



## **Physiological Basis of Nonmemory Cognition in Alzheimers Disease-An Overview**

**A. S. V. Prasad<sup>1\*</sup>**

<sup>1</sup>*Department of Internal Medicine, G.I.T.A.M, Rishikonda, Visakhapatnam, India.*

### **Author's contribution**

*The sole author designed, analysed, interpreted and prepared the manuscript.*

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### **ABSTRACT**

Alzheimer's (AD) disease is foremost of the neurodegenerative diseases affecting cognition. Though memory, out of all cognitive functions of the brain, received much attention, the nonmemory cognitive functions including the higher brain functions, are equally important. In fact what is perceived by the five senses is the basis of our learning, what is learned thus, is stored in the brain as memory of various types and the retrieved memory appropriate to the situation is the basis for higher brain functions. Thus all are interconnected when overall cognitive functions of the brain are considered. This article focuses on the nonmemory functions of the brain (like attention, perception, language and learning) as well as higher brain functions like (thinking, planning, execution, judgement, emotional and social behaviour etc). Their physiological basis as well as how they are affected in AD are discussed in this article.

*Keywords: Nonmemory cognition; attention; perception; learning; language.*

## 1. INTRODUCTION

### 1.1 Definition of Cognition

Cognition is "the mental process of acquiring knowledge and understanding through thought, experience, and the senses". Cognition includes, thus, comprehension, contemplation, description, reasoning, memory judgement, planning and execution etc.

#### Classification of cognitive functions:

The cognitive functions can be broadly devised into

- Memory,
- Nonmemory cognition including the higher brain functions.

**Memory:** Memory is the faculty of the brain by which data or information is encoded, stored, and retrieved when needed. The subject matter is recently reviewed by this author [1].

**Nonmemory cognition:** The nonmemory cognitive functions are as important as the memory itself. In fact, they are responsible for initiating the process of memory. The nonmemory cognition is lost earlier than executive memory in Alzheimer's disease. Thus, the imaging techniques described the loss of brain volume in areas responsible for nonmemory cognition, as in AD. Physiological dysfunction in the association cortex is evident in early Alzheimer's type dementia before the neuro-psychological consequences of that dysfunction are demonstrable" [2]. Effect of amyloid on memory and non-memory decline from preclinical to clinical Alzheimer's disease was studied by Yen Ying Lim, Paul et al (2014) [3]. They found that "amyloid-positive individuals with mild cognitive impairment (MCI) showed additional moderate decline in language, attention and visuo-spatial function, and amyloid-positive individuals with Alzheimer's disease showed large decline in all aspects of memory and non-memory function.

The nonmemory cognitive functions are shown in Table 1.

The higher brain functions are shown in the Table 2.

## 2. DISCUSSION

**Higher order or intellectual brain functions:** The cerebral cortex is responsible for the higher

brain functions as listed on Table 2. Most of these functions are controlled by frontal lobe cortex, especially prefrontal cortex except stereognosis which controlled by parieto-temporal association cortex.

**Table 1. Types of non- memory cognitive processes**

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<ul style="list-style-type: none"> <li>• Attention</li> <li>• Perception</li> <li>• Language</li> <li>• Learning</li> </ul>
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**Table 2. Table showing the higher brain functions**

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<ul style="list-style-type: none"> <li>• Thinking</li> <li>• Reasoning</li> <li>• Judgement</li> <li>• Planning</li> <li>• Motivation and drive.</li> <li>• Emotional control.</li> <li>• Language formation.</li> <li>• Voluntary movement,</li> <li>• Social and sexual behavior.</li> <li>• Speech</li> <li>• Sense of time, place and position</li> <li>• Vibration &amp; joint sense</li> <li>• Stereognosis</li> </ul>
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**Loss of nonmemory cognition and higher brain functions in AD:** All nonmemory and the higher brain functions are lost during the clinical course of AD sooner or later. The most commonly observed cognitive defect is the changes in memory. First to be lost is the executive memory and the other types of memories follow later. The first nonmemory cognitive function to be affected in AD is perception of sense of smell. It is even suggested as an important parameter in the early recognition of onset of AD. Attention and learning difficulties are common. They may develop impairment of speech and language. The patient may suffer from lack of self motivation and drive. Stereognosis (also known as haptic perception or tactile gnosis) is the ability to perceive and recognize the form of an object in the absence of visual and auditory information, by using tactile information to provide cues from texture, size, spatial properties, and temperature, etc. It may be caused by disease of the sensory cortex or posterior columns. Astereognosis can be caused by damage to sensory cerebral cortex the posterior association areas of the parietal, temporal, or occipital lobes, or the postcentral

gyrus of either hemisphere. People suffering from Alzheimer's disease show a reduction in stereognosis. For other types of dementia, stereognosis does not appear to decline.

Some of the symptoms observed in AD due to loss of one or the other higher brain functions are dealt with AD symptomatology in AD.

**Symptomatology due to loss of higher brain functions in AD:** The AD patient may forget the names of objects, the purpose for which they are used, find difficulty in choosing the right word, fail to tell the time or place where they are. They may fail to recognise even familiar faces and their relationship to the sufferer of AD. fail to learn or repeat simple tasks, may suffer from loss of smell, taste and hearing. In addition, impairment of short time memory that effects their executive functions (they find it difficult to recollect where they kept their keys or spectacles), find it difficult remembering past memories (events like their marriage anniversary date), and even forget some skills they learnt before. Emotional and personality changes, sleep disturbances etc are apart of the AD symptomatology. Psychiatric manifestations like anxiety – depression, hallucinations and delusions may occur. Finally, they may fail to take care of themselves and totally become dependent on their caretakers even for their day to day tasks. The loss of orientation of time and place may result in AD patients losing their way and are found wandering.

The physiological basis of higher brain functions and each of the important nonmemory cognitive functions are discussed separately.

**Brief consideration of the physiology of higher brain functions:**

The higher functions of the brain are observed by 3 specialized areas of the brain

1. The primary sensory area
2. The secondary sensory area
3. Association areas:  
These in turn receive feedback from lower centers. They are-

**Thalamus  
Brainstem and  
Cerebellum**

**Thalamus** is the central sensory and motor relay station of the brain, reciprocally communicating

and relaying signals between subcortical and cerebellar regions and the cortex, playing a role in sleep, arousal, and primary sensory processing (Adams and Victor, 1993) [4].

**The cerebellum** contributes primarily to the planning and execution of movements Schmahmann, [5]. **The brainstem** is related to executive dysfunction, attentional deficits and a decline in general intellectual capacity represent the most common cognitive findings, but memory, visuospatial skills, language and praxis may be impaired as well. Almost half of the cases presented with behavioural or affective changes. Detect specific sensations—visual, auditory, or somatic—transmitted directly to the brain from peripheral sensory organs [6].

**1. Primary sensory areas:**

- a) Primary somatosensory cortex: (the sense of touch).
- b) Primary auditory cortex: processes sound and hearing.
- c) Primary visual cortex: processing information about static and moving objects and pattern recognition

**2. Secondary sensory areas:**

Analyse the meanings of the specific sensory signals.

**3. Association areas:**

Deceive and analyze signals simultaneously from multiple regions of both the motor and sensory cortices, as well as from subcortical structures. There are three important association areas

• **Parieto-occipitotemporal Association Area:**

Receives sensory information from all surrounding areas.

1. Analysis of the Spatial Coordinates of the Body
2. **Wernickes area:** Comprehension of language.
3. **Angular gyrus:** Initiation of visual language ( reading ).
4. **Area for Naming Objects:** Located in lateral most portions of the anterior occipital lobe and posterior temporal Lobe.

• **Prefrontal Association Area:** In association with the motor cortex to plan

complex patterns and sequences of motor Movements also essential to carrying out “thought” processes in the mind.

**Broca's area:** Plans and motor patterns for expressing individual words or even short phrases are initiated and executed.

- **Limbic association area:** It is concerned primarily with behavior, emotions, and motivation.

**Area for Recognition of Faces:** Prosopagnosia is inability to recognize faces. Occurs in people with damage on the medial.

Undersides of occipital lobes and along the medioventral surfaces of the temporal lobes

**Physiology of attention:**

RA. Med Hypotheses, 1990 [7] Hypotheses. 1990

**Types of attention**

- **Selective attention** takes place when we block out certain features of our environment and focus on one particular feature, like the conversation you are having with your friend.
- **Devised attention** When we are paying attention to two things at one time, we are using divided attention.
- **Sustained attention (attention span)** Ability to keep attention on one eventful prolonged period is called sustained attention.
- **Executive attention;** Blocking all other events in the environment and concentrating on the task on hand.
- **Alternating attention:** The attention is distributed between to alternating stimuli.

The difference between this and devised attention is, that, in the later, the whole attention is devised between several stimuli. In the former, the whole attention is shifted alternatively between two stimuli.

- **Auditory attention:** The focus of attention is on what you hear at the near exclusion of other stimuli. Imagine that you are hearing an important broadcast. You feel irritated to any other stimuli that distract your attention from hearing.
- **Visual attention:** Here more attention is paid to what you see on hand, than other stimuli. Auditory and visual attentions are perhaps subdivisions of devised attention. This is because, other stimuli are also received depending on their priority. A calling bell rings, when you are focusing on a visual Your attention is drawn in spite of your focus on what you see.

**Attention deficit in AD:** The readers are directed to the elaborate article on Attention deficits in Alzheimer's disease. Richard J. Perry, John R. Hodges, et al. 1999 [8] in AD, Of the various attention functions, divided and selective attention are particularly vulnerable, than sustained attention. The attention deficits might even precedes the executive function defects and may be contributory to the later. The aetiology of attentional deficits in AD arises from damage to areas of frontal and parietal association cortex, disconnection between the anterior and posterior attentional networks, and decreased cholinergic function. Severity of illness is associated with attentional deterioration. Complexity or increased load of a task may further disrupt attentional function. Knowledge of attentional changes in AD is important to the understanding of disease-related changes in other cognitive domains such as memory, visuospatial functions, and language.

**Table 3. Summary of essentials of the Med hypothesis**

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1. The sub cortical centres and the cortical centres. first scan the sensory inputs.
2. Thalamus concentrates them into one sensory modality by Thalamic-Prefrontal-Thalamic reflex inhibition.
3. Engrams are activated in the Posterior Inferior Temporal Cortex, and
4. Posterior Inferior Parietal Cortex, and then the information passes to
5. Posterior and Anterior Association Cortex, which project to the Prefrontal Association Cortex (PAC).
6. The PAC sends signals to the Hippocampus,
7. The theta activity of the hippocampus transiently interrupts the blanket inhibition of orientation, alertness, awareness, and arousal, through the Hypothalamus
8. Thus the focussed attention occurs. Types of attention.

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### Types of perception:

- Visual
- Auditory
- Taste
- Smell
- Touch

### Perception defects in AD:

**Vision:** The following defects occur in AD.

- **Colour:** Blue- violet colour perception is affected.
- **Contrast:** AD patients has difficulty in identifying objects if they have same colour as it's surrounding. For instance, the toilet seat may be difficult to identify if the walls and floor also have white tiles.
- **Depth:** The loss of depth perception may make an AD patient to mistake the edge of a core as a step.
- **Movement:** For an AD patient, surrounding is like 'still photo' unlike a video in normal people. Stationary surroundings confuse and make them lose their way.
- **Reduced peripheral vision:** This leads AD patient to bump against obstacles as sideways vision is narrowed.

### Common vision mistakes in AD:

- **Failure to recognise faces:**
- **Failure to recognise objects:**
- **Misperception:**

### Misinterpretation:

#### Illusions:

#### Hallucinations:

#### Delusions:

### Defects in auditory perception in AD

An AD, the patient hears better in calm surroundings than among the din.

They may suffer auditory hallucinations.

**Smell and Taste dysfunction in AD:** The senses of smell and taste (gustatory system) are often referred to together as the **chemosensory system**, because they

both give the brain information about the chemical composition of objects through a process.

**Olfactory perception:** The uncus houses the **olfactory cortex** which includes the piriform cortex (posterior orbitofrontal cortex), amygdala, olfactory tubercle, and Para hippocampal gyrus. The olfactory tubercle connects to numerous areas of the amygdala, thalamus, hypothalamus, hippocampus, brain stem, retina, auditory cortex, and olfactory system, In total it has 27 inputs and 20 outputs. Patients with Alzheimer disease show changes in detection, discrimination, and identification of odours compared with age-matched controls. The severity of dysfunction is correlated to disease progression, although in most cases, olfactory loss is present years before motor or cognitive symptoms; this is usually a gradual loss and often goes unnoticed or unreported by patients. An association has also been recognized between smell loss and increased risk of mortality studies confirmed that olfactory dysfunction was possibly one of the earliest clinical symptoms of AD [9]. In addition, typical AD pathology first involves the entorhinal cortex. The disease then gradually spreads to the whole brain and eventually affects the entire cerebral cortex. [10]. several studies have suggested that olfactory deficit can be used as a biomarker for early diagnosis of AD [11,12]. Olfactory function is decreased even in early stages of AD [13,14] study confirmed reduction of acetylcholine in AD patients, which might induce olfactory dysfunction [15].

**Gustatory dysfunction in AD:** The central **lesions** affecting **Thalamus and Uncial** region are implicated. Structural involvement of thalamus and its connection with limbic system and frontal lobes which show definite involvement in AD might be involved here. The olfactory tubercle connects to numerous areas of the **amygdala, thalamus, hypothalamus, hippocampus, brain stem, retina, auditory cortex and olfactory system**. In total it has 27 inputs and 20 outputs.

**Tactile perception in AD:** For the individual with Alzheimer, touch may be the only way of reciprocal communication when that individual becomes nonverbal or at the end

stage of the disease. When a gentle caring touch is offered to someone with Alzheimer it can ease anxieties and help increase feelings of general well-being. Even with advanced dementia do not lose the capacity to recognize caring touches. Touch can calm agitated behaviours, reduce agitation, ease physical discomfort and promote sleep. Touch also can make emotional connections to others, particularly because individuals with AD have such difficulty with communication.

Touch activates part of the orbitofrontal cortex of the brain and stimulates production of a hormone known as oxytocin, what scientists call the "care and connection" hormone. This reaction in the brain leads to feelings of safety, trust, and a reduction in stress and anxiety.

**Language:** Most right-handed people have intellectual skills for speaking and understanding language largely concentrated in the left inferior frontal lobe, and cognitive skills for attention control and memory concentrated in the right inferior frontal lobe. Even in left-handed people, speaking and language skills are more commonly concentrated on the left side.

**Language impairment in AD** is primarily a result of decline in semantic and pragmatic levels of language processing [16]. Semantic processing involves language content, such as meaning of words and the associated impairments include difficulties with word finding, naming, and word comprehension, as well as semantic paraphasia (choosing incorrect words), empty speech (using ambiguous referents), inventing words, and loss of verbal fluency. Pragmatic processing goes beyond words and their meaning and concerns language adaptation to the social situation. Examples of pragmatic problems are speaking too much at inappropriate times, talking too loudly, repeating ideas, and digressing from the topic. Deficits in pragmatic processing may also be influenced by other AD symptoms, such as impairments in memory and concentration, and disinhibition.

**Learning:** It is the process of acquiring new, or modifying existing, knowledge, behaviours, skills, values, or preference [17]. Storing in the brain of what is learnt is the

memory. So learning and memory are interdependent.

**Learning function deficit in AD:** "Gary Van Hoesen's laboratory at Iowa in the early 1980s established that the hallmark neuropathology of Alzheimer's disease first appears in specific parts of the hippocampal formation and entorhinal cortex, effectively isolating the hippocampus from much of its input and output. with association cortices, basal forebrain, thalamus, and hypothalamus and causing the distinctive impairment of new learning that is the leading early characteristic of the disease" [18].

Memory impaired participants learn less and show less rapid learning than healthy participants (i.e., memory impaired individuals require many more trials to acquire only a fraction of what healthy participants can learn) (2004; Skotko et al. [19].

The hallmark deficit in the early stages of AD is a profound impairment in acquiring new declarative memory for both semantic (facts, vocabulary) and episodic (events of daily life) information [20].

The observed memory impairment is consistent with evidence suggesting that early pathological involvement is localized in the mesial temporal lobes, especially in the hippocampal formation and entorhinal cortices (Hyman et al., 1984).

Previous studies have found that education is one of the best protectors against Alzheimer's disease, though it's never been clear exactly how the two are correlated. They hypothesized it was the cognitive reserve of highly educated people that allowed them to live with the plaques and tangles associated with Alzheimer's without the symptoms of memory loss.

This finding that education apparently contributes little to cognitive reserve is surprising given that education affects cognitive growth and changes in brain structure," Wilson et al all The researchers point out that learning itself is still a valuable protective factor against neurodegeneration. (Robert S. Wilson,

Ph.D., of Rush University Medical Center in Chicago).

**The physiological Change in brain that occur during learning:**

- Formation of new connections between nerve cells.
- formation of new network systems.
- Formation of new neurons which are not functionally significant.

An often-quoted example is the singing bird which, during the mating season develops new neurons which are associated with the singing which perish after mating season, only to regrow during next mating season. The basis of this is existence of a few stem cells among neurons.

**Table 4. Types of learning (discussed below in detail)**

Associative	Non-Associative
1) Classical conditioning	Habituation
2) Operant conditioning	Sensitization

**Associative learning:**

- **Classical conditioning:** The classical example is 'Pavlov experiments on dog. In his experiments, he found that the very sight of an attendant's bringing food (Conscious stimulus) evoked saliva in the mouth of the dog (an unconscious response [21]. A conscious stimulus: not an unconscious response is called classical conditioning. Saliva secretion is conditioned to sight of the food here.
- **Operant Conditioning:** Operant conditioning (sometimes referred to as instrumental conditioning) is a method of learning that occurs through rewards and punishments for behaviour

[22]. Through operant conditioning, an association is made between a behaviour and a consequence for that behaviour. An example is that a mouse that learn to get a food pellet by pressing a blue button presses the same for reward and avoids pressing the red button that gives it an electric shock. Punishment. An example is that a car driver is conditioned to stop, when red signal appears and start when green signal is flashed.

The linking of the stimulus to the operant occurs in Hippocampus and Amygdala and integration occurs in orbito-frontal cortex (OFC).

**Non associative learning:**

**Habituation:** It is a type of non-associative Learning. Habituation is behavioural responsiveness to a test stimulus decreases with repetition. It has the important function of enabling us to ignore repetitive, irrelevant stimuli, so that we can remain responsive to sporadic stimuli, typically of greater significance. (international encyclopaedia of learning and behavioural Science, 2001). For example, a ring tone of a mobile may distract our attention at first, but on repeated exposures fails to draw the same attention i.e the response to repetition has diminished [23].

**Sensitization,** refers to a non-associative learning process through which repeated exposure to a stimulus results in the progressive amplification (increasing strength) of the reaction to the stimulus [24]. The organism becomes more sensitive to the stimulus as time progresses. It is opposite of habituation. (American Psychology Association, *Alleydog.com's online glossary*). For example, repeated blurring's on a loudspeaker may increase your annoyance.

**Table 5. Other learning types described in literature**

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- 1) **Receptive learning:** A passive type of learning. ex-student attending a teachers lecture.
- 2) **Cooperative learning:** It is a group learning involving each group members contribution.
- 3) **Rote learning:** Learning without deep insights.
- 4) **Emotional learning:** involves development of emotional intelligence that manages ones
- 5) **Experimental learning:** A subjective learning by trial and error.
- 6) **Observational learning:** learning by observing/ imitating somebody.
- 7) **Implicit learning:** Learning without involving conscious mind.
- 8) **Explicit learning:** learning by applying the conscious mind.

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### 3. CONCLUSION

The nonmemory cognitive functions including the loss of higher brain functions, are as important as cognitive memory. The constituents of the nonmemory cognition and the higher brain functions are enumerated, their physiological basis is discussed and how the loss of these functions are correlated to the symptoms of Alzheimer's disease are focused in this article.

### COMPETING INTERESTS

Author has declared that no competing interests exist.

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