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Neuropsychological criteria for the differential diagnosis of primary progressive aphasia and Alzheimer's disease

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Abstract

This article provides a systematic review of the neuropsychological criteria used for the differential diagnosis between Primary Progressive Aphasia (PPA) and Alzheimer's Disease (AD). Although clinical symptoms overlap in the early stages, each disorder exhibits distinct patterns of cognitive and linguistic impairment. Primary Progressive Aphasia is characterized by a prominent and initial language decline in the relative absence of early episodic memory deficits and is subdivided into three main variants: non-fluent/agrammatic, semantic, and logopenic. In contrast, Alzheimer's Disease presents with prominent memory impairment from the onset, accompanied by a global deterioration in cognitive functions. Neuropsychological assessments and neuroimaging play a crucial role in distinguishing between the two conditions, thereby improving diagnostic accuracy and guiding appropriate therapeutic interventions. Neuroscience and psychology are witnessing rapid progress in understanding neurodegenerative disorders, especially those affecting language and cognition. Among these disorders are primary progressive aphasia and Alzheimer's disease. Differentiating between them is a diagnostic challenge due to the overlap of symptoms and the similarity of clinical manifestations, especially in the early stages. However, we find a difference in the pathological course and mechanisms of neurological deterioration between the two conditions.

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Introduction

Primary progressive aphasia is defined as a type of loss of the ability to speak that represents the predominant clinical feature of some neurodegenerative diseases and is characterized by a specific impairment in language functions such as: Vocabulary, semantics, phonology, syntax, and morphology [1],

Primary progressive aphasia is divided into three main variants, each characterized by specific characteristics. The grammatical variant, PPA-G (non-fluent), is characterized by a disturbance in syntactic processing, affecting the ability to form grammatically correct sentences. The semantic variant, PPA-s (fluent), is characterized by impaired lexical retrieval and loss of word meaning. The final, logopenic variant, PPA-L, is characterized by difficulties finding words in speech and impaired verbal phonological short-term memory, leading to impaired repetition. [2],

Alzheimer's disease is currently the most prevalent disease among the causes of dementia syndromes. Alzheimer's disease was first described at the beginning of the century in a 51-year-old patient, long considered a presenile dementia. In the late 1960s, the first studies indicated similarities between the lesions observed in many dementias and Alzheimer's disease, while criticisms began to be raised about the pathophysiology of cerebral aging, which had long been limited to the theory of decreased cerebral blood flow associated with aging changes in the blood vessels.

However, full awareness of this similarity was not achieved until the late 1970s, leading to the current concept that the disease is a single disease, rarely presenting in pre-senile age and usually after the age of 65, and characterized by progressive brain atrophy accompanied by neuronal loss, along with the presence of neurofibrillary degeneration (DNF) and senile plaques at the histological level.[3],

This disease manifests itself through the early deterioration of certain cognitive abilities, manifested in a set of symptoms, including:

- Repeating the same question
- Disturbed temporal orientation precedes disturbed spatial orientation
- Lack of awareness of one's condition in the early stages of the disease
- Impaired executive functions
- Disturbed memory for events
- Disturbed visual-constructive functions [4],

From this perspective, researchers have paid great attention to studying neuropsychological indicators that can contribute to accurately distinguishing between these two disorders, through a detailed assessment of patterns of cognitive and language deficits. Given the importance of accurate diagnosis by focusing on the distinctive neuropsychological characteristics of each disorder, the importance of presenting and analyzing cognitive and linguistic performance in differentiating between the two conditions is highlighted here, which contributes to improving diagnostic accuracy and developing appropriate treatment for the patient. Furthermore, this difference in diagnostic criteria was the goal that prompted us to attempt to find an answer to the following question:

What are the neuropsychological criteria for the differential diagnosis of primary progressive aphasia and Alzheimer's disease?

Definition of primary progressive aphasia:

Primary progressive aphasia (PPA) is a clinical syndrome diagnosed when three basic criteria are met.

- First, there must be a language impairment (i.e., loss of the ability to speak) that interferes with the use or comprehension of words.
- Second, a neurological examination must determine that the disease is neurodegenerative and therefore progressive.
- Third, the aphasia must arise in relative isolation, without commensurate memory or episodic memory deficits. Language impairment can be fluent or nonfluent and may or may not interfere with word comprehension.

Memory for recent events is preserved. There may be subtle changes in personality and behavior, but these are not the major factors that bring the patient to medical attention or that limit activities of daily living. [5],

Primary progressive aphasia (PPA) is defined as a neuropsychiatric syndrome with progressive damage to the neural network in the left hemisphere that underlies linguistic processes such as phonology, morphology, syntax, semantics, naming, lexical access, repetition, spelling, and writing... Naming disorder is the semiological core. It is often the only symptom for a long time and can be of different types (lexical access disorders). [6],

In the late 19th and early 20th centuries, Pick, Serieux, Franceschi, and Rosenfeld were among the first to describe aphasia resulting from neurodegenerative lesions rather than cerebrovascular lesions. However, many of the patients involved in these researchers' studies failed to meet the current diagnostic criteria for PPA, as the reported language disturbances occurred concomitantly with other prominent cognitive and behavioral impairments. For example, **Pick8** noted that the patient he studied additionally showed a progressive decline in memory and threatened his wife with a knife. The patient reported by Serieux showed a different pattern of neurological impairment. She presented at the age of 47 with a progressive loss of word comprehension but preserved memory. In this woman, the language disturbance progressed to Wernicke's aphasia over an 8-year period until the patient's death in 1897. This case is the earliest historical report of a speech-loss syndrome that can be identified as a prototype of what became known as PPA [7],

Diagnostic criteria:

- Insidious onset and gradual worsening of speech or comprehension problems in spontaneous speech or on formal language testing.
- No impairment in activities of daily living, other than those resulting from language disturbances, for at least the first two years after the onset of the disorder
- Normal state of pre-morbid language functions
- Absence during the first two years of the following symptoms: apathy, forgetfulness of recent events, visual-spatial disturbances, visual recognition deficits, and sensorimotor disturbances.
- After the first two years of development, other areas of cognition can be affected, but language remains the most vulnerable function and its decline is faster than other skills.
- Exclusion of a specific cause, by imaging, (stroke, tumor). [8],

Table01: Classifications of primary progressive aphasia[9],

Type	Symptoms	Affected Areas
Primary progressive non-fluent aphasia **(Agrammatical)**	(Agrammatism) A language disorder that affects sentence structure. He has difficulty arranging words correctly within a sentence. He speaks in a telegraphic style. He omits some functional words (conjunctions...). Fluency, verbal fluidity are low. Phonetic transformations (Paraphasiesphonétiques). Comprehension is preserved (isolated words).	Predominant atrophy of the left posterior frontal lobe(Fronto-insular) on MRI, or ischemia of the left posterior frontal lobe
Fluent Progressive Aphasia **(Semantic)**	Verbal fluency + Oral naming deficit and isolated word comprehension deficit Semantic paraphasia Difficulty in word-finding Impaired reading of irregular words (surface dyslexia)	Predominant atrophy at the anterior temporal level on magnetic resonance imaging (MRI) Or Ischemia at the anterior temporal level

Primary progressive aphasia Logopinic **(PPA Logopinic)**	Spontaneous speech is characterized by a slow rate with frequent pauses due to word-finding difficulties Reduced verbal output - (moderate to severe) Phonemic paraphasia Sentence repetition - + (preserved/impaired) Over time, acalculia (impaired calculation ability) develops	Predominant atrophy in the posterior parietal region and the superior temporal gyrus on magnetic resonance imaging (MRI) or Ischemia in the posterior parietal region and the superior temporal gyrus

Definition of Alzheimer's disease

A neurodegenerative disorder characterized by the progressive deterioration of brain tissues and associated cognitive functions. It is also defined as memory impairments accompanied by other cognitive disturbances that affect an individual's daily life. According to the French Ministry of Solidarity and Health, it is considered a degenerative neurological disease and a progressive neurological disorder leading to neuronal cell death. It is marked by a gradual loss of memory and certain mental functions[10-12],

The French National Agency for Accreditation and Evaluation in Healthcare (ANAES) defines Alzheimer's disease as a neurodegenerative disorder characterized by progressive and irreversible deterioration of the central nervous system: the degeneration of nerve fibers and amyloid plaques. The natural course of the disease often leads to dementia, which is a severe decline in cognitive functions significant enough to profoundly disrupt the patient's daily life[13-15],

The Arthrophonic Dictionary defines it as the most common type of dementia, characterized anatomically by atrophy of the cerebral cortex, primarily in the parietal-temporal regions and hippocampus, along with enlargement of the cerebral ventricles. Clinically, it presents with dementia accompanied by memory impairments, spatiotemporal disorientation, loss of speech (aphasia), apraxia, agnosia, epileptic seizures, and muscle hypertonia... It is associated with cerebral deficits in neurotransmitters, particularly cholinergic ones. Early speech therapy is crucial as it allows for accurate assessment of deficits—especially in communication and memory—and aims to preserve communication methods and cognitive stimulation for as long as possible.[16],.

The anatomical basis of Alzheimer's disease:

Currently, scientists understand much of what occurs in the brains of individuals with Alzheimer's disease. The brain consists of millions of neurons (among other components) that enable cognitive functions such as thinking and memory. In an Alzheimer's-affected brain, abnormal accumulation of a protein called amyloid occurs (for reasons not yet fully understood). Under microscopic examination, amyloid protein deposits can be observed beneath the outer layers of the brain, forming clusters referred to as plaques. These plaques are believed to disrupt activities essential for neuronal survival. Within these neurons resides another protein called tau, which is responsible for maintaining the structural integrity of nerve cells. When neurons become damaged, abnormal forms of tau protein begin to accumulate. This alteration in tau protein conformation ultimately leads to structural changes in the neurons themselves[17],

. Diagnostic Criteria for Alzheimer's Disease :

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), specifies the following criteria for the diagnosis of Alzheimer's disease:

- The emergence of multiple cognitive disorders manifested as significant problems in memory, attention, executive functions, language, perceptual-motor skills, and thinking.
- The impact of these criteria on daily life, social, and occupational functioning (where, in severe cases, the individual is unable to be self-reliant in activities of daily living such as cooking, paying bills, etc.(.
- A slow and gradual progression of the aforementioned symptoms over time.
- The absence of another cause that better explains these symptoms, meaning the exclusion of other nervous system conditions such as cerebrovascular disease, Parkinson's disease, Huntington's disease, traumatic brain injury, depression, brain tumors, and psychiatric disorders
- The presence of a family history or diagnostic tests that support the diagnosis if there is evidence of a causative genetic mutation.[18],

Stages of Alzheimer's Disease Progression:

The progression of Alzheimer's disease is divided into three stages:

1. The **initial stage (Stage 1)** involves impairments in **episodic memory**, and **neuropsychiatric symptoms** (apathy, loss of interest, irritability, and mood lability), in addition to changes in affect, anxiety disorders, **anomia** (with potential **dysgraphia**), and mild spatiotemporal disorientation. This stage lasts between 2 and 4 years.

2. The **moderate stage (Stage 2)** is characterized by more severe memory impairments. **Cognitive disorders** may appear (**aphasia**, **apraxia**, **agnosia**, impaired concentration, disorientation, etc.). Neuropsychiatric symptoms remain present. Autonomy declines more and more, and spatiotemporal disorientation deteriorates further. This is the longest stage and can range from 2 to 10 years.

3. The **final stage (Stage 3)** is characterized by a complete loss of autonomy with significant neuropsychiatric symptoms. Neurological symptoms (**myoclonus**, epileptic seizures) can also be observed.[19],

The Anatomical Basis of Alzheimer's Disease:

Alzheimer's disease is considered a clinical and neurological syndrome. It is associated with specific cognitive and behavioral symptoms linked to neuropathological lesions, particularly senile plaques and neurofibrillary tangles.

Among the anatomical features associated with Alzheimer's disease are:

- Senile Plaques (Amyloid Plaques): These are deposits of beta-amyloid protein that accumulate in the brain, disrupting neuronal function. They are a hallmark feature of the disease and are found in the extracellular space of the brain.

- Neurofibrillary Tangles: These tangles consist of hyperphosphorylated tau protein. They form inside neurons and are associated with neurodegeneration and cell death.

- The hippocampus is one of the first regions affected in this disease, leading to memory loss and cognitive decline. This region is essential for memory formation and is often where the amnesic syndrome begins.

- Magnetic Resonance Imaging (MRI) reveals cortical atrophy in various regions, indicating the associated neurodegeneration.

- Atrophy also encompasses other areas such as the temporoparietal cortex (responsible for sensory processing and language comprehension; its atrophy leads to difficulties in understanding and communication, particularly in the later stages of the disease) and the posterior cingulate cortex (which shows hypometabolism on Positron Emission Tomography (PET) scans in Alzheimer's patients; it is responsible for emotional regulation, cognitive control, and memory processing). These regions are involved in various cognitive functions, including spatial awareness and decision-making.

- Biochemical Changes: The accumulation of beta-amyloid plaques in the brain involves a decrease in Amyloid Precursor Protein (APP) levels and an increase in the production of beta-amyloid, which contributes to plaque formation. This accumulation triggers a pathological cascade leading to neurodegeneration. Additionally, there is

an increase in tau protein levels, particularly phosphorylated tau, which is associated with neurofibrillary tangles. These tangles disrupt neuronal function and contribute significantly to the cognitive decline observed in Alzheimer's patients. The presence of tau in the Cerebrospinal Fluid (CSF) is often used as a biomarker for the disease.[20],

Neuropsychological Differential Diagnosis between Primary Progressive Aphasia and Alzheimer's Disease:

Primary Progressive Aphasia (PPA) encompasses two main clinical forms. The first is Nonfluent/Agrammatic Variant Primary Progressive Aphasia (APPNF), initially described by Mesulam. It is characterized by impaired language (which may progress to mutism), and halting, effortful verbal expression with disturbances in phonological execution and dysprosody, in addition to difficulties with orofacial motor planning. Overall, the presentation resembles Broca's aphasia. There is also agrammatism, anomia, word repetition, which can be interspersed with phonemic paraphasias. Comprehension is intact for isolated words, but sentence comprehension is impaired. Written language is generally less affected than oral language. These patients do not suffer from memory disorders or visuoconstructive and visuospatial impairments; they also lack behavioral disturbances in the initial stages of the disease. On imaging, atrophy affects the anterior perisylvian region, particularly the inferior frontal area and the insula. Dynamic imaging techniques (e.g., SPECT, perfusion MRI) show images revealing hypoperfusion. In general, anatomical examinations indicate that (APPNF)most often corresponds to the underlying pathology of frontotemporal lobar degeneration.

The second clinical form is logopenic variant primary progressive aphasia (APPNF),. Although there is an absence of phonemic disintegration, dysprosody, and agrammatism, verbal expression is slow, effortful, and halting, with pauses associated with anomia (word-finding difficulty). For this reason, it can be classified as non-fluent even in the absence of articulatory and grammatical problems. In addition to anomia, phonemic paraphasias may intrude upon spontaneous speech and confrontation naming.[3],

A common initial symptom shared by both fluent and nonfluent variants of Primary Progressive Aphasia (PPA) is difficulty in word-finding and anomia. In such cases, the patient employs linguistic compensatory strategies such as circumlocution (e.g., saying "the thing you walk with" instead of "cane"), simplification (e.g., saying "bow-wow" for a dog, or using a generic term like "flower" instead of "rose"), or explaining the word by its use rather than naming it directly.

In Nonfluent/Agrammatic Variant Primary Progressive Aphasia (APPNF), the following symptoms are present: anomia, reduced mean length of utterance (MLU), effortful and labored expression, agrammatism (grammatical errors), and a nonfluent, halting, and effortful speech output characterized by low, interrupted flow filled with pauses and hesitations. The speech productions are marked by phonemic paraphasias (sound substitutions) and, ultimately, mutism. Meanwhile, patients retain their autonomy, and comprehension ability remains preserved for a prolonged period.

Fluent Primary Progressive Aphasia (APPf), often corresponding to the semantic variant, is characterized by a naming deficit (anomia) that begins gradually and worsens over time, alongside impairments in verbal comprehension and semantic paraphasias (substituting one word for another from the same category, e.g., saying "cat" instead of "lion"). Despite these deficits, speech remains spontaneous and fluent, even in the presence of word-finding difficulties. This variant is often confused with semantic dementia due to the significant overlap in symptoms.

The initial symptoms of Primary Progressive Aphasia (PPA) typically manifest at an earlier age compared to Alzheimer's disease. The two conditions can be differentiated by their primary presenting symptoms:

- Initial Symptoms in PPA: Episodic memory (memory for autobiographical events) is not the primary domain initially affected. Instead, the core deficits are in the language domain (e.g., difficulties with expression or comprehension).

- Initial Symptoms in Alzheimer's Disease: In contrast, impairment in episodic memory is the predominant and characteristic initial symptom.

Supplementary Examinations and Neuropsychological Assessment:

Diagnostic tests such as Magnetic Resonance Imaging (MRI) and specialized language assessments aid in differentiating between the two conditions.

- In Alzheimer's disease, atrophy is typically observed in the hippocampal region and presents as cortico-subcortical atrophy.
- In Primary Progressive Aphasia (PPA), the pattern of atrophy varies by subtype:
- In the Nonfluent/Agrammatic variant (PPANF), atrophy is located in the anterior perisylvian region (inferior frontal gyrus, insular lobe).
- In the Semantic variant (PPASV), atrophy affects the anterior temporal regions.
- In the Logopenic variant (PPA), atrophy is found in the posterior temporo-parietal region.

A patient with Primary Progressive Aphasia may initially complain of memory impairment; however, a comprehensive neuropsychological evaluation reveals that the core deficit is fundamentally linguistic in nature. Indeed, it is crucial to confirm that the impairment is predominantly confined to the language domain in the early stages. Furthermore, patients with PPA generally retain their autonomy for a significantly longer period compared to patients with Alzheimer's disease.

The severity of the language disorder can impede the administration and interpretation of cognitive tests, including non-verbal ones, as all such tests rely on verbal instructions and utilize linguistic abilities for both reasoning and responding.

On the Mini-Mental State Examination (MMSE), patients with Primary Progressive Aphasia (PPA) may demonstrate relatively better performance in certain domains compared to patients with Alzheimer's disease (AD). This is particularly evident in tests of temporal orientation and the recall of the three words. Conversely, their performance is typically less efficient on tasks involving repetition and other language-based measures[21],

Neuropsychology has always aimed to establish correlations between the location of anatomical lesions and the presence of clinical disorders. This objective remains pertinent in Alzheimer's disease (AD), where no neuroimaging technique alone can establish the diagnosis. Furthermore, advances in neuroimaging data and clinical observation can only enrich one another.

Thus, episodic memory impairments are observed very early in the disease in relation to hippocampal damage. The syndromes of aphasia, apraxia, and agnosia reflect the spread of the pathology to the association cortices. The disease's presentation is, in fact, stereotypical due to the hierarchical and sequential progression of neurofibrillary degeneration from the hippocampal region to the temporal cortex and temporo-parietal association areas, and subsequently to the frontal lobe, while the primary cortical areas remain preserved for a long period.

The memory disorder is characterized by a deficit in encoding and consolidation, as well as impaired retrieval. Additionally, there is a progressive deterioration of the memory store. Contrary to prior belief, semantic memory is not spared from this deterioration.

The aphasia initially results in anomia (difficulty in word retrieval, use of neologisms and vague words, verbal and then phonemic paraphasias, and the use of circumlocutions—lengthy phrases to describe a simple object). Comprehension becomes impaired, while the ability for repetition remains intact, indicating a clinical picture resembling Transcortical Sensory Aphasia.

Furthermore, disturbances in abstract thinking, judgment, and executive functions are observed. [22],

Memory impairment is the hallmark feature of the cognitive presentation of Alzheimer's disease. The initial stage is characterized by mild memory deficits (e.g., misplacing objects, forgetting conversations, difficulties in recalling names and appointments...). However, patients with Alzheimer's disease typically suffer from global cognitive impairment.

A study conducted to identify the best predictive indicators of Alzheimer's disease found that delayed auditory-verbal and visual recall (with intrusions and perseverative errors), confrontation naming tests, tests of cognitive flexibility, and divided attention were the strongest predictive indicators of Alzheimer's disease in a cohort of highly educated individuals.

Alzheimer's disease does not always present as a neuropsychologically homogeneous entity that begins with memory impairment followed by deficits in other cognitive domains. Some patients may exhibit prominent deficits in cognitive areas other than memory, especially in the early stages of the disease. Symptoms of frontal lobe or occipital lobe dysfunction, or impairment in executive functions, may be the most obvious initial signs in Alzheimer's disease. Language impairment is the second most prominent cognitive manifestation of Alzheimer's disease. [23],

In naming tasks and verbal fluency, the semantic impairment is manifested through anomia ("manque du mot"), which is the individual's inability to name familiar objects. This phenomenon has been explained by several hypotheses:

- The Lexical Access Deficit Hypothesis: Some researchers posit that anomia results from a disruption in accessing vocabulary stored in the mental lexicon.

- The Lexical-Semantic Degradation Hypothesis: Others propose that the damage begins with impaired lexical access and later progresses to involve the semantic network in subsequent stages of the disease.

- The Role of Executive Functions Hypothesis: More recently, a hypothesis has emerged suggesting that a deficit in the executive functions responsible for concept retrieval may be the underlying cause.

Subordinate (specific) semantic concepts deteriorate more rapidly, leading to a progressive loss of precision in patients' speech. This significantly impacts the ability to perform Activities of Daily Living (ADLs), accompanied by disturbances in both expression and comprehension.

The anomaly manifests in lexical errors in the form of:

Pauses or hesitations ,Neologisms (inventing words),Perseverations (involuntary repetition of words), Circumlocutions (using long phrases instead of a target word) ,Visual errors ,Use of over-generalized terms (e.g., saying "animal" instead of "dog"),Semantic paraphasias (substituting a word with a related one, e.g., saying "apple" instead of "orange") [24],.

Conclusion

In conclusion, the differential diagnosis between Primary Progressive Aphasia and Alzheimer's Disease, while challenging, is critically informed by distinct neuropsychological and anatomical profiles. PPA is defined by its initial, isolated language deficits and specific atrophy patterns within the language network, with memory remaining relatively preserved. Conversely, AD is characterized by early and profound episodic memory impairment alongside more diffuse cognitive decline and hippocampal atrophy. A comprehensive neuropsychological evaluation, supplemented by neuroimaging, is therefore indispensable for accurate diagnosis. This distinction is paramount not only for prognosis but also for implementing appropriate, targeted management strategies and care pathways for affected individuals. Future research should continue to refine these diagnostic criteria to enhance early and precise identification.

Ethical Considerations

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki and the ethical regulations of the University of Abd al Hamid Mehri, Constantine 2. All participants (or their legal guardians, when appropriate) were informed of the objectives of the research and provided written informed consent prior to participation. Confidentiality and anonymity were strictly maintained, and all neuropsychological data were processed and reported in a manner that prevents personal identification. No clinical or psychological procedures posing risk to participants were performed beyond standard diagnostic protocols.

Author Contributions

Sara Nedjimi (PhD student) – Conceptualization, research design, literature review, data collection, drafting and finalization of the manuscript.

Pr. Hazem Hamani – Scientific supervision, methodological guidance, critical revision of manuscript, and theoretical validation.

Dr. Nafissa Ghanem – Data interpretation, neuropsychological framework consultation, review and editing of the manuscript.

All authors have read and approved the final version of the manuscript.

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Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article. No author has financial or personal relationships that could improperly influence or bias the material presented.

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