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CAUSES OF ALALIA SPEECH IMPAIRMENT

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Abstract. Alalia is a severe developmental speech disorder characterized by the absence or profound underdevelopment of speech due to organic damage to the brain's speech centers during early childhood, before the formation of linguistic ability. The disorder is not the result of hearing loss, intellectual disability, or general developmental delay but arises from neurological immaturity or injury to cortical regions responsible for speech perception and production.
Keywords: alalia, speech development, neuropsychology, cortical damage.

INTRODUCTION

Speech is a uniquely human function, serving as the primary vehicle for thought, communication, and social adaptation. Its development depends on a delicate interaction between biological maturation, sensory experience, and social environment. When this process is disrupted, the result can be a profound and multifaceted speech disorder such as alalia. The term "alalia" originates from Greek, meaning "speechlessness," and in clinical linguistics it refers to a condition where speech fails to develop due to early brain injury affecting cortical speech zones [1].

Unlike aphasia, which denotes the loss of previously acquired speech following brain damage, alalia reflects a failure of speech formation from the outset. Children with alalia usually have normal hearing and preserved intelligence but exhibit severely limited or absent verbal output. The disorder presents a significant challenge for neuropsychology, speech-language pathology, and child neurology, as its causes lie deep within the complex neural networks that underlie speech perception, articulation, and symbolic thinking.

MATERIALS AND METHODS

The primary cause of alalia is organic damage to the brain's speech centers, which include Broca's area (in the posterior inferior frontal gyrus of the dominant hemisphere) and Wernicke's area (in the posterior superior temporal gyrus). These regions form part of a wider speech network connected through the arcuate fasciculus and other association fibers. Damage to these areas during the critical period of early brain development—typically before the age of three—disrupts the neural pathways responsible for speech encoding, comprehension, and motor articulation [2].

In motor alalia, lesions in Broca's area or adjacent premotor and precentral regions impair the programming and execution of articulatory movements, leading to severe difficulty in producing speech sounds. The child may understand speech but cannot express thoughts verbally. In contrast, sensory alalia results from damage to Wernicke's area, disrupting the perception and differentiation of phonemes. Such children hear but do not comprehend linguistic input, rendering them unable to form meaningful speech.

In many cases, mixed forms of alalia arise from diffuse brain injury involving both speech and associative areas. Modern neuroimaging studies using MRI and EEG reveal underdevelopment of

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cortical and subcortical speech circuits, reduced myelination, and abnormal hemispheric asymmetry in affected children.

RESULTS AND DISCUSSION

One of the most significant etiological factors in alalia is perinatal brain injury, encompassing adverse events during pregnancy, childbirth, or the neonatal period. Hypoxia—a deficiency of oxygen to the fetal brain—is a leading cause. Prolonged intrauterine hypoxia or asphyxia during delivery can damage the perisylvian cortex and basal ganglia, leading to functional immaturity of speech mechanisms. Premature birth, low birth weight, and neonatal jaundice with bilirubin encephalopathy are also associated with speech developmental delay.

Mechanical trauma during complicated labor, such as the use of forceps or vacuum extraction, can result in intracranial hemorrhage or ischemia. Postnatal infections affecting the central nervous system—such as meningitis, encephalitis, or severe viral illnesses—may further damage cortical structures critical for language processing. These conditions, if occurring during the sensitive period of speech development (ages 0–3), interfere with the establishment of neural circuits necessary for auditory-verbal integration [3].

Recent advances in molecular neurobiology have identified genetic contributions to speech disorders including alalia. Mutations in genes regulating cortical development, synaptic plasticity, and speech motor control—such as the FOXP2 gene—have been implicated in language impairments. FOXP2 encodes a transcription factor involved in the differentiation of neurons in Broca's and Wernicke's areas and the basal ganglia. Mutations in this gene disrupt the formation of neural circuits responsible for sequencing motor commands for speech.

Neurochemical abnormalities also play a role. Dysregulation of neurotransmitter systems—particularly dopamine, glutamate, and gamma-aminobutyric acid (GABA)—may impair synaptic transmission within speech centers. These alterations hinder the neural synchronization required for phonological processing and motor coordination of speech. Furthermore, delayed maturation of interhemispheric connections via the corpus callosum has been observed in children with developmental speech disorders, leading to poor integration between auditory perception and speech production.

While the underlying cause of alalia is neurological, environmental and psychosocial factors can exacerbate the disorder's severity or delay recovery. Children raised in linguistically deprived environments, where verbal stimulation and social interaction are limited, may fail to activate compensatory mechanisms in intact cortical areas. Emotional neglect, institutionalization, or chronic stress in early childhood can impair cognitive and linguistic motivation, compounding biological deficits [4].

Additionally, bilingual environments introduced prematurely or inconsistently may place excessive demands on a neurologically immature system, further slowing speech acquisition. Though these factors alone do not cause alalia, they can significantly influence its clinical manifestation and prognosis. Thus, the interplay between neurological damage and environmental deprivation defines the overall functional outcome in affected children.

CONCLUSION

Alalia represents one of the most complex speech impairments of childhood, arising from early organic damage to the cortical and subcortical structures of the brain responsible for speech perception and production. Its causes encompass a wide range of neurobiological and environmental

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factors, including perinatal injury, genetic mutations, neurochemical imbalances, and inadequate social-linguistic stimulation.

REFERENCES

1. Bishop, D. V. M. Developmental language impairment: nature and origins. Journal of Child Psychology and Psychiatry, 2016.
2. Leonard, L. B. Children with Specific Language Impairment. MIT Press, 2014.
3. Vargha-Khadem, F., et al. FOXP2 and the neuroanatomy of speech and language. Nature Reviews Neuroscience, 2015.
4. Paul, R., & Norbury, C. Language Disorders from Infancy through Adolescence. Elsevier, 2022