http:// jpp.mums.ac.ir

Original Article (Pages: 19650-19660)

Exploring Hemispheric Contributions in the Processing of Social Speech in Autism

* Sara Sharghilavan [1,2], Leila Mehdizadeh Fanid [1], Oana Geman [2,3,4] , Hassan Shahrokhi [5] , Hadi Seyedarabi [6]

[1] Department of Cognitive Neuroscience, Faculty of Education and Psychology, University of Tabriz, Tabriz, Iran.
[2] Data Science and AI, Computer Science and Engineering Department, Stefan cel Mare University of Suceava, Suceava, Romania.
[3] Data Science and AI, Computer Science and Engineering Department, Chalmers University of Technology.
[4] Data Science and AI, Computer Science and Engineering Department, University of Gothenburg, Gothenburg, Sweden.
[5] Autism and Related Neurodevelopmental Disorders Research Team, Tabriz University of Medical Sciences, Tabriz, Iran.
[6] Faculty of Electrical and Computer Engineering, University of Tabriz, Tabriz, Iran.

Abstract

Background: Based on the lateralization of hemispheres in certain cognitive functions and the lack of knowledge of these dimensions in autism, this study aimed to compare the contribution of hemispheres to auditory spatial attention during socially relevant speech processing in children with autism compared to their typically developing peers (TD).

Methods: The participants were 27 Romanian children, including 12 children with autism and 15 typically developing children. Auditory stimuli were simple three-word Romanian sentences presented in an oddball pattern under three listening conditions: right ear, left ear, and binaural. We extracted the P300 event-related potential (ERP) component in response to all conditions and compared the two groups. Statistical analyses were performed using multivariate analysis of variance (MANOVA) with follow-up tests for between-subject effects.

Results: Multivariate analyses showed no significant overall differences across groups in the different conditions. However, between-subjects effects tests revealed a significant reduction in P300 amplitude for the left ear condition in the Autism spectrum disorder (ASD) group compared with TD peers ($p = 0.042$, partial $\eta^2 = 0.155$). Across all conditions, latency differences were not statistically significant.

Conclusion: The selective reduction in P300 amplitude for left ear input in children with ASD indicates reduced attentional engagement with socio-semantic aspects of speech that are predominantly processed in the right hemisphere. The findings emphasize the importance of lateralization-sensitive auditory patterns in understanding and addressing communication deficits in ASD.

Key Words: Auditory Spatial Attention; Autism; Event-related potentials; Hemispheric lateralization; P300; Speech Processing.

* Please cite this article as: Sharghilavan S, Mehdizadeh Fanid L, Geman O, Shahrokhi H, Seyedarabi H. Exploring Hemispheric Contributions in the Processing of Social Speech in Autism. J Ped Perspect 2025; 13 (9):19650-19660. **DOI: 10.22038/jpp.2025.90441.5589**

Corresponding Author:

Sara Sharghilavan; Department of Cognitive Neuroscience, Faculty of Education and Psychology, University of Tabriz,Tabriz, Iran.Data Science and AI, Computer Science and Engineering Department, Stefan cel Mare University of Suceava, Suceava, Romania. Email: sara.sharghi2020@gmail.com; sara.sharghi@tabrizu.ac.ir

1- INTRODUCTION

Autism spectrum disorder (ASD) is recognized as a neurodevelopmental disorder marked by limited interests, unusual sensory responses, and communication difficulties (1,2,3). Among all sensory domains, auditory spatial processing plays a critical role in how individuals with ASD interact with their social environment (4). The inability to properly navigate social stimuli in their environment, including speech, which is referred to as social orientation deficits (5, 6), may deprive children with ASD of opportunities for learning and social development (7,8). However, the neural dynamics underlying auditory spatial attention in autism remain insufficiently understood.

Auditory spatial attention is a cognitive function that directs auditory perception toward a specific sound source in an auditory spatial environment (9). The auditory spatial environment includes three dimensions: depth, vertical, and horizontal, with the horizontal dimension helping locate objects for navigation and routing (10). Auditory spatial attention processing and orientation involve interhemispheric coordination, where right-ear stimuli are mainly directed to the left hemisphere and left-ear stimuli to the right hemisphere (11, 12). The left hemisphere primarily processes rapid sound patterns, such as phonemes and syllables, and handles precise temporal features of language. The right hemisphere, in contrast, is specialized in social and pragmatic aspects of auditory input, slower patterns like sentences, and acoustic features such as pitch and timbre (13,14). Together, the hemispheres integrate spatial and linguistic information, enabling recognition of both sound location and meaning. Disruptions in interhemispheric coordination may impair these abilities. Disruption in the interaction or coordination between the hemispheres can impair both the spatial and linguistic aspects of auditory perception. Based on the neuroanatomical organization of the auditory pathways, as mentioned, speech stimuli are initially transmitted to the contralateral hemisphere before reaching the hemisphere corresponding to the receiving ear. Therefore, assessing hemispheric function via contralateral ear stimulation in an auditory spatial environment is crucial for accurately investigating the attention and neural processing of speech in individuals with ASD. Many studies have examined auditory processing in ASD, focusing on early sound recognition and speech perception. However, how children with ASD allocate spatial attention to speech cues in the horizontal plane remains unclear. For example, Soskey et al. reported that children with autism exhibit more dispersed auditory spatial attention compared to neurotypical children when oriented forward (9). They also responded to non-target stimuli at close range. Furthermore, studies indicate that children with ASD have more diffuse spatial attention and difficulty accurately locating sounds (15). Studies further show that autistic individuals exhibit poorer performance than neurotypical individuals in detecting the spatial origin of sounds, both when sounds are co-located and when they come from two distinct directions (left and right ±30°) (16). Although deficits in auditory spatial attention among individuals with ASD have been documented, most existing studies focus on target selection tasks involving spatially distributed distractors. In contrast, limited research has examined how spatial attention functions in response to isolated speech stimuli. Therefore, it seems necessary to investigate speech stimuli in the auditory spatial environment.

Detailed examination of differences between ASD and neurotypical individuals can be achieved through neurophysiological methods, including

EEG, which enables continuous monitoring of brain activity (17), and event-related potentials (ERPs), which reflect the brain's responses to sensory input (18, 19). The P300 is an ERP component often used to study auditory spatial attention. It appears as a positive EEG deflection following an infrequent sound amid frequent stimuli, peaking around 300 ms after onset (20). Amplitude ranges from 2 to 20 μV, and latency from 250 to 500 ms (21,22), with strongest expression in parieto-central regions (23). Latency reflects cognitive processing speed, while amplitude indicates attentional resource allocation (24).

Therefore, filling this gap in the literature, this study aimed to examine auditory spatial attention to speech-based directional cues in ASD and to compare it with typically developing (TD) peers by analyzing the amplitudes and latencies of the P300 ERP component across three horizontal direction conditions.

2- MATERIALS AND METHODS

2-1. Participants

The study included 27 participants, 12 Romanian children diagnosed with ASD [mean age = 9.7 years, range 7–12] and 15 neurotypical children (mean age = 9.3 years, range 7–12). They were matched for intelligence, handedness, culture, socioeconomic status, and age. All participants had normal hearing thresholds (≤25 dB) across frequencies ranging from 250 to 8000 Hz and had no history of psychiatric, neurological, or other mental disorders. They were not taking any medications at the time of the study. Medical records showed that children with ASD had been assessed with the CELF-4 and the Sensory Profile (SP) previously and didnot show clinically significant impairments in language abilities or auditory sensory processing. Level-1 ASD diagnoses were confirmed thourgh formal assessment using the Autism Diagnostic Interview–Revised (ADI-R) (25), Autism Diagnostic Observation Schedule (ADOS), Childhood Autism Rating Scale (CARS), and Vineland Adaptive Behavior Scales. The study protocol was approved by the Research Ethics Committees of Ștefan cel Mare University and the University of Tabriz (IR.TABRIZU.REC.1403.172). Written informed consent was obtained from the legal guardians or parents of all participants prior to the study initiation, following the Declaration of Helsinki (26).

2-2. Stimuli

This research is part of a larger project. The auditory stimuli consisted of very short three-word sentences describing simple objects in Romanian derived from elementary school textbooks (for example: Vaza este albă = The vase is white). Each sentence had two syllables and was presented to the subjects in an oddball task. The auditory stimuli were recorded at a natural Romanian speech rate of 3.5 syllables per second, at a frequency of approximately 174 Hz, and then the fundamental frequency (F0) of all auditory stimuli was normalized to insure a constant and uniform sound range to minimize individual and physiological effects. The stimuli were then manipulated for binaural, right-ear monocular, and left-ear monocular presentation using the PyAudio library.

2-3. Apparatus

Neurophysiological techniques, such as ERPs, which isolate specific brain responses, have greatly enhanced our understanding of neural function. ERPs reflect changes in EEG activity elicited by sensory stimuli (27). In this study, we utilized the Ultracortex Mark IV EEG headset, developed by OpenBCI, as the brain-computer interface (BCI). The headset is equipped with 16 dry electrodes arranged according to the international 10–20 system, providing broad coverage of critical cortical regions. It connects to the

Cyton board, a versatile biosensor platform capable of recording EEG, EMG, and ECG signals. Data are sampled at 250 Hz and transmitted wirelessly to a computer via an RFduino Bluetooth module with a USB dongle. This wireless configuration reduces movement restrictions, making it especially suitable for experiments involving children or conducted in low-stimulation settings (28).

2-4. Experimental Procedure

In this study, eligible participants underwent individual, eyes-closed EEG recording sessions in a quiet, controlled environment to minimize artifacts. Brain activity was recorded using a 19-channel Ultracortex Mark IV EEG headset (OpenBCI). This study was part of a 4-session Oddball task experiment that examined the effects of different acoustic aspects of speech stimuli on the processing, perception, and attention of the ASD group compared to the TD group. In this part of the Oddball task, where directional stimuli were presented, participants were asked to actively listen to auditory stimuli. These stimuli were Romanian spoken sentences that were presented binaurally at 65 dB SPL at baseline and to the right ear at 65 dB SPL and the left ear at 65 dB SPL for deviants. In this task, the auditory stimuli were presented in a pseudo-random order with 25% deviant sentences and 75% standard sentences. The experimental session, which lasted 18 minutes and 33 seconds, consisted of three blocks of 100 trials each. To reduce participant fatigue, a 2-minute break was given between blocks. The duration of each sentence as an auditory stimulus was 1.71 seconds, and the inter-stimulus interval (ISI) was 1.2 seconds. The entire task took approximately 18 minutes and 33 seconds. ERP analysis was synchronized with the onset of each sentence to ensure accurate timing.

A custom Python interface was developed to precisely control stimulus presentation and synchronize ERP event markers with EEG recordings (29), thereby facilitating high-resolution analyses of neural responses to speech stimuli. Upon completion of each recording session, participants' attentiveness was verified through comprehension questions related to the auditory material. Data from participants who failed to provide satisfactory responses were excluded from subsequent analyses.

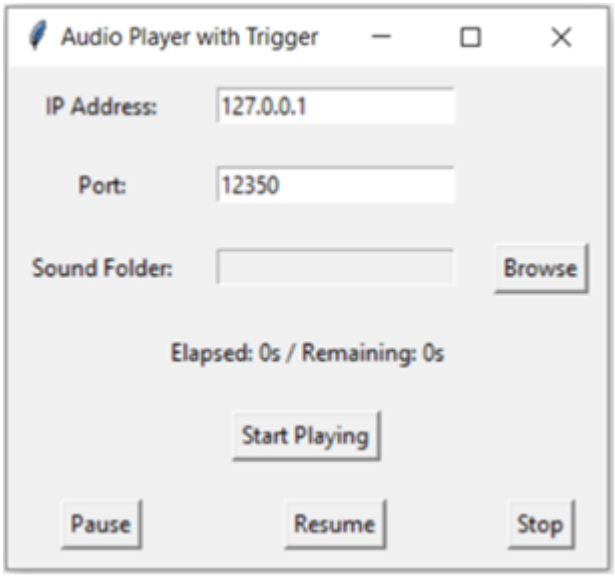

Figure-1: Custom Python interface for this project (29).

After data acquisition, EEG signals containing ERP markers were processed using Python, with the Pandas, NumPy, and Matplotlib libraries employed for data management, statistical analysis, and visualization.

2-5. Data Analysis

2-5-1. Python

After acquiring the data, EEG signals in BDF format containing ERP markers were processed using Python libraries, such as NumPy, Pandas, and Matplotlib. The preprocessing involved applying a 4th-order Butterworth band-pass filter (0.1–40 Hz) to reduce noise, followed by Independent Component Analysis (ICA) to remove ocular and muscle artifacts. Trials with residual artifacts exceeding ±100 µV at any electrode after ICA were excluded, and participants with more than 25% rejected trials in any condition were removed from the final dataset. Baseline correction was performed by subtracting the mean voltage of the 200-ms pre-stimulus interval. Stimulus-locked epochs were extracted from 300 ms before to 1000 ms after stimulus onset, excluding incomplete trials. ERP components were quantified in terms of amplitude (average peak-to-peak voltage) and latency (time of peak or minimum within the expected window), followed by statistical analyses.

For P300 analysis, the maximum positive peak was identified at centro-parietal electrodes within a 250–500 ms post-stimulus window of each epoch. Amplitude was defined as the voltage at this peak relative to baseline, and latency as the time point of the peak. These measures were then submitted to statistical analyses.

2-5-2. SPSS

Statistical analyses were performed using IBM SPSS Statistics 27. A multivariate analysis of variance (MANOVA) was conducted to explore group differences (ASD vs. TD) in P300 peak amplitude and latency. Before analysis, the assumptions of multivariate normality, homogeneity of variance-covariance matrices (Box's M test), and absence of multicollinearity were verified.

3- RESULTS

Descriptive statistics for P300 amplitude and latency are summarized in Figure 2, preceding the inferential analyses.

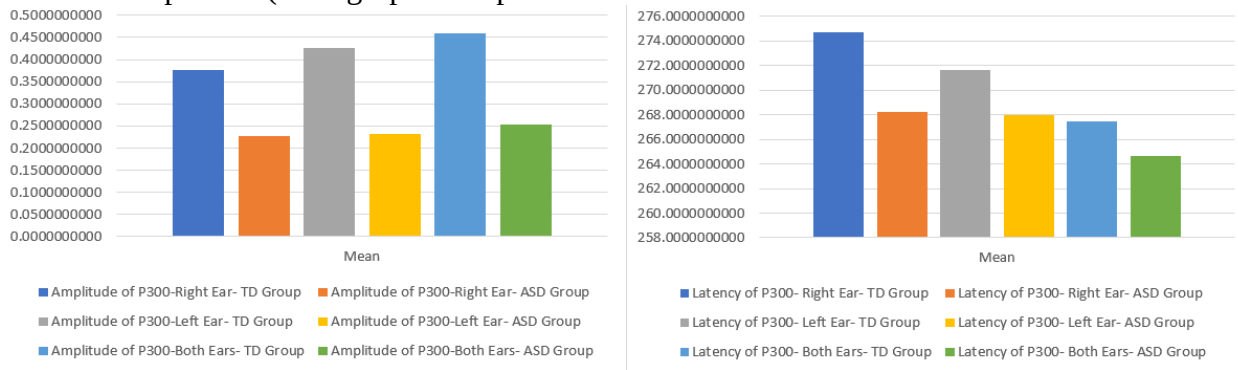

Figure-2: Comparison of P300 amplitude and latency between the ASD group and TD peers across all auditory direction conditions (right-ear, left-ear, and binaural presentation).

The descriptive analysis revealed that the typically developing group had higher P300 amplitudes than the ASD group across all direction conditions, while latencies were slightly shorter in the ASD group. To further examine these differences, a MANOVA was conducted on P300 amplitude and latency across ear

conditions. All assumptions for MANOVA were carefully checked. Univariate normality was confirmed with the Kolmogorov–Smirnov test (all $p > 0.05$), and homogeneity of variance–covariance matrices was supported by a non-significant Box's M test. Linearity and the absence of multicollinearity were confirmed through scatterplots and correlations, and outlier analyses using standardized residuals and Mahalanobis distance found no influential points. With all assumptions met, the data were considered appropriate for MANOVA. The next section details the group differences in ERP amplitude and latency.

Table-1. Summary of multivariate test results for the main effect of group on P300 amplitude and latency, considering all direction deviation conditions combined.

Effect		Value	F	Hypothesis df	Error df	Sig.	Partial Eta Squared
Group	**Pillai's Trace**	0.295	1.397	6.000	20.000	0.264	0.295
	Wilks' Lambda	0.705	1.397	6.000	20.000	0.264	0.295
	Hotelling's Trace	0.419	1.397	6.000	20.000	0.264	0.295
	Roy's Largest Root	0.419	1.397	6.000	20.000	0.264	0.295

A MANOVA was performed to assess the main effect of group on P300 amplitude and latency across all deviation directions. The results were not statistically significant across all indices (Pillai's Trace =0.295, Wilks' Lambda =0.705, Hotelling's Trace=0.419, Roy's Largest Root =0.419; $F(6, 20)=1.397$, $p =0.264$, partial $\eta^2 =0.295$), indicating no significant group effect, although the partial η^2 indicates a moderate effect size. To explore group differences in each directional condition for amplitude and latency of P300, a test of between-subjects effects was conducted.

Table-2. Tests of between-subjects effects on P300 amplitude and latency across directional conditions comparing children with ASD and TD peers.

Source		Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Group	**Amplitude of P300- Right Ear**	0.150	1	0.150	2.777	0.108	0.100
	Latency of P300- Right Ear	276.872	1	276.872	3.071	0.092	0.109
	Amplitude of P300- Left Ear	0.256	1	0.256	4.572	0.042	0.155
	Latency of P300- Left Ear	88.547	1	88.547	1.158	0.292	0.044
	Amplitude of P300- Both Ears	0.285	1	0.285	3.900	0.059	0.135
	Latency of P300- Both Ears	52.267	1	52.267	0.466	0.501	0.018

Subsequent univariate follow-up analyses were conducted to decompose the multivariate effect of group on individual P300 measures. A significant group effect was observed for P300 amplitude in the left ear condition, $F(1, df\ error) =4.572$, $p= 0.042$, partial $\eta^2=0.155$, indicating that approximately 15.5% of the variance in this measure was attributable to group membership. Amplitude measures for the right ear ($F=2.777$, $p=0.108$, partial η^2

=0.100) and binaural presentation (F 3.900, p =0.059, partial η^2 =0.135) were not statistically significant, although effect sizes fell within the small-to-moderate range. Latency measures for the right ear (F =3.071, p =0.092, partial η^2 =0.109), left ear (F=1.158, p=0.292, partial η^2 =0.044), and binaural presentation (F=0.466, p=0.501, partial η^2 =0.018) were all non-significant, with effect sizes ranging from negligible to small. Overall, these findings suggest that group differences were more pronounced in amplitude than latency, with the left ear amplitude representing the only statistically significant effect.

4- DISCUSSION

Our study found a significant group effect on P300 amplitude only in the left-ear condition, with lower amplitudes in children with ASD compared to typically developing peers. Left-ear input mainly activates right-hemisphere regions, which support pragmatic working memory, monitoring of speech, and detection of logical inconsistencies (30). The right hemisphere also encodes long-term speech structures such as sentences and is linked to theory of mind, often impaired in autism (31,32). Thus, our findings align with prior reports of deficits in speech inference (33), pragmatic working memory (34,35), right-hemisphere speech encoding (36,37) and theory of mind in ASD (38–40). The reduced P300 amplitude in the left-ear condition likely reflects weaker engagement with socially relevant semantic information.

Despite the significant group differences observed in P300 amplitude exclusively in the left-ear condition, no statistically significant differences emerged for either the right-ear or binaural conditions. This likely reflects the lateralized organization of auditory processing. Right-ear input activates left-hemisphere regions responsible for phonological, syntactic, and structural aspects of language (41–43). These processes, essential for language comprehension, may be relatively preserved in ASD compared to the pragmatic and social-communicative functions of the right hemisphere.

In the binaural condition, input to both ears activates bilateral pathways and interhemispheric interactions, which may compensate for deficits (44). This integration also increases response variability, making subtle differences harder to detect. Thus, the lack of significant effects in right-ear and binaural conditions does not imply intact processing, but rather that impairments may be weaker, more variable, or require larger samples and sensitive methods to reveal. Additionally, in this study, there was no difference in the latency of the P300 component in either the right or left ear or binaural presentation of the auditory stimulus. Since cognitive load can affect time perception and processing speed (45), and time perception is associated with the latency of event-related potential components (46), the brain provides the possibility of extensive and distributed processing that may stabilize neural temporal responses.

5- CONCLUSION

Our findings indicate ear-specific alterations in P300 amplitude in children with ASD, observed exclusively in the left-ear condition. This underscores the role of right-hemisphere auditory processing in social-communicative function. These findings highlight the importance of hemispheric and ear-specific approaches in ASD research and warrant further investigation in larger samples to confirm and extend these observations.

5-1. Limitations and Future Directions

This study only included male participants to reduce variability in verbal abilities. Females, both with and without

ASD, tend to exhibit stronger language skills and different processing patterns. Therefore, caution is necessary when applying these findings to autistic girls. Despite the small sample size, groups were matched on age, intelligence, native language, socioeconomic and cultural factors, handedness, and ASD functioning level, with no comorbid neurological or psychiatric conditions. Prior therapeutic interventions were also taken into account to minimize environmental influences. Future research with larger, more diverse, and longitudinal samples would enhance generalizability and clinical relevance.

6- ACKNOWLEDGMENTS

The authors would like to sincerely thank Dr. Morteza Izadifar, Dr. Diana Sînziana Duca, Cristina Lemeni, Tiberiu Ciortan, Roxana Toderean, and the Star of Hope Autism Center for their invaluable support and assistance in this research.

7- FUNDING

This research did not receive any external funding.

8- DECLARATIONS

8-1. Conflict of Interest

None of the authors have any potential conflicts of interest to disclose.

8-2. Ethics Approval

All procedures involving human participants were conducted in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by the Research Ethics Committees of the University of Tabriz (IR.TABRIZU.REC.1403.172).

8-3. Consent to Publish

Written informed consent for publication was obtained from all participants or, where applicable, from their parents or legal guardians.

8-4. Consent to Participate

Written informed consent was obtained from all individual participants. For minors, consent was obtained from their parents or legal guardians.

8-5. Declaration of Generative AI and AI-assisted Technologies in the Writing Process

The authors did not use AI and AI-assisted technologies in the writing process.

9- REFERENCES

1. Belmonte MK, Cook EH, Anderson GM, Rubenstein JL, Greenough WT, Beckel-Mitchener A, Courchesne E, Boulanger LM, Powell SB, Levitt PR, Perry EK. Autism as a disorder of neural information processing: directions for research and targets for therapy. Molecular psychiatry. 2004 Jul;9(7):646-63.

2. Dawson G, Toth K. Autism spectrum disorders. Developmental psychopathology: volume three: risk, disorder, and adaptation. 2015 Sep 5:317-57.

3. Naji WA, Waheeb MQ, Hamza DH. Autism Spectrum Disorder. Medico-legal Update. 2020 Apr 1;20(2):321.

4. Goncalves 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. Journal of neural transmission. 2023 Mar;130(3):325-408.

5. Zwaigenbaum L, Bryson S, Rogers T, Roberts W, Brian J, Szatmari P. Behavioral manifestations of autism in the first year of life. International journal of

developmental neuroscience. 2005 Apr 1;23(2-3):143-52.

6. Franchini M, Glaser B, Wood de Wilde H, Gentaz E, Eliez S, Schaer M. Social orienting and joint attention in preschoolers with autism spectrum disorders. PloS one. 2017 Jun 9;12(6):e0178859.

7. Mundy P, Neal AR. Neural plasticity, joint attention, and a transactional social-orienting model of autism. In International review of research in mental retardation 2000 Jan 1 (Vol. 23, pp. 139-168). Academic Press.

8. Chevallier C, Kohls G, Troiani V, Brodkin ES, Schultz RT. The social motivation theory of autism. Trends in cognitive sciences. 2012 Apr 1;16(4):231-9.

9. Soskey LN, Allen PD, Bennetto L. Auditory spatial attention to speech and complex non-speech sounds in children with autism spectrum disorder. Autism Research. 2017 Aug;10(8):1405-16.

10. Voss P. Auditory spatial perception without vision. Frontiers in psychology. 2016 Dec 20;7:1960.

11. Zatorre RJ, Belin P. Spectral and temporal processing in human auditory cortex. Cerebral cortex. 2001 Oct 1;11(10):946-53.

12. Tervaniemi M, Hugdahl K. Lateralization of auditory-cortex functions. Brain research reviews. 2003 Dec 1;43(3):231-46.

13. Friederici AD, Alter K. Lateralization of auditory language functions: a dynamic dual pathway model. Brain and language. 2004 May 1;89(2):267-76.

14. Poeppel D. The neuroanatomic and neurophysiological infrastructure for speech and language. Current opinión in neurobiology. 2014 Oct 1;28:142-9.

15. Teder-Sälejärvi WA, Pierce KL, Courchesne E, Hillyard SA. Auditory spatial localization and attention deficits in autistic adults. Cognitive Brain Research. 2005 May 1;23(2-3):221-34.

16. Emmons KA, KC Lee A, Estes A, Dager S, Larson E, McCloy DR, et al. Auditory attention deployment in young adults with autism spectrum disorder. Journal of autism and developmental disorders. 2022 Apr;52(4):1752-61.

17. Mihai AS, Geman O, Toderean R, Miron L, SharghiLavan S. The Next Frontier in Brain Monitoring: A Comprehensive Look at In-Ear EEG Electrodes and Their Applications. Sensors. 2025 May 25;25(11):3321.

18. Jeste SS, Nelson III CA. Event related potentials in the understanding of autism spectrum disorders: an analytical review. Journal of autism and developmental disorders. 2009 Mar;39(3):495-510.

19. Cotter M, Reisli S, Francisco AA, Wakim KM, Oakes L, Crosse MJ, et al. Neurophysiological measures of auditory sensory processing are associated with adaptive behavior in children with Autism Spectrum Disorder. Journal of neurodevelopmental disorders. 2023 Apr 1;15(1):11.

20. Joos K, Gilles A, Van de Heyning P, De Ridder D, Vanneste S. From sensation to percept: the neural signature of auditory event-related potentials. Neuroscience & Biobehavioral Reviews. 2014 May 1;42:148-56.

21. Polich J. Updating P300: an integrative theory of P3a and P3b. Clinical neurophysiology. 2007 Oct 1;118(10):2128-48.

22. Didoné DD, Oppitz SJ, Gonçalves MS, Garcia MV. Long-latency auditory evoked potentials: Normalization of protocol applied to normal adults. Arch Otolaryngol Rhinol. 2019;5(03):69-73.

23. Luck SJ. Event-related potentials.2023.

24. Raggi A, Serretti A, Ferri R. The P300 component of the auditory event-related potential in adult psychiatric and neurologic disorders: a narrative review of clinical and experimental evidence. International Clinical Psychopharmacology. 2024:10-97.

25. Lord C, Rutter M, Le Couteur A. Autism Diagnostic Interview-Revised: a revised version of a diagnostic interview for caregivers of individuals with possible pervasive developmental disorders. Journal of autism and developmental disorders. 1994 Oct;24(5):659-85.

26. World Medical Association. World Medical Association Declaration of Helsinki. Ethical principles for medical research involving human subjects. Bulletin of the world health organization. 2003 Jul 2;79(4):373.

27. Barry RJ, De Blasio FM, Rushby JA, MacDonald B, Fogarty JS, Cave AE. Stimulus intensity effects and sequential processing in the passive auditory ERP. International Journal of Psychophysiology. 2022 Jun 1;176:149-63.

28. OpenBCI. (n.d.). The complete headset (EEG). Retrieved October 30, 2024, Available from: https://shop.openbci.com/products/the-complete-headset-eeg

29. SharghiLavan S, Geman O, Toderean R. Speech Perception and Speech Attention: A Case Study Based on Event-Related Potential Using the OpenBCI System. In2024 E-Health and Bioengineering Conference (EHB) 2024 Nov 14 (pp. 1-4). IEEE.

30. Ptak R, Schnider A. Disorganised memory after right dorsolateral prefrontal damage. Neurocase. 2004 Feb 1;10(1):52-9.

31. Griffin R, Friedman O, Ween J, Winner E, Happé F, Brownell H. Theory of mind and the right cerebral hemisphere: Refining the scope of impairment. Laterality. 2006 Apr 1;11(03):195-225.

32. Andreou M, Skrimpa V. Theory of mind deficits and neurophysiological operations in autism spectrum disorders: a review. Brain sciences. 2020 Jun 20;10(6):393.

33. Ozonoff S, Miller JN. An exploration of right-hemisphere contributions to the pragmatic impairments of autism. Brain and language. 1996 Mar 1;52(3):411-34.

34. Schuh JM. Pragmatic language abilities: Working memory influences on mutual information. University of Connecticut; 2011.

35. Razavi F, Pourmohamadreza-Tajrishi M, Haghgoo H, Bakhshi E, Tavakoli S, Miri SM. Relationship between executive functions and pragmatic language in children with autism spectrum disorders. Iranian Rehabilitation Journal. 2019 Sep 10;17(3):225-34.

36. Haesen B, Boets B, Wagemans J. A review of behavioural and electrophysiological studies on auditory processing and speech perception in autism spectrum disorders. Research in autism spectrum disorders. 2011 Apr 1;5(2):701-14.

37. Persichetti AS, Shao J, Gotts SJ, Martin A. Maladaptive laterality in cortical networks related to social communication in autism spectrum disorder. Journal of Neuroscience. 2022 Nov 30;42(48):9045-52.

38. Baron-Cohen S. The theory of mind deficit in autism: How specific is it?. British Journal of Developmental Psychology. 1991 Jun;9(2):301-14.

39. Adams MP. Explaining the theory of mind deficit in autism spectrum disorder. Philosophical Studies. 2013 Mar;163(1):233-49.

40. Jones CR, Simonoff E, Baird G, Pickles A, Marsden AJ, Tregay J, et al.

The association between theory of mind, executive function, and the symptoms of autism spectrum disorder. Autism research. 2018 Jan;11(1):95-109.

41. Friederici AD. The brain basis of language processing: from structure to function. Physiological reviews. 2011 Oct;91(4):1357-92.

42. Friederici AD, Chomsky N, Berwick RC, Moro A, Bolhuis JJ. Language, mind and brain. Nature human behaviour. 2017 Oct;1(10):713-22.

43. Turker S, Kuhnke P, Eickhoff SB, Caspers S, Hartwigsen G. Cortical, subcortical, and cerebellar contributions to language processing: A meta-analytic review of 403 neuroimaging experiments. Psychological Bulletin. 2023 Nov;149(11-12):699.

44. Hugdahl K. 12 dichotic listening in the study of auditory laterality. The asymmetrical brain. 2004;441.

45. Barrouillet P, Bernardin S, Portrat S, Vergauwe E, Camos V. Time and cognitive load in working memory. Journal of Experimental Psychology: Learning, Memory, and Cognition. 2007 May;33(3):570.

46. Kononowicz TW, van Rijn H. Decoupling interval timing and climbing neural activity: a dissociation between CNV and N1P2 amplitudes. Journal of Neuroscience. 2014 Feb 19;34(8):2931-9.