

Aphasia: A Comprehensive Language Disorder, Clinical Treatments and Clinical Contexts

Granthali Shape, Pooja Rasal*, Poonam Yadav

Department of Pharmacology, JES's SND College of Pharmacy, Babhulgaon, Yeola (Nashik)

ABSTRACT

Aphasia is the language disorder which mostly occurs in the left hemisphere of posterior and inferior frontal gyrus of Brodmann Area. It is basically the language impairment in which there is a loss of communication. Patient with this disorder are unable to communicate properly and they face a lots of problems while speaking. It can be worse while it can cure through various assessments and methods and mainly this disorder can be cured by communicating continuously. This mostly occurs after stroke which damages the part of language namely (Broca's And Wernicke's Area) which are responsible for language producing. Aphasiology is an important field of study, which it's studies included in many basic and clinical sciences such as speech-language pathology; psychology; neurology and linguistics. In this review we discuss about the neuroanatomy, and there clinical assessments methods through which aphasia can be controlled and also the speech therapy which remains unchanged. The method used to do transcranial is innovative repetitive transcranial magnetic stimulation and direct current stimulation, which are used to regulate cortical excitability by delivering cortical brain stimulation and are only now being investigated. In the diagnosis and comprehension of aphasia, neuroimaging is essential so here are some techniques which have often being used such as T1- weighted Magnetic Resonance Imaging offers excellent anatomical information of acute ischemic or hemorrhagic lesions, whereas structural imaging techniques like Axial Computed Tomography (CT) identify them, subcortical and cortical regions. Post-stroke alterations are better seen with T2-weighted MRI, which highlights white matter abnormalities and edema.

Keywords: Aphasia, Neuroanatomy, Neuroimaging, Clinical Assessment Methods, Neuroimaging, T1-Weighted MRI

INTRODUCTION

The neurological disorder known as "aphasia" (from the Greek ἀφασία, mutism) is defined as the loss of the capacity to comprehend, produce, and reproduce a language as a result of damage to the parts of the brain involved in its processing. Thus, aphasias do not include speech impairments brought on by intellectual deficiencies, basic sensory deficiencies, mental diseases, or musculoskeletal weakness [1-2]. Despite having a higher frequency of Wernicke's aphasia than men, some research indicates that women may have a reduced incidence of aphasia due to their greater bilaterality of language function [3]. In its therapeutic context, aphasia as a pathological state may have the potential to adversely impact other psychological illnesses, whether they are concurrent or preceding, or contribute to the emergence of new psychopathologies, which may exhibit more severe symptoms, as is the case with anxious states [4]. Age

is a common element in recovery; some studies indicate that stroke aphasia patients over seventy years of age have not recovered as well as younger patients; nonetheless, different degrees of recovery can occur at any age, even in distant ages from brain injury [5, 6]. Between 21 and 38 percent of acute strokes involve aphasia, which is linked to increased mortality, morbidity, and medical resource use [7]. It is clear that the most important elements are evaluation and treatment. A precise evaluation is necessary because it enables medical practitioners to identify the precise nature and extent of the ailment, which is critical for choosing the best course of action to promote healing. The majority of aphasia evaluations are conducted using language exams, which may not be sufficient [8]. as well as time-consuming. Innovative methods are desperately needed to enhance this procedure. Similar to this, in order to optimize the efficiency of intense language training in rehabilitation, it is frequently required to

Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



combine it with other techniques due to limitations in medical and financial resources [9]. In order to provide adjustments to compensate for these linguistic deficiencies, it was determined that these individuals needed to be discharged to more restrictive environments [10].

Types of Aphasia:

Based on the patient's linguistic skill and the affected region of the brain, there are several types of aphasia. Laborious, non-fluent speech with preserved understanding but poor repetition results from Broca's aphasia [11] Wernicke's aphasia results in fluent but senseless speech with little comprehension or

repetition [12]. The most serious kind of global aphasia greatly affects speech and understanding. Though speech stays fluid and comprehension is still rather strong, conduction aphasia mostly results in repetition [13]. Though repetition persists, transcorticomotor aphasia is similar to Broca's; transcortical sensory aphasia is akin to Wernicke's despite the fact that repetition continues. Mixed transcortical aphasia enables repetition despite its bad impact on fluency and understanding. Finally, though comprehension, fluency, and repetition remain unchanged, word-finding problems point to anomia aphasia [14] (Table 1).

Table 1: Different types of Aphasia

Types	Speech	Comprehension	Repetition	Main Feature
Broca's	Non-fluent	Good	Poor	Struggles to speak
Wernicke's	Fluent	Poor	Poor	Nonsense speech
Global	None/minimal	Poor	Poor	Severe loss of language
Anomia	Fluent	Good	Good	Trouble finding words
Conduction	Fluent	Good	Poor	Can't repeat
Transcortical Motor	Non-fluent	Good	Good	Like Broca's but can repeat
Transcortical Sensory	Fluent	Poor	Good	Like Wernicke's but can repeat
Mixed Transcortical	Non-fluent	Poor	Good	Like Global but can repeat

Etiology:

Aphasias can manifest as a range of brain lesions, such as acute (degenerative), chronic; progressive (chronic), intermittent (sporadic), long-lived (local), diffuse (dominant), and broad. [15]. Aphasia is always the result of acquired brain injury, in contrast to dysarthria, which is characterized by articulation problems. Illnesses that affect the muscles, neuromuscular junction, or Aphasia cannot be caused by the peripheral nervous system [16]. In line with the etiological profile, it is important to highlight that any brain disease that affects the language processing mechanisms and the dominant hemisphere can lead to aphasia. We'll discuss acquired aphasia in this section. The following are some of the most common factors that might result in either a temporary or permanent aphasic status [17-21].

a) The left anterior cerebral artery, posterior cerebral artery, or Sylvian artery territory are often affected by strokes or cerebral infarcts, which usually cause softening in these areas;

b) A brief ischemic episode

c) Hemorrhages in the brain, including minor thalamic hemorrhages, lobar hematomas, and big central nucleus hemorrhages;

d) Expansive processes (typically tumors) in the left, frontal, or temporal hemispheres, which primarily cause progressive aphasia;

e) A sign of deteriorating processes (brain atrophies) that result in progressive cognitive decline is speech difficulties.

f) Post-traumatic arterial thrombosis (internal carotid artery and Sylvian artery), intracranial hematoma, especially in the left temporal lobe, and brain contusions are examples of head injuries;

g) Infectious processes that cause encephalitis or a brain abscess;

h) A migraine attack with aura in its early stages (within the first half hour before the start of the migraine), or a partial or complicated epileptic seizure.

Neuroanatomy:

A fundamental concept in functional language neuroanatomy is that bio-encoding labor must be divided to achieve the necessary processing to understand the complex and varied data found in language and its context. According to one convincing model, there are two streams: a dorsal stream that maps sound onto motoric actions and articulation, and a ventral stream that maps sound onto meaning [22-25]. The linguistic mediation comparison additionally, a number of additional studies focused on non-verbal spatial processing. According to Tranel and Kemmerer (2004), a lesion subtraction analysis of individuals with focal brain injury revealed the presence of three structures: the left frontal operculum, supramarginal gyrus, and anatomically similar sites. Performance on tasks that measured production, understanding, and semantic analysis revealed a correlation between the underlying white matter and bad locative preposition knowledge. A study using fMRI to investigate neural correlates of locative constructions, such as those on the left, revealed that prepositions were present in both verbal and visual domains. Only the left supramarginal gyrus (BA 40) showed a noticeable increase in activation either at or to the right of [26]; the left frontal operculum [27] was not found to have participated. Studies using transcranial magnetic stimulation demonstrate that, contrary to what would be predicted if motor prediction is crucial, impairments in speech perception do not follow injury to the motor system. Another theory is that speech recognition is improved by ventral stream forward prediction [28]. The brain performs parallel processing to synthesize data via interconnected neural networks [29] and computes a transform between thought and an audio signal delivered across parallel, ascending channels of the auditory brain stem and cortex [30]. Studies of the neocortex, which reveal vertically aligned columns of neurons perpendicular to the cortex, provide evidence for this intricate neuronal circuitry [31]. According to new studies, the neurological processes involved in speech production are not limited to motor commands that regulate muscle movement, even if all speech a succession of muscular motions leads to creation. Before issuing muscular commands, the speaker must first mentally picture the sounds that compose the words. At this level, one can see that the term "snow"

rhymes with "blow" but not with "plow" without speaking these words aloud.

Broca's Neuroanatomy:

The most important component may have been found by the French physician and anatomist Pierre Paul Broca, who named the Broca (Broca's) area after he found a shared area in the brain of two of his patients who had speech impairments. Language depends on this area, which is situated in the dominant hemisphere's posterior inferior frontal gyrus in Brodmann areas 44 (pars opercularis) and 45 (pars triangularis) [32]. The back is connected to phonology, which is the sound of language the front section helps with semantics, or the meaning of words. Additionally, verbal repetition, gesture generation, sentence syntax and fluency, and the ability to interpret the behaviors of others all depend on the Broca region [33-35]. A dissociation between the region and Broca's aphasia is also observed during neurosurgical excision of the Broca's region. In [36] Chronic cases of Broca's aphasia need damage to a wider range of areas, including the anterior superior temporal gyrus (STG; BA22), supramarginal gyrus (BA40), dorsolateral prefrontal cortex (BA46; BA9), and underlying white matter in each of these areas [37].

Wernicke's Neuroanatomy:

Carl Wernicke, a German neurologist, discovered the Wernicke area in 1874. This region is one of two parts of the cerebral cortex that are also involved in sensory processing has been recognized as regulating speech. In the dominant hemisphere, the Wernicke region is situated in the posterior section of the superior temporal gyrus, or Brodmann area 22 [38]. Wernicke region injury causes fluent but receptive aphasia. This area is in charge of understanding spoken and written language. [39] According to more recent imaging research, the Wernicke area may really be located in a larger region of the temporal lobe. However, this is not always the case, as it has long been understood that receptive aphasia is caused by damage to the Wernicke region. Isolated damage to the Wernicke region, which spares the white matter, may not result in severe receptive aphasia since some people use the right hemisphere for language [40]. Wernicke and Lichtheim created the standard model of aphasia in

the 19th century, and Geschwind improved it neuroanatomically in the 1960s [41,42]. The basis for comprehending the clinical characteristics of aphasia and the associated neuroanatomical abnormalities is provided by this model. Typically, the peri-Sylvian area of the dominant hemisphere which is often the left contains the brain's language centers. Following its reception by the main auditory cortices in the Heschl gyrus (transverse temporal gyrus), spoken language is processed in the posterior superior Wernicke region.

Pathophysiology:

Aphasia is caused by lesions in the brain's language-related areas, which are typically located in the dominant hemisphere (usually the left hemisphere for the majority of individuals) [43]. The Wernicke and Broca areas are significant sites along the arcuate fasciculus. Nonfluent, labored aphasia is caused by impairment to the dorsal stream, primarily in the frontoparietal regions, according to the contemporary dual-stream neuroanatomic model of aphasia. However, damage to the temporal region's ventral stream results in fluent aphasia accompanied by comprehension difficulties. Severe ischemic stroke is the most common cause of aphasia, while the vascular area most frequently affected is the left main MCA. When the entire dominant MCA area is affected, the end result is typically aphasia, which affects the entire world. In nonfluent Broca aphasia, also known as dorsal stream aphasia, the anterior superior branch of the MCA is frequently occluded, which is characterized by a the majority of challenges in language production cause fluent Wernicke aphasia, also known as ventral stream aphasia, while understanding is unaffected. This illness is related to substantial comprehension deficits brought about by the obstruction of the posteroinferior branch. Patients who have this ailment frequently have trouble

reading, writing, and repeating [44]. A decrease in blood supply to the Broca area is the most frequent cause of damage, with strokes being the worst instance. A prime example of this is the fact that both ischemic and hemorrhagic strokes in the area of the middle cerebral artery can injure the Broca area. Due to its size and proximity to the inner carotid artery, the middle cerebral artery is the blood vessel that is most often affected by strokes. As a result, many individuals who have had such cerebrovascular events experience Broca aphasia [45,46]. Wernicke's area can be found in the posterior portion of the superior temporal gyrus (Brodmann's area 22) of the dominant cerebral hemisphere. It has a close connection to the auditory cortex. to this area. Language function is concentrated in the left cerebral hemisphere for almost all right-handed individuals and 60% of left-handed people [47,48]. Severe hypotension or cardiac arrest can result in watershed infarcts, which in turn can cause transcortical aphasia. A watershed infarction between the anterior cerebral artery and the MCAs can occur when the dominant internal carotid artery is occasionally blocked, leading to transcortical motor aphasia. The transcortical sensory aphasia is caused by a watershed infarction between the posterior and dominant middle cerebral infarcts [49]. Furthermore, aphasia can occasionally be caused by injury to subcortical regions located deep inside the left hemisphere, such as the thalamus, caudate nucleus, and internal and external capsules [50]. Neurodegenerative diseases like Alzheimer's disease, frontotemporal dementia, and TBI can result in aphasia. Aphasia, the main sign of primary progressive aphasia, is a type of frontotemporal dementia brought on by the progressive loss and death of nerve cells in the brain's language centers. Brain tumor Mass has an effect on language areas, and infections are two more things that can harm them [51].

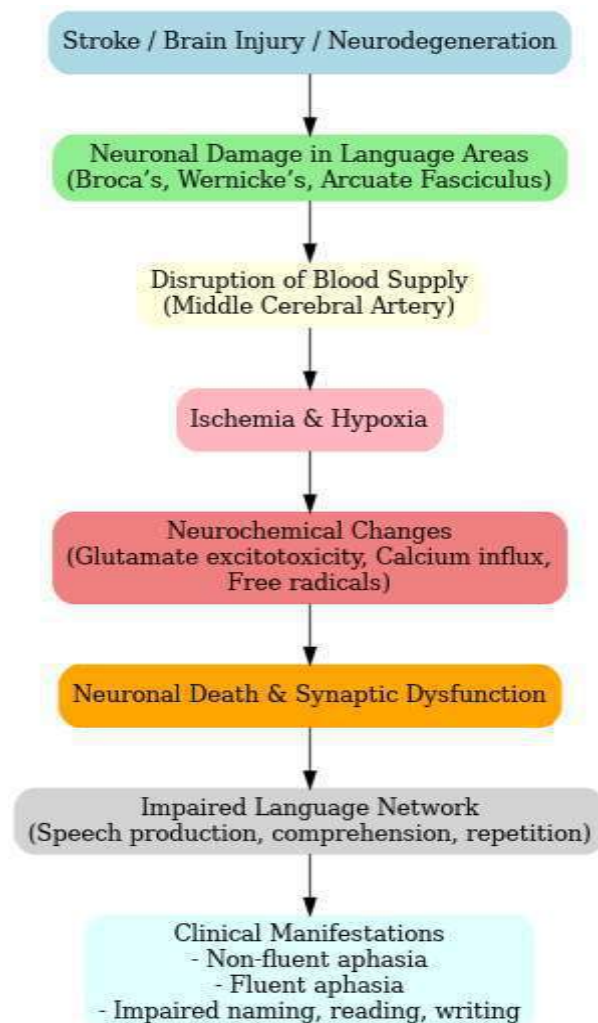


Figure 1: Pathophysiology of Aphasia

Clinical Assessments:

The majority of left hemisphere (LH) stroke survivors continue to experience persistent aphasia [52] and never regain their full language skills. Instead of identifying traditional aphasia syndromes the primary aim of modern assessments of aphasia is to identify issues in specific language functions [53], like those that occur with Broca's aphasia. Evaluating damage to distinct language components can assist healthcare providers in tailoring rehabilitation strategies for individual patients, potentially resulting in improved outcomes. Nonetheless, correctly identifying a patient's weaknesses necessitates a comprehensive grasp of how commonly used language tests relate to the separable core processes they evaluate. Moreover, examining the relationships between the location of lesions and distinct language functions, instead of focusing solely on specific test scores, could offer a more thorough understanding of how the brain processes language. Factor analysis is a technique for

simplifying data that helps uncover the essential variables within a dataset, allowing for the conversion of numerous behavioral scores into a more manageable number of variables. Only a few "latent variables" that provide the best explanation for the variability in the data [54]. The scientific literature is unequivocal that there are multiple aphasia assessment tools, which often produce differences and make it difficult to compare results of studies [55]. These additional techniques, such as virtual reality (VR) and transcranial direct current stimulation (tDCS) [56], may considerably improve the outcomes of treatment. Aphasia tests employ aphasia scales rather frequently. Two examples of such scales are the Boston Diagnostic Aphasia Examination (BDAE) [57] and the Western Aphasia Battery (WAB) [58]. These steps aim mostly at assessing fundamental linguistic skills including spontaneous speech, naming, writing, and auditory understanding [59]. Diagnosing aphasia presents difficulties for doctors in knowing which nonlinguistic abilities to assess. For

example, there is debate over which cognitive processes should be evaluated to establish a person with aphasia's decision-making capacity [60].

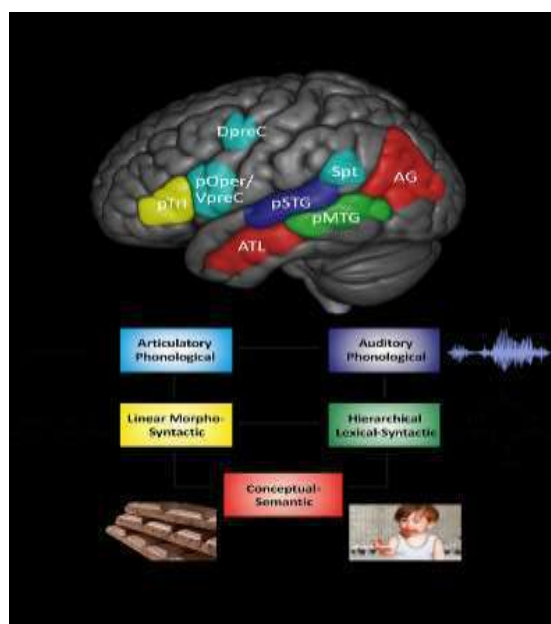


Figure 2: Dual Stream Model for Aphasia

The behavioral results of the study indicated a favorable association between AQ and the sum of the scores from the three cognitive assessment methods, therefore implying that language The various forms of cognitive evaluation were affected by the impairment [61]. The findings point toward more cognitive and language impairments in NFA stroke patients. Consequently, great caution should be used while choosing a cognitive assessment for these people. First, we disassembled the scales' constituent parts. The remaining exam questions in the MoCA rely on verbal expression abilities in addition to visual space and executive function. MOCA has thirty items grouped into twenty-four categories. The MMSE, though, has a possible total of 25 points. For both, oral expression is essential. One could finish the NLCA assessment procedure using audio-visual perception without talking. Still, earlier behavioral studies have found a connection between nonverbal CI [62] and a deficit in understanding. MoCA can be utilized to evaluate cognitive impairment. in stroke patients without aphasia, as previous research has shown in Figure 2.

Treatments:

People with aphasia less functional outcomes, fewer social circles, and fewer excursions to their home or

place of employment [64-67]. Aphasia affects financial, emotional, and physical life greatly. Speech-language pathology therapy forms the basis of treatment for aphasia [68]. Two choices are behavior therapy, compensatory behavior modification, and restorative therapy. The treatment for patients with Wernicke's aphasia may center on speech processing in order to support their ability to understand terms or phrases. The treatment for Broca's aphasia might focus on the word or sentence level of sound motor speech production [69] . It could be difficult for people with aphasia to communicate their needs and desires. Many people are aware of their shortcomings and illnesses, which can lead to annoyance, hopelessness, and a reluctance to seek assistance. Early diagnosis of depression is hence necessary to successfully treat persons suffering from aphasia. Having spiritual leaders, friends, and family emotional support is critical. Referrals to a psychiatrist, neuropsychologist, or psychologist may be necessary for assessment and treatment. Pharmacological medication is a common component of depression treatment [70] . The visualization of activation in the language regions of the brain is now possible thanks to advancements in neuroimaging technology, which helps advance the study and treatment of aphasia [71,72] . Crucially, the results of recent studies on aphasia therapy highlight that short

bursts of intensive treatment are more beneficial than an equivalent number of sessions over longer time frames [73]. Authentic user (patient) involvement, the development of engaging experiences, user control, and responsibility are all components of the collaborative process that constitutes the patient-centered model of therapy [74]. Functional performance, like communicating, may be facilitated by practicing a language exercise that focuses on a certain competence, like confronting naming. communicative intents, due to the brain's adaptive property. One example of a patient using the Life Participation Approach to Aphasia is paradigm of centered therapy [75]. By removing obstacles to communication in the environment, enhancing communication success through any modality (gestures, drawings, pointing, etc.), and providing caregiver training to improve communication, functional communication approaches place an emphasis on assisting the individual in communicating in everyday situations [76].

Neuroimaging:

It has been a little over ten years since neuroplasticity was proven to exist in the adult brain [77], offering neurophysiological support for behavior. findings that even in the chronic stage, people with aphasia frequently display significant improvement after brain injury to the left perisylvian regions [78] Concerning the brain's operations and regions.

Two major patterns have been identified that aid in this rehabilitation:

1) Linguistic function, the affected left hemisphere previously served as the premorbid foundation, which is now transferred to right hemisphere regions.

2) Neural and similar to areas of the left hemisphere that are in charge of the linguistic impairments. The left hemisphere's tissue is added to the functional map. Areas around the wound. This tissue has the potential to take over processes that are usually under the control of possibly because of functional redundancy, the damaged areas. These tendencies may be questioned to the extent that right and left are interchangeable prior to brain injury, the hemisphere areas that were proven to be active were involved in language processing. Neuroimaging research of language processing with healthy volunteers display extra sylvian recruitment as well as right hemisphere activation throughout language areas [79,80]. These findings imply that the right hemisphere activation observed in the extensive neural network that might be partially represented in studies involving aphasic patients is the subject of these investigations. Under typical circumstances, it serves language. Thompson et al. reported in their recent study [81] that verb argument structure processing in three out of five participants showed right posterior perisylvian activation patients suffering from Broca's aphasia brought on by lesions in this area of the hemisphere on the left However, the aphasic people showed bilateral activation in the same location as 12 healthy control patients who were age-matched with them. Therefore, the activation observed in aphasic people may indicate a premorbidly accessible network in the right hemisphere.

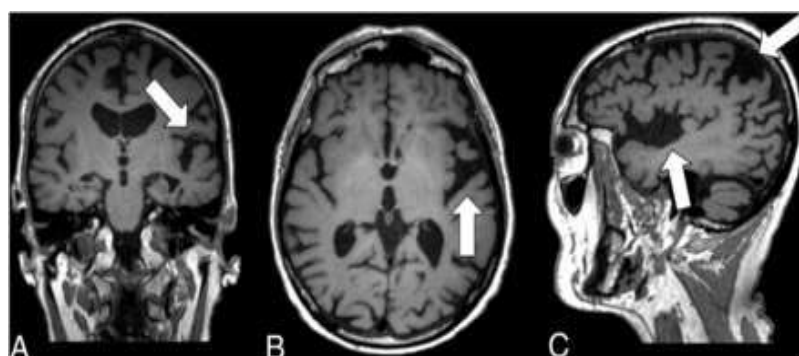


Figure 3: T1-weighted magnetic resonance imaging was performed on a right-handed patient with repetition problems using coronal (A), axial(B) and sagittal(C) demonstrate asymmetric widening of the left Sylvian fissure with left posterior peri-Sylvian and temporoparietal atrophy, which is suggestive of lvPPA (white arrows, A-C).

The discovery made by Thompson et al. in a recent study was significant. atrophy. Complex MR imaging techniques such as DTI and voxel-based morphometry analysis can be used to show focal GM atrophy (Figure 3) and WM alterations. Respectively [82]. Specifically, the FDA-approved NeuroQuant is an example of a cortical volumetric program. In a study examining the usefulness of MR imaging in distinguishing PPA variants, MR imaging showed great specificity for the characteristic atrophy patterns of highlighting poor sensitivity for both (43% for lvPPA; 21% for nvfPPA) for lvPPA (95%) and nvfPPA (91%) [83]. The possibility of having lvPPA is not excluded by the presence of atrophy in the left posterior peri-Sylvian or temporal region, which is an effective indicator of both but not necessarily an exclusion from the diagnosis (the condition may be excluded by radiological analysis, it could be termed "anti" or "examined"). In a prospective trial

examining 130 patients with neurodegenerative aphasia, 52 of whom had lvPPA, GM loss was found in those with lvPPA, especially on the left and largest in the posterior temporal lobe, which extends to the frontal and parietal lobes [84]. Fractional anisotropy and mean diffusivity analyses within this cohort the bilateral superior and inferior longitudinal fasciculi as well as the inferior occipitofrontal fasciculus. The bilateral WM involvement was found to be left greater than right, with the posterior left temporal WM showing the most activity and extending into the anterior temporal, frontal, parietal, and occipital WM [85]. It has been shown that the inferior parietal and posterior superior temporal cortices. Therefore, these regions and WM tract involvement in the superior and inferior longitudinal fasciculi is most likely what gives them their function in the phonological loop the poor repetition, naming, and comprehension observed in individuals with lvPPA [86,87].

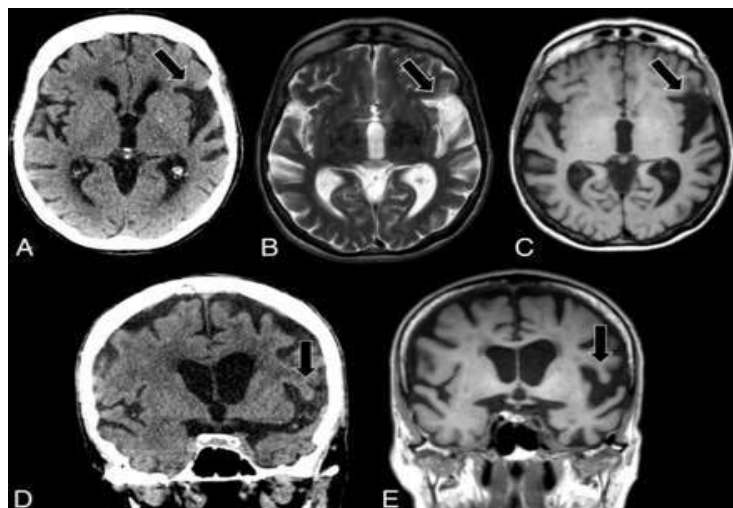


Figure 4: Include both axial CT (A) and axon T2-weighted MR imaging (B), as well as axite T1-weighting MRI imaging(C), coronal CT laterally (D) or coronetly T1, the weight of mri imaging. A right-handed person with speech apraxia shows asymmetrical enlargement of the left Sylvian fissure and prominent atrophy of the left posterior frontoinsular region (black arrows, A–E), suggesting nvfPPA.

In Figure 4, the presence of atrophy in the left posterior peri-Sylvian or temporoparietal region is a strong indication of lvPPA, it should not be disregarded as an undetermined condition. WM injury in the dorsal pathway (superior longitudinal fasciculus) [88] and nvfVPA, which is not covered in other PPA subtypes [89,90]. In addition, GM atrophy has been observed. The striatum, supplementary motor region, and premotor regions have all been characterized in addition to these [91,92]. In patients

with nvfPPA, structural deficiencies have been linked to impaired syntactic processing. and problems involving the posterior region of the inferior frontal gyrus's function [93]. Although the majority of cases involve the left hemisphere, right-hemisphere involvement has also been documented, this is thought to happen in left-handed people or those who have had developmental learning disorders, like dyslexia [94]. The existence of atrophy in the left posterior peri-Sylvian or temporal region is an effective indication of both but not necessarily

exclusion from the diagnosis (the condition may be excluded by radiological analysis, it could be termed "anti" or "examined"), and thus does not exclude the possibility of having lvPPA [95]. Additionally, atrophy often spreads via the frontal aslant tract, which is involved in the supplementary motor complex, from the frontal operculum. the start and execution of movements, especially articulation, and eventually to the basal ganglia and supramarginal gyrus [96]. The frontal aslant tract's integrity is compromised by the lack of integrity. in nvPPA, patients exhibit verbal fluency task performance and distortion mistakes in spontaneous speech. Consequently, nvPPA serves as an illustration. of a network malfunction affecting the circuit of areas and connections involved in speech production [96].

FUTURE SCOPE:

The intersection of current technology, individualized treatments, and fresh biomedical approaches will help to define aphasia research and treatment. Early diagnosis and careful mapping of the brain networks engaged in language are now feasible thanks to new neuroimaging techniques including fMRI and diffusion tensor imaging. Supported by digital platforms, mobile applications, and artificial intelligence, personalized rehabilitation looks to be able to provide continuous and patient-specific therapy. Neurostimulation techniques being studied to increase brain plasticity and transcranial direct current (tDCS) and transcranial magnetic stimulation (TMS) are among the therapeutic options for recovery outcomes inducing stimulation (tDCS). Hope for repairing damaged neuronal circuits comes from new pharmacological developments, stem cell therapies, and regenerative medicine. Finding biomarkers and potential therapeutic targets using molecular and genetic research is becoming more and more crucial. Thanks to the integration of holistic approaches like mindfulness, music therapy, and caregiver training, the direction of aphasia treatment is moving toward more efficient, reasonably priced, patient-centered care.

CONCLUSION:

Practice is influenced by science and theory. The treatment of aphasia must take into account advancements in neuroimaging, the creation of novel

ideas about how language works, and shifts in the norms of clinical practice. The capacity to modify and adjust clinical treatment is the hallmark of a wise clinician, and it is necessary to guarantee that people with aphasia receive outstanding rehabilitation to maximize their recovery. This disease is identified by the presence of a distinctive feature the left posterior peri-Sylvian or temporoparietal region might atrophy, but this does not always indicate that the lvPPA's presence. Recent advancements in our understanding of its neuroanatomy, varieties, causes, and pathophysiology have highlighted the important functions of both cortical and subcortical systems. as the value of timely and precise clinical evaluations. While conventional speech-language therapy remains the mainstay of rehabilitation, factors such as age, the severity of a stroke, and where exactly one occurred are all important in making up the outcome. the available resources and the lesion. Neuroimaging has provided us with crucial direction in creating customized treatment strategies as well as helping us better understand the connection between the brain and language. The integration of cutting-edge technologies like functional imaging, non-invasive neurostimulation, AI-based evaluation methods, and patient-centered treatment paradigms gives fresh optimism for enhancing rehabilitation in the future. The range of treatment options is expanded by pharmacological methods, regenerative medicine, and holistic therapies such mindfulness and music therapy. The best therapy for aphasia sufferers is ultimately achieved by combining cutting-edge scientific advancements with flexible, individualized clinical practice, which leads to improved outcomes. which is practical in addition to a higher overall quality of life.

REFERENCE

1. Angelini C, Battistin L. *Neurologia clinica*. Bologna: Società Editrice Esculapio; 2019.
2. Perrotta G. *Psicologia clinica*. 1st ed. Luxco Edizioni; 2019.
3. Kandel ER, Schwartz JH, Jessell TM. *Principi di Neuroscienze*. 5th ed. Casa Editrice Ambrosiana; 2017.
4. Perrotta G. Anxiety disorders: definitions, contexts, neural correlates and strategic therapy. *J Neurol Neurosci*. 2019; 6:046.

5. Ellis C, Urban S. Age and aphasia: a review of presence, type, recovery and clinical outcomes. *Top Stroke Rehabil.* 2016;23(6):430–9.
6. Croteau C, McMahon-Morin P, Le Dorze G, Baril G. Impact of aphasia on communication in couples. *Int J Lang Commun Disord.* 2020;55(4):547–57.
7. Hickok G, Poeppel D. The cortical organization of speech processing. *Nat Rev Neurosci.* 2007;8(5):393–402.
8. Rohde A, Worrall L, Godecke E, O'Halloran R, Farrell A, Massey M. Diagnosis of aphasia in stroke populations: a systematic review of language tests. *PLoS One.* 2018;13(3): e0194143.
9. Fridriksson J, Hillis AE. Current approaches to the treatment of post-stroke aphasia. *J Stroke.* 2021;23(2):183–201.
10. Gonzalez-Fernandez M, Christian AB, Davis C, Hillis AE. Role of aphasia in discharge location after stroke. *Arch Phys Med Rehabil.* 2013;94(5):851–5.
11. Friedrich P, Anderson C, Schmitz J, Schlüter C, Lor S, Stacho M, et al. Fundamental or forgotten? Is Pierre Paul Broca still relevant in modern neuroscience? *Laterality.* 2019;24(2):125–38.
12. Ochfeld E, Newhart M, Molitoris J, Leigh R, Cloutman L, Davis C, et al. Ischemia in Broca area is associated with Broca aphasia more reliably in acute than in chronic stroke. *Stroke.* 2010;41(2):325–30.
13. Chang EF, Raygor KP, Berger MS. Contemporary model of language organization: an overview for neurosurgeons. *J Neurosurg.* 2015;122(2):250–61.
14. Yourganov G, Smith KG, Fridriksson J, Rorden C. Predicting aphasia type from brain damage measured with structural MRI. *Cortex.* 2015; 73:203–15.
15. Berthier ML, Starkstein SE, Leiguarda R, et al. Transcortical aphasia: importance of the nonspeech dominant hemisphere in language repetition. *Brain.* 1991;114(3):1409–27.
16. Berthier ML. Poststroke aphasia: epidemiology, pathophysiology and treatment. *Drugs Aging.* 2005;22(2):163–82.
17. Radakovic R, Colville S, Cranley D, Starr JM, Pal S, et al. Multidimensional apathy in behavioral variant frontotemporal dementia, primary progressive aphasia, and Alzheimer disease. *J Geriatr Psychiatry Neurol.* 2020;33(4):231–41.
18. Funakoshi Y, Imamura H, Tokunaga S, Murakami Y, Tani S, et al. Carotid artery stenting before surgery for carotid artery occlusion associated with acute type A aortic dissection: two case reports. *Interv Neuroradiol.* 2020;26(4):502–8.
19. National Aphasia Association. Aphasia FAQ's. 2017 Nov 7. Available from: <https://www.aphasia.org/aphasia-faqs>
20. National Institute on Deafness and Other Communication Disorders (NIDCD). Aphasia.
21. Rauschecker JP, Scott SC. Maps and streams in the auditory cortex: nonhuman primates illuminate human speech processing. *Nat Neurosci.* 2009;12(6):718–24.
22. Weiller C, Bormann T, Saur D, Musso M, Rijntjes M. How the ventral pathway got lost: and what its recovery might mean. *Brain Lang.* 2011;118(1):29–39.
23. Noordzij ML, et al. Neural correlates of locative prepositions. *Neuropsychologia.* 2008;46(7):2080–9.
24. Amorapanth P, et al. The neural basis for spatial relations. *J Cogn Neurosci.* 2010;22(8):1739–53.
25. Hickok G. The cortical organization of speech processing: feedback control and predictive coding in the context of a dual stream model. *J Commun Disord.* 2012;45(6):393–402.
26. Aboitiz F, Garcia VR. The evolutionary origin of language areas in the human brain: a neuroanatomical perspective. *Brain Res Rev.* 1997;25(3):381–96.
27. Dennett DC. *Consciousness explained.* Boston: Little Brown; 1991.
28. Mountcastle VB. An organizing principle of cerebral function: the unit module and the distributed system. In: Edelman GM, Mountcastle VB, editors. *The mindful brain.* Cambridge, MA: MIT Press; 1978. p. 7–50.
29. Keller SS, Crow T, Foundas A, Amunts K, Roberts N. Broca's area: nomenclature, anatomy, typology and asymmetry. *Brain Lang.* 2009;109(1):29–48.
30. Skipper JI, Goldin-Meadow S, Nusbaum HC, Small SL. Speech-associated gestures, Broca's area, and the human mirror system. *Brain Lang.* 2007;101(3):260–77.

31. Flinker A, Korzeniewska A, Shestyuk AY, Franaszczuk PJ, Dronkers NF, Knight RT, et al. Redefining the role of Broca's area in speech. *Proc Natl Acad Sci U S A*. 2015;112(9):2871–5.
32. Brown S, Yuan Y. Broca's area is jointly activated during speech and gesture production. *Neuroreport*. 2018;29(14):1214–6.
33. Andrews JP, Cahn N, Speidel BA, Chung JE, Levy DF, Wilson SM, et al. Dissociation of Broca's area from Broca's aphasia in patients undergoing neurosurgical resections. *J Neurosurg*. 2023;138(3):847–57.
34. Dronkers NF, Baldo JV. Language: aphasia. *Encycl Neurosci*. 2009; 5:499–507.
35. Paulraj S, Schendel K, Curran B, Dronkers N, Baldo J. Role of the left hemisphere in visuospatial working memory. *J Neurolinguist*. 2018; 48:133–41.
36. Baldo JV, Dronkers NF, Wilkins D, Ludy C, Raskin P, Kim J. Is problem solving dependent on language? *Brain Lang*. 2005;92(3):240–50.
37. Binder JR. The Wernicke area: modern evidence and a reinterpretation. *Neurology*. 2015;85(24):2170–5.
38. Yourganov G, Smith KG, Fridriksson J, Rorden C. Predicting aphasia type from brain damage measured with structural MRI. *Cortex*. 2015; 73:203–15.
39. Nasios G, Dardiotis E, Messinis L. From Broca and Wernicke to the neuromodulation era: insights of brain language networks for neurorehabilitation. *Behav Neurol*. 2019; 2019:9894571.
40. Lahiri D, Dubey S, Sawale VM, Das G, Ray BK, Chatterjee S, Ardila A. Incidence and symptomatology of vascular crossed aphasia in Bengali. *Cogn Behav Neurol*. 2019;32(4):256–67.
41. Kuljic-Obradovic DC. Subcortical aphasia: three different language disorder syndromes? *Eur J Neurol*. 2003;10(4):445–8.
42. Fridriksson J, Bonilha L, Rorden C. Severe Broca's aphasia without Broca's area damage. *Behav Neurol*. 2007;18(4):237–8.
43. Levine RL, Dulli DA, Dixit S, Hafeez F, Khasru M. Isolated Broca's area aphasia and ischemic stroke mechanism. *J Stroke Cerebrovasc Dis*. 2003;12(3):127–31.
44. Cattaneo L. Language. *Handb Clin Neurol*. 2013; 116:681–91.
45. Jiménez de la Peña MM, Gómez Vicente L, García Cobos R, Martínez de Vega V. Neuroradiologic correlation with aphasia: cortico-subcortical map of language. *Radiologia (Engl Ed)*. 2018;60(3):250–61.
46. Fridriksson J, den Ouden DB, Hillis AE, Hickok G, Rorden C, Basilakos A, et al. Anatomy of aphasia revisited. *Brain*. 2018;141(3):848–62.
47. Kuljic-Obradovic DC. Subcortical aphasia: three different language disorder syndromes? *Eur J Neurol*. 2003;10(4):445–8.
48. Deng X, Zhang Y, Xu L, Wang B, Wang S, Wu J, et al. Comparison of language cortex reorganization patterns between cerebral arteriovenous malformations and gliomas: a functional MRI study. *J Neurosurg*. 2015;122(5):996–1003.
49. Jianu DC, Jianu SN, Petrica L, Dan TF, Munteanu G. Vascular aphasia. In: Sanchette P, editor. *Ischemic Stroke*. London: IntechOpen; 2021. p. 37–59.
50. Pulvermüller F, Berthier ML. Aphasia therapy on a neuroscience basis. *Aphasiology*. 2008;22(6):563–99.
51. Laska AC, Martensson B, Kahan T, von Arbin M, Murray V. Recognition of depression in aphasic stroke patients. *Cerebrovasc Dis*. 2007;24(1):74–9.
52. Fama ME, Turkeltaub PE. Treatment of poststroke aphasia: current practice and new directions. *Semin Neurol*. 2014;34(5):504–13.
53. Santos NC, Costa PS, Amorim L, et al. Exploring the factor structure of neurocognitive measures in older individuals. *PLoS One*. 2015;10(4):e0124229.
54. Kwakkel G, Lannin NA, Borschmann K, English C, Ali M, Churilov L, et al. Standardized measurement of sensorimotor recovery in stroke trials: consensus-based core recommendations from the Stroke Recovery and Rehabilitation Roundtable. *Int J Stroke*. 2017;12(5):451–61.
55. Picano C, Quadrini A, Pisano F, Marangolo P. Innovative approaches to aphasia rehabilitation: a review on efficacy, safety, and controversies. *Brain Sci*. 2020;11(1):41.

56. Kertesz A. Aphasia and associated disorders: taxonomy, localization and recovery. New York: Grune & Stratton; 1979.
57. Goodglass H, Kaplan E. The assessment of aphasia and related disorders. Philadelphia: Lea & Febiger; 1972.
58. Wertz RT, Deal JL, Robinson AJ. Classifying the aphasias: a comparison of the Boston Diagnostic Aphasia Examination and the Western Aphasia Battery. In: Clinical Aphasiology: Proceedings of the Conference. BRK Publishers; 1984. p. 40–7.
59. Kim ES, Suleman S, Hopper T. Decision making by people with aphasia: a comparison of linguistic and nonlinguistic measures. *J Speech Lang Hear Res.* 2020;63(6):1845–60.
60. Bonini MV, Radanovic M. Cognitive deficits in post-stroke aphasia. *Arq Neuropsiquiatr.* 2015; 73:840–847. doi:10.1590/0004282X20150133
61. Caplan D, Michaud J, Hufford R. Short-term memory, working memory, and syntactic comprehension in aphasia. *Cogn Neuropsychol.* 2013; 30:77–109. doi:10.1080/02643294.2013.803958
62. Dong Y, Sharma VK, Chan BP, Venketasubramanian N, Teoh HL, Seet RC, et al. The Montreal Cognitive Assessment (MoCA) is superior to the Mini-Mental State Examination (MMSE) for the detection of vascular cognitive impairment after acute stroke. *J Neurol Sci.* 2010; 299:15–18. doi: 10.1016/j.jns.2010.08.051
63. Black-Schaffer RM, Osberg JS. Return to work after stroke: development of a predictive model. *Arch Phys Med Rehabil.* 1990; 71:285–290.
64. Lazar RM, Minzer B, Antoniello D, Festa JR, Krakauer JW, Marshall RS. Improvement in aphasia scores after stroke is well predicted by initial severity. *Stroke.* 2010;41(7):1485–1488.
65. Tippet DC, Niparko JK, Hillis AE. Aphasia: current concepts in theory and practice. *J Neurol Transl Neurosci.* 2014;2(1):1042.
66. Allida S, Cox KL, Hsieh CF, House A, Hackett ML. Pharmacological, psychological and non-invasive brain stimulation interventions for preventing depression after stroke. *Cochrane Database Syst Rev.* 2020;5:CD003689.
67. Pulvermüller F, Berthier ML. Aphasia therapy on a neuroscience basis. *Aphasiology.* 2008;22(6):563–599.
68. San Lee J, Yoo S, Park S, Kim HJ, Park KC, Seong JK, et al. Differences in neuroimaging features of early- versus late-onset nonfluent/agrammatic primary progressive aphasia. *Neurobiol Aging.* 2020; 86:92–101.
69. Bhogal SK, Teasell R, Speechley M. Intensity of aphasia therapy: impact on recovery. *Stroke.* 2003; 34:987–993. doi: 10.1161/01.STR.0000062343.64383.D0
70. Byng S, Cairns D, Duchan J. Values in practice and practising values. *J Commun Disord.* 2002;35(2):89–106.
71. Chapey R, Duchan RJ, Garcia LJ, Kagan A, Lyon JG, Simmons-Mackie N. Life participation approach to aphasia: a statement of values for the future. In: Chapey R, editor. *Language Intervention Strategies in Aphasia and Related Neurogenic Communication Disorders.* 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2001. p. 279–289.
72. Martin N, Thompson CK, Worrall L. Aphasia Rehabilitation: The Impairment and Its Consequences. San Diego: Plural Publishing; 2007.
73. Hickok G, Poeppel D. Dorsal and ventral streams: a framework for understanding aspects of the functional anatomy of language. *Cognition.* 2004; 92:67–99.
74. Hickok G, Poeppel D. The cortical organization of speech processing. *Nat Rev Neurosci.* 2007; 8:393–402.
75. Merzenich M, Wright B, Jenkins W, et al. Cortical plasticity underlying perceptual, motor, and cognitive skill development: implications for neurorehabilitation. *Cold Spring Harb Symp Quant Biol.* 1996; 61:1–8.
76. Holland A, Fromm D, DeRuyter F, Stein M. The efficacy of treatment of aphasia: a brief synopsis. *J Speech Hear Res.* 1996; 39:525–534. doi:10.1044/jshr.3905.s27
77. Thompson CK. Plasticity of language networks. In: Baudry M, Bi X, Schrieber SS, editors. *Synaptic Plasticity: Basic Mechanisms to Clinical Applications.* New York: Marcel Dekker; 2005. p. 255–270.
78. Crosson B, McGregor K, Gopinath KS, et al. Functional MRI of language in aphasia: a review of the literature and the methodological

- challenges. *Neuropsychol Rev.* 2007; 17:157–177.
79. Thompson CK, den Ouden DB, Bonakdarpour B, et al. Neural correlates of treatment-induced recovery of sentence processing and production in agrammatism. *Brain Lang.* (in press).
 80. Josephs KA, Martin PR, Botha H, et al. [18F] AV-1451 tau-PET and primary progressive aphasia. *Ann Neurol.* 2018; 83:599–611. doi:10.1002/ana.25183
 81. Mueller A, Bullich S, Barret O, et al. Tau PET imaging with 18F-PI-2620 in patients with Alzheimer disease and healthy controls: a first-in-humans study. *J Nucl Med.* 2020; 61:911–919. doi:10.2967/jnumed.119.236224
 82. Xia C, Makaretz SJ, Caso C, et al. Association of in vivo [18F] AV-1451 tau PET imaging results with cortical atrophy and symptoms in typical and atypical Alzheimer disease. *JAMA Neurol.* 2017; 74:427–436. doi:10.1001/jamaneurol.2016.5755
 83. Josephs KA, Martin PR, Botha H, et al. [18F] AV-1451 tau-PET and primary progressive aphasia. *Ann Neurol.* 2018; 83:599–611. doi:10.1002/ana.25183
 84. Xia C, Makaretz SJ, Caso C, et al. Association of in vivo [18F] AV-1451 tau PET imaging results with cortical atrophy and symptoms in typical and atypical Alzheimer disease. *JAMA Neurol.* 2017; 74:427–436. doi:10.1001/jamaneurol.2016.5755
 85. Werry EL, Bright FM, Piguet O, et al. Recent developments in TSPO PET imaging as a biomarker of neuroinflammation in neurodegenerative disorders. *Int J Mol Sci.* 2019; 20:121. doi:10.3390/ijms20133161
 86. Mandelli ML, Caverzasi E, Binney RJ, et al. Frontal white matter tracts sustaining speech production in primary progressive aphasia. *J Neurosci.* 2014; 34:9754–9767. doi:10.1523/JNEUROSCI.3464-13.2014
 87. Wilson SM, Galantucci S, Tartaglia MC, et al. Syntactic processing depends on dorsal language tracts. *Neuron.* 2011; 72:397–403. doi: 10.1016/j.neuron.2011.09.014
 88. Grossman M. The non-fluent/agrammatic variant of primary progressive aphasia. *Lancet Neurol.* 2012; 11:545–555. doi:10.1016/S1474-4422(12)70099-6
 89. Gorno-Tempini ML, Dronkers NF, Rankin KP, et al. Cognition and anatomy in three variants of primary progressive aphasia. *Ann Neurol.* 2004; 55:335–346. doi:10.1002/ana.10825
 90. Mandelli ML, Vilaplana E, Brown JA, et al. Healthy brain connectivity predicts atrophy progression in non-fluent variant of primary progressive aphasia. *Brain.* 2016; 139:2778–2791. doi:10.1093/brain/aww195
 91. Wilson SM, Dronkers NF, Ogar JM, et al. Neural correlates of syntactic processing in the nonfluent variant of primary progressive aphasia. *J Neurosci.* 2010; 30:16845–16854. doi:10.1523/JNEUROSCI.2547-10.2010
 92. Miller ZA, Mandelli ML, Rankin KP, et al. Handedness and language learning disability differentially distribute in progressive aphasia variants. *Brain.* 2013; 136:3461–3473. doi:10.1093/brain/awt242
 93. Mandelli ML, Vilaplana E, Brown JA, et al. Healthy brain connectivity predicts atrophy progression in non-fluent variant of primary progressive aphasia. *Brain.* 2016; 139:2778–2791. doi:10.1093/brain/aww195
 94. Mandelli ML, Caverzasi E, Binney RJ, et al. Frontal white matter tracts sustaining speech production in primary progressive aphasia. *J Neurosci.* 2014; 34:9754–9767. doi:10.1523/JNEUROSCI.3464-13.2014
 95. Dick AS, Garic D, Graziano P, et al. The frontal aslant tract and its role in speech, language and executive function. *Cortex.* 2019; 111:148–163. doi: 10.1016/j.cortex.2018.10.015
 96. Xia C, Makaretz SJ, Caso C, et al. Association of in vivo [18F] AV-1451 tau PET imaging results with cortical atrophy and symptoms in typical and atypical Alzheimer disease. *JAMA Neurol.* 2017; 74:427–436. doi:10.1001/jamaneurol.2016.5755
 97. Werry EL, Bright FM, Piguet O, et al. Recent developments in TSPO PET imaging as a biomarker of neuroinflammation in neurodegenerative disorders. *Int J Mol Sci.* 2019; 20:121. doi:10.3390/ijms20133161.

HOW TO CITE: Granthali Shape, Pooja Rasal*, Poonam Yadav, Aphasias: A Comprehensive Language Disorder, Clinical Treatments and Clinical Contexts, *Int. J. Sci. R. Tech.*, 2025, 2 (10), 351-363. <https://doi.org/10.5281/zenodo.17374075>