorders, including hyperacusis, are common in ASD, yet the correlation between Auditory Brainstem Response (ABR) wave latencies and ASD severity, especially with hyperacusis, is under-researched. This study investigates ABR wave latencies in ASD children, exploring their relationship with ASD severity and h as a potential screening tool for ASD. Early diagnose and therapy could enhance the quality of life in ASD patients.

Materials and Methods: A cross-sectional study was conducted by recruiting normal-hearing children aged 3-8 years old with ASD presenting to a national referral ENT clinic between October and December 2023. The severity of ASD was assessed using the Childhood Autism Rating Scale (CARS), while hyperacusis was diagnosed using Modified Check List for Autism in Toddlers, Revised (M-CHAT-R).

Results: A total of 26 children with ASD, 23 of whom were male (88%), aged 3-8 years, were included in the analyses. Among these children, 18 (69.2%) had hyperacusis. Analysis of ABR click revealed a prolonged interpeak latency wave I and III (88.5%), followed by a prolonged latency in wave III (42.3%) and V (21.2%). Neither ABR wave latencies nor hyperacusis were correlated with the severity of ASD, although there was a marginally significant association between wave III latency and CARS score in the left ear (r=0.359, p=0.072). However, wave V latency and interpeak wave I-V latency were significantly longer in children without hyperacusis (right ear: p=0.042 and p=0.050; left ear: p=0.005 and p=0.004), while interpeak wave III-V only in the left ear (p=0.006) and wave III only in the right ear (p=0.029). Conclusion: There was no significant correlation between ABR wave latencies or hyperacusis and the severity of ASD, while ABR wave latencies were generally longer in children without hyperacusis. Further large studies involving a broader spectrum of children with ASD are warranted to confirm our findings.

KEYWORDS:
Autism spectrum disorder, brainstem evoked response audiometry, hyperacusis

INTRODUCTION

ASD is a heterogeneous condition impacting social communication deficits, repetitive behavior disorders, and limited interests.[1] According to the World Health Organization, about 1 in 100 children were affected by autism spectrum disorder (ASD) worldwide, with boys at a four-fold increased risk than girls. In the US, ASD is about 18.5 cases per 1000 children, or 1 in 54 children.[2] Indonesian data is limited but tertiary care hospital reported that ASD constitute nearly 10% of pediatric outpatients aged 18-48 months.[3] ASD are more prone to auditory disorders, including hyperacusis which could be the sign of auditory pathway disturbance or sign of abnormalities in the limbic system , which significantly impacts their quality of life.[4]

ASD diagnosed Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5). However, diagnosis is often delayed, with an average of four years old, despite early symptoms onset.[5] This puts the children at higher risks of morbidity as prompt intensive treatments for example habituation training, cognitive behavioural therapy (CBT), noise-attenuated headphones, and medication such as risperidone that may induce long-term benefits for the children's social and life quality.[6,7] Currently, auditory brainstem response (ABR) testing is being explored as a potential diagnostic tool for ASD.[8] Studies have shown prolonged ABR wave latencies in ASD, particularly in waves III, V, and interpeak interval, suggesting its diagnostic value.[8,9] Despite this, evidence is lacking on ABR characteristics across the ASD spectrum and its correlation with hyperacusis.

This article was accepted: 16 September 2024
Corresponding Author: Semiramis Zizlavsky
Email: semiramiszizlavsky@gmail.com

Therefore, this study aims to characterize ABR wave latencies in children with ASD and correlate its findings with ASD severity and explore the association between ABR wave latencies and hyperacusis in children with ASD.

MATERIALS AND METHODS

This study was an analytical cross-sectional study involving children aged 3-8 years old diagnosed with ASD according to the Fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) and a national guideline (Pedoman Penggolongan dan Diagnosa Gangguan Jiwa III) presenting to our outpatient clinic between October and December 2023.[9]

The sample size was calculated using a sampling formula, with an additional 10% added to account for potential dropouts, resulting in a total of 26 patients.[10] To control for potential confounders, we only included children with normal hearing function, as defined by a normal tympanometry and distortion product otoacoustic emission finding, and a normal hearing threshold prediction of 20 dB from ABR click and 500 Hz tone burst. Children with known neurological disabilities e.g., cerebral palsy, Down syndrome, or epilepsy were excluded.

The included children were tested for ABR click and tone burst 500 Hz used (Bio Logic Natus 102 System Corp Navigator Pro BERA device). We recorded the absolute latency of waves I, III, and V, and the interpeak latency of waves I-III, III-V, and I-V. Wave prolongation was included if the absolute latency of waves I, III, V exceeded 1.8 m/s, 3.9 m/s, and 5.8 m/s, and if the interpeak latency 1-III, III-V, I-V if exceeded 2.2 m/s, 2.2 m/s, and 4.4 m/s.[11] ABR electrode placement was consistent across all patients. Hyperacusis was diagnosed using the M-CHAT-R questionnaire.[12] Lastly, the severity of ASD was assessed using the Childhood Autism Rating Scale (CARS) instrument.[z]

Descriptive findings were tabulated and presented in frequencies and proportions for categorical variables, and in mean ± standard deviation (SD) or median and interquartile ranges (IQR) for continuous variables, depending on the result of normality distribution tests with Shapiro-Wilk test, acknowledging the small number of subjects recruited in this study (n<50). The association between hyperacusis with ABR findings and CARS score were tested using independent sample T-tests or Mann-Whitney U tests, while the correlation between CARS and ABR findings were analysed using Pearson or Spearmen tests, whichever appropriate. These statistical tests were chosen due to their suitability for comparing means between groups and assessing correlations based on the data distribution and measurement scale. All analyses were performed using the Statistical Package for Social Science 27.0 (SPSS Inc., Chicago, IL), and a two-sided p-value of <0.05 were deemed statistically significant.

RESULTS

A total of 26 normal-hearing children with ASD (52 ears) were included for analysis. Twenty-two of them were boys (88.5%), and a majority were aged between three and five years old (61.5%) with median (5.00 [4.75-6.25]). CARS in children were categorize into mild- moderate (10 [38.5%]) and severe (16 [61.5%]) with median of (40.00 [35.00 - 43.00]). About 69.2% children had reported symptoms pertaining to hyperacusis (Table I). ABR findings revealed that a majority of the study population had a prolonged latency of interpeak wave I-III (46 ears, 88.5%), followed by wave III (22 ears, 42.3%), wave V (11 ears, 21.6%), and interpeak wave I-V (8 ears, 15.4%). On the other hand, prolonged latency in wave I and interpeak wave I-III were observed in only one (1.9%) and three ears (5.8%), respectively (Table II).

Correlation between ABR findings, hyperacusis and the severity of ASD

No statistically significant correlation was found between ABR wave latencies and the severity of ASD, as tested using CARS (Table III). However, we found a slight, marginally significant correlation between wave III latency in the left ear and CARS score (r=0.359, p=0.072). Similarly, the severity of ASD was also not associated with the occurrence of hyperacusis (median CARS score: 39.5 [IQR 35.0-43.0] vs. 40.5 [35.5-43.0], p=0.737. (Figure 1) The box plot shows the distribution of CARS scores in individuals with and without hyperacusis. There was no significant difference in median CARS score between the two group (p=0.737) (Figure 1).

Correlation between ABR findings and hyperacusis

We found that the absolute latency of wave V in both ears were shorter in children with hyperacusis (right ear: median 5.57 ms [IQR 5.39-5.72] vs 5.74 ms [IQR 5.65-6.03], p=0.042; left ear: 5.57 [5.42-5.72] vs 5.85 [5.67-6.14], p=0.005). Similarly, the latency of interpeak wave I-V were also shorter in children with hyperacusis (right ear: 4.16 ms [3.89-4.22] vs 4.25 [4.10-4.66], p=0.050; left ear: 4.08 ms [3.83-4.16] vs 4.40 [4.21-4.67], p=0.004). (Figure 2A, 2B) Meanwhile, the absolute latency of wave III was only significantly shorter in the right ear (3.80 ms [3.70-3.92] vs 3.93 ms [3.87-3.98], p=0.029), while the latency of interpeak wave III-V only in the left ear (1.73 ms [1.61-1.87] vs 1.90 ms [1.80-2.16], p=0.006). No significant correlation was found for other ABR waves (Figure 2A, 2B).

DISCUSSION

Auditory brainstem response (ABR) is an examination of the synchronization activity of the auditory nerve in response to acoustic stimuli. The electrical potentials from cranial nerve VIII and neurons along the brainstem are recorded with electrodes on the scalp in the form of electrophysiological waves.[14] There are three important waves in ABR: wave I, originating from the distal part of nerve VIII; wave III, from the cochlear nucleus; and wave V, from the lateral lemniscus and inferior colliculus.[15] Previous studies have shown that there were substantial changes in the superior olivary complex in children with ASD, suggesting that hypoplasia and dysmorphology occur throughout the auditory brainstem in the pathophysiologic process of ASD, thus resulting in abnormal wave latencies during ABR testing.[16] This is consistent with our findings where most abnormalities were observed in wave III and V, thus confirming that structural abnormalities in ASD children occurred in the central auditory system.

Table I: Subject characteristics

Characteristics	N	(%)
Age (years)		5.00 (4.75-6.25)
3-5	16	61.5%
6-8	10	38.5%
Sex		
Boys	23	88.5%
Girls	3	11.5%
Hyperacusis		
Yes	18	69.2%
No	8	30.8%
CARS		40.00 (35.00 - 43.00)
Mild- moderate	10	38.5%
Severe	16	61.5%

Data are presented in frequencies and proportions

Table II: Proportion of prolonged absolute latency among the study population as tested with auditory brainstem response click (N=52 ears)

		Right ear; n (%)		Left ear; n (%)		Total; n (%)	
		Prolonged	Normal	Prolonged	Normal	Prolonged	Normal
ABR wave	Wave I	0 (0%)	26 (100%)	1 (3.8%)	25 (96.2%)	1 (1.9%)	51 (98.1%)
	Wave III	11 (42.3%)	15 (57.7%)	11 (42.3%)	15 (57.7%)	22 (42.3%)	30 (57.7%)
	Wave V	4 (15.4%)	22 (84.6%)	7 (26.9%)	19 (73.1%)	11 (21.6%)	41 (78.4%)
	Int wave I-III	24 (92.3%)	2 (7.7%)	22 (84.6%)	4 (15.4%)	46 (88.5%)	6 (11.5%)
	Int wave III-V	2 (7.7%)	24 (92.3%)	1 (3.8%)	25 (96.2%)	3 (5.8%)	49 (94.2%)
	Int wave I-V	3 (11.5%)	23 (88.5%)	5 (19.2%)	21 (80.8%)	8 (15.4%)	44 (84.6%)

Table III. Correlation between auditory brainstem response wave latencies and the severity of autism spectrum disorders

	Latency	Correlation (r)	P-value
Right ear	Wave I	-0.212	0.299[b]
	Wave III	0.201	0.325[a]
	Wave V	0.065	0.752[b]
	Int wave I-III	0.088	0.667[a]
	Int wave III-V	-0.085	0.680[b]
	Int wave I-V	0.135	0.512[a]
Left ear	Wave I	-0.03	0.883[b]
	Wave III	0.359	0.072[a]
	Wave V	0.092	0.655[a]
	Int wave I-III	0.209	0.305[b]
	Int wave III-V	-0.179	0.382[a]
	Int wave I-V	0.120	0.561[b]

[a]Pearson correlation test; [b]Spearman Rank test

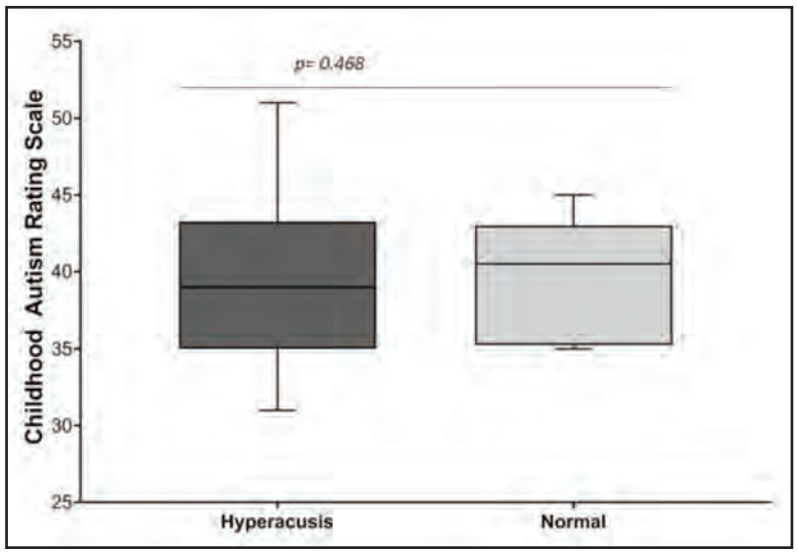

Fig. 1: Association between ASD severity and occurrence of hyperacusis

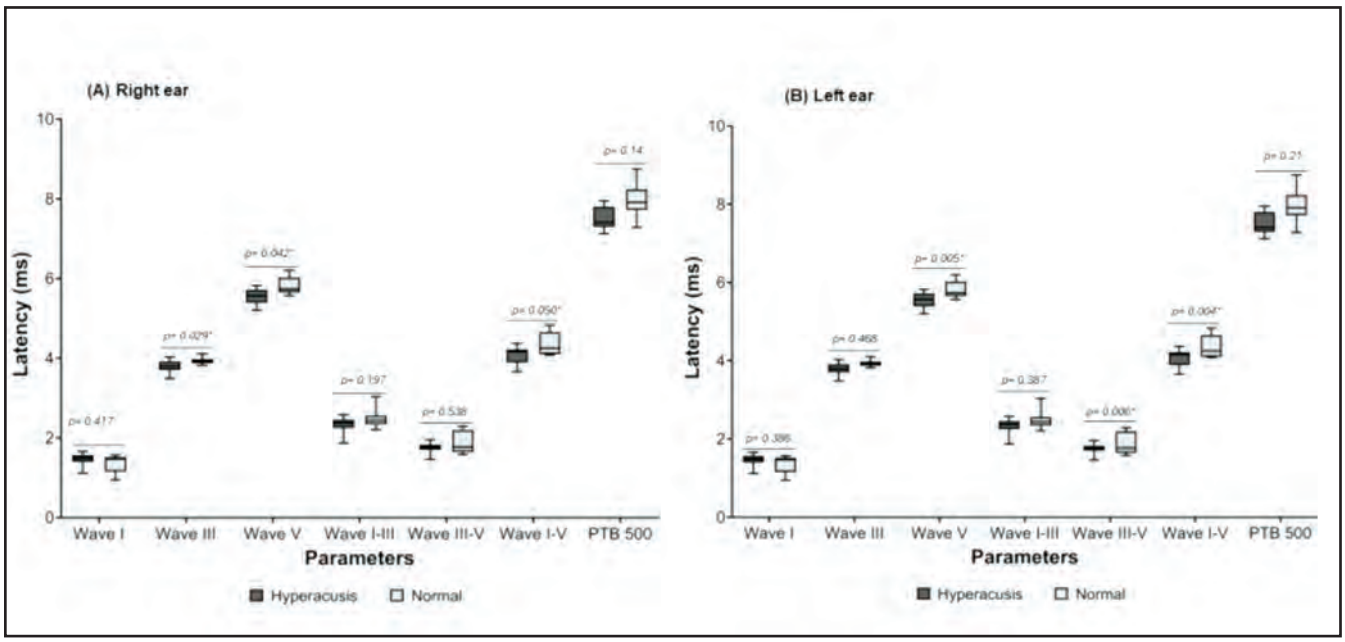

Fig. 2: Box plot illustrating ABR findings in individual with hyperacusis and normal hearing. Asterisk (*) denotes statistically significant difference between groups for specific ABR wave.

The ASD population is highly heterogeneous, leading to varied ABR responses. For example, a study by Madelyn Cate found that while some ASD children showed significantly longer latencies, others did not, suggesting that the differences may be more central in the auditory system rather than at the brainstem level. Additionally, ABR findings can vary based on the age and developmental stage of children, with younger children exhibiting different latencies. Sensory processing differences is a hallmark of ASD, it can also affect ABR results, as observed in Cate's study.[17]

The present study revealed that most ASD children had a prolonged latency in waves III and V, and in interpeak waves I-III, III-V, and I-V; with the prolongation of interpeak waves I-III observed in most cases. These finding are consistent with a meta-analysis by Talge et al., which demonstrated a link between ASD and prolonged latencies in wave III, V, and interpeak latencies I-III and I-V. 18 The speed of action potentials is primarily determined by the degree of myelination, path length, and axonal diameter but also can influenced by synchronization of neuronal signal release of changes in synaptic efficacy, as noted in studies by Distefano et al. and Waddington et al. Hypermyelination and hypomyelination of brainstem pathways is also frequently associated with ASD.[18–20]

These auditory brainstem abnormalities, particularly in the superior olivary complex, may contribute to delayed speech development, difficulty with sound localization, and altered sensory responses to auditory stimuli. These deficits can impair auditory processing and attention, which are clinically relevant in managing sensory challenges in ASD.[21,22] Furthermore, we also observed an asymmetrical prolongation of absolute wave latencies between the right and left ears, which might be explained by the fact that changes in cortical thickness asymmetry was found mostly in frontal, orbitofrontal, and temporal areas. The impact on ABR wave latency changes remain unconclusive and still needs further research to be understood.[23]

In addition to slower sound perception process, children with ASD are also vulnerable to sound hypersensitivity. These children typically frequently cover their ears when hearing exhausts, blenders, or certain phone rings that are usually perceived as normal sound stimuli in healthy children.[24,25] Our study proves the prevalent hyperacusis in children with ASD with more than two-thirds of the study population experiencing the symptoms. This is in line with the previous findings by Carson et al., who reported a prevalence of hyperacusis of 60.2% in children with ASD.[26] Nevertheless, further analysis revealed that the occurrence of hyperacusis was not correlated with the severity of ASD. While a plausible explanation for this phenomenon is yet to be fully known, it has been postulated that there is an extreme variability in hyperacusis with different individual ASD cases.[7] In addition, the severity of hyperacusis, which was not sought in the present study, may also contribute to the observed findings, especially considering in previous study most ASD children mild hyperacusis.[2]

To this date, the mechanism of hypersensitivity and hyposensitivity to auditory stimulus in children with ASD remain unclear. Imaging studies have identified weakened noise control that disrupts verbal message processing from the cochlear nucleus to the inferior colliculus and cortex.[27] There are several possible mechanisms leading to sound hypersensitivity. First, the medial olivary system in the superior olivary complex, which plays an essential part in filtering background noise in noisy environments and modulate cochlear function to prevent cochlear damage due to loud sounds, is found to be dysmorphic in children with ASD.16 In addition, in ASD children, auditory stimuli from

the lateral lemniscus are directly propagated to amygdala and the limbic system without passing through the geniculate body, thus resulting in excessive emotional responses upon hearing certain sounds.[27]

In the present study, we did not observe any correlation between ABR wave latencies and ASD severity. Our findings conform to a previous report by Samy et al., who also found no significant correlation between the absolute latency and interpeak latency of ABR click with CARS.[10] However, different findings was reported by Liu et al., who demonstrated a significant association between CARS and the wave III latency on the right ear (r=0.693), and interpeak latency I-III on the right (r=0.62) and left ear (r=0.594).[28] These suggest that the correlation between ABR wave latencies and CARS score remain equivocal, thus warranting further investigation as the underlying mechanism leading to these conflicting findings remain unknown. However, it should be noted that ABR wave latencies are likely to be influenced by stimulation frequency and repetition, and thus ABR patterns in children with ASD may vary widely.[28]

On the other hand, the present study indicates that ABR wave latencies may be correlated with hyperacusis, especially in wave V and I-V. This has been previously demonstrated by Refat et al., which showed that the absolute latency of wave III and V were shorter in children with hyperacusis.[29] Additionally, this correlation was observed in an exposure-gradient relationship where absolute wave III and V latencies shortened with increasing duration of hyperacusis. This might be explained by the possibility of specific over-activation of the medial olivocochlear system in the brainstem, and the type II cochlear afferents at the level of outer hair cells, thus resulting in a diminished baseline motile responses due to the activation of posteroventral cochlear nucleus neurons triggered.[29]

The present study presents with several limitations. First, the absence of healthy controls limited the interpretability of our findings. In particular, we were unable to investigate the correlation of ABR wave latencies with the occurrence of ASD, and the association between ABR wave latencies and ASD severity with a broader spectrum of hyperacusis severity. In addition, the cross-sectional design limited our ability to investigate the long-term trends of ABR findings and the progression of ASD and hyperacusis over time. To our knowledge, this is one of the very few studies investigating the association between ABR wave latencies, hyperacusis, and ASD severity. Further large-scale studies investigating ASD children with a broader spectrum of severity of ASD and hyperacusis are required to corroborate our findings.

CONCLUSION

There was no significant correlation between the absolute latency of ABR waves, hyperacusis, and ASD severity in young children with normal hearing. However, there was prolonged absolute and interpeak latencies, particularly in waves I-III, III, and V. Interestingly, ABR latencies were generally shorter in children with hyperacusis, likely reflecting its underlying pathophysiology. ABR wave latency could be a useful tool for early ASD screening, enabling timely intervention and therapy, which can help improve outcomes by reducing morbidity and enhancing the quality of life for patients.

ACKNOWLEDGEMENT

None

FUNDING

None

CONFLICT OF INTEREST

The authors declare no conflict of interest

REFERENCES

1. Baumer N, Spence SJ. Evaluation and Management of the Child With Autism Spectrum Disorder. Contin Lifelong Learn Neurol 2018; 24(1): 248–75.
2. Maenner MJ, Warren Z, Williams AR, Amoakohene E. Prevalence and Characteristics of Autism Spectrum Disorder Among Children Aged 8 Years — Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2020. 2024; 72(2).
3. Windiani IGT, Soetjiningsih S, Sugitha AGA, Lestari KA. Indonesian Modified Checklist for Autism in Toddler, Revised with Follow-Up (M-CHAT-R/F) for Autism Screening in Children at Sanglah General Hospital, Bali-Indonesia. Indones Modif Checkl Autism Toddler, Revis with Follow Autism Screen Child Sanglah Gen Hosp Bali-Indonesia. 2016; 5(2).
4. McKenna K, Prasad S, Cooper J, King AM, Shahzeidi S, Mittal J, et al. Incidence of Otolaryngological Manifestations in Individuals with Autism Spectrum Disorder: A Special Focus on Auditory Disorders. Audiol Res 2024 4; 14(1): 35–61.
5. Rinaldi LJ, Simner J, Koursarou S, Ward J. Autistic traits, emotion regulation, and sensory sensitivities in children and adults with Misophonia. J Autism Dev Disord 2023; 53(3): 1162–74.
6. Zwaigenbaum L, Penner M. Autism spectrum disorder: advances in diagnosis and evaluation. BMJ 2018; k1674.
7. Danesh AA, Howery S, Aazh H, Kaf W, Eshraghi AA. Hyperacusis in Autism Spectrum Disorders. Audiol Res 2021; 11(4): 547–56.
8. Martin A, Bloch MH, Volkmar FR. Autism Spectrum Disorder. In: Lewis's Child And Adolescent Psychiatry. 5th ed. 2018. p. 1164–86.
9. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorder. 2013. 50 p.
10. Samy KL, Osman DM, Selim MH, Mohamed RA. Communication skills, sensory integration functions, and auditory brainstem response: findings in a group of Egyptian children with autistic features. Egypt J Otolaryngol 2012; 28(2): 117–26.
11. Prendergast G, Tu W, Guest H, Millman RE, Kluk K, Couth S, et al. Supra-threshold auditory brainstem response amplitudes in humans: Test-retest reliability, electrode montage and noise exposure. Hear Res 2018; 364: 38–47.
12. Soetjiningsih, Windiani IGAT, Adnyana IGANS. Pedoman Pelatihan- Deteksi Dini dan Diagnosis Gangguan Spektrum Autisme (GSA). UKK Tumbuh Kembang-Pediatri Sosial Bagian/SMF Ilmu Kesehatan Anak FK UNUD-Sanglah, editor. Denpasar; 2015. 60 p.
13. Moon SJ, Hwang JS, Shin AL, Kim JY, Bae SM, Sheehy-Knight J, et al. Accuracy of the Childhood Autism Rating Scale: a systematic review and meta-analysis. Dev Med Child Neurol 2019; 61(9): 1030–8.
14. Picton TW. Auditory brainstem responses: peaks along the way. In: Human auditory evoked potentials. 2011. p. 215.

15. Källstrand J, Niklasson K, Lindvall M, Claesdotter-Knutsson E. Reduced thalamic activity in ADHD under ABR forward masking conditions. Appl Neuropsychol Child 2022;16:1–7.
16. Mansour Y, Kulesza R. Three dimensional reconstructions of the superior olivary complex from children with autism spectrum disorder. Hear Res 2020;393:107974.
17. Cate M. Auditory Brainstem Response in Autistic Children: Potential Implications for Sensory Processing. Brigham Young University; 2022.
18. Talge NM, Tudor BM, Kileny PR. Click-evoked auditory brainstem responses and autism spectrum disorder: A meta-analytic review. Autism Res 2018;11(6):916–27.
19. Gonçalves AM, Monteiro P. Autism Spectrum Disorder and auditory sensory alterations: a systematic review on the integrity of cognitive and neuronal functions related to auditory processing. J Neural Transm 2023; 14;130(3):325–408.
20. Hanaie R, Mohri I, Kagitani-Shimono K, Tachibana M, Matsuzaki J, Hirata I, et al. White matter volume in the brainstem and inferior parietal lobule is related to motor performance in children with autism spectrum disorder: A voxel-based morphometry study. Autism Res 2016;9(9):981–92.
21. Kabil SE, Abdelshafy R, Ahmed AIA, Zahran AM, Attalah M, Sallam Y, et al. Mismatch Negativity and Auditory Brain Stem Response in Children with Autism Spectrum Disorders and Language Disorders. J Multidiscip Healthc 2023;16:811–7.
22. Miron O, Ari-Even Roth D, Gabis L V., Henkin Y, Shefer S, Dinstein I, et al. Prolonged auditory brainstem responses in infants with autism. Autism Res 2016;9(6):689–95.
23. Postema MC, van Rooij D, Anagnostou E, Arango C, Auzias G, Behrmann M, et al. Altered structural brain asymmetry in autism spectrum disorder in a study of 54 datasets. Nat Commun 2019;10(1):4958.
24. Gravel JS, Dunn M, Lee WW, Ellis MA. Peripheral Audition of Children on the Autistic Spectrum. Ear Hear 2006;27(3):299–312.
25. Gomes E, Pedroso FS, Wagner MB. Hipersensibilidade auditiva no transtorno do espectro autístico. Pró-Fono Rev Atualização Científica 2008;20(4):279–84.
26. Carson TB, Valente MJ, Wilkes BJ, Richard L. Brief Report: Prevalence and Severity of Auditory Sensory Over-Responsivity in Autism as Reported by Parents and Caregivers. J Autism Dev Disord 2022; 10;52(3):1395–402.
27. Stefanelli ACGF, Zanchetta S, Furtado EF. Hiper-responsividade auditiva no transtorno do espectro autista, terminologias e mecanismos fisiológicos envolvidos: revisão sistemática. CoDAS 2020;32(3).
28. Liu Y, Liu Z, Diao T, Song P, Hu Q, Jiang N. Exploring the Relationship Between Auditory Brainstem Response Testing and Disease Progression in Pediatric Autism Spectrum Disorder. Altern Ther Health Med 2024;30(3):51–5.
29. Refat F, Wertz J, Hinrichs P, Klose U, Samy H, Abdelkader RM, et al. Co-occurrence of Hyperacusis Accelerates With Tinnitus Burden Over Time and Requires Medical Care. Front Neurol 2021;18;12.