

## Review Article

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## Cortical V/S Subcortical Dementias- A Comparative Review of Cognitive Functions

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### ABSTRACT

**BACKGROUND:** The typical clinical features of AD are indicative of cortical dysfunction which are dyscalculia, dysphasias, dyspraxias and agnosias. Parallel to this, the clinical features of subcortical dementias, mainly reflect the subcortical areas affected which usually are personality changes and affective disorders and bradyphrenia. The neuropsychological deficits are also different in these dementias. In present review we will highlight these neuropsychological differences.

**METHODOLOGY:** A detailed search of all the studies of cortical and subcortical dementias was done in all medical databases. All the studies involving neuropsychological aspects of cortical and subcortical dementias were included in the study. Major categories of neuropsychological aspects were separately categorized and compared between the subcortical and cortical dementias.

**RESULTS:** Major neuropsychological categories identified were 1) Executive functions 2) Memory dysfunctions and 3) Perceptuo-motor dysfunctions. In memory, The differential effects of the test format is that in subcortical dementia the main difficulty is in retrieving information while in cortical dementia encoding or storage (or both) are thought to be defective. On the other hand, the visuo motor dysfunctions are far more severe in subcortical as compared to cortical dementias. Its cardinal features are: forgetfulness, i.e., difficulty in retrieving learned material; slowing of mental and motor processes; intellectual deterioration characterized by impaired ability to manipulate acquired knowledge to generate problem solving; impairment of arousal, attention, and motivation and affective changes (depression); and impairment of set-shifting.

**DISCUSSION:** There are significant differences in various neuropsychological domains of cortical and subcortical dementias.

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## **INTRODUCTION**

The idea that dementia can be separated into cortical and subcortical types has found support from both the clinical and etiologic perspectives. But this view came into picture only in mid-1970s only after the concept of Subcortical Dementia started becoming more frequently used. However this usage actually was quite delayed as Subcortical dementia was first described in 1912 when it was identified as a clinical syndrome. Our understanding of cortical and subcortical dementias have come from studying various neurological disorders affecting different parts of brain. While Alzheimer's Disease and Frontotemporal dementia are primarily cortical dementias and Huntington's disease, Parkinson's disease, Progressive supranuclear palsy, Binswanger Disease, Multiple sclerosis and Sydenham's chorea are subcortical dementias, other disease like Corticobasal degeneration, Multiple system atrophy and Vascular dementia and HIV dementia involve both cortical and subcortical regions producing a very complicated picture of dementia [1]. Here we try to summarize the neuropsychological complexities of Cortical and Subcortical Dementias in the form of a comparative analysis.

As is well known, subcortical structures including the basal ganglia, diencephalon (thalamus and hypothalamus), mesencephalon (midbrain), and cerebellum have physiologically well established roles in arousal, attention, mood, motivation, memory, abstraction, and visuospatial skills. These areas can be affected in a wide spectrum of diseases including Parkinson's disease, Progressive supranuclear palsy, Huntington's chorea, hydrocephalus, and the AIDS dementia complex. Additionally, depression may produce a picture similar to subcortical dementia [2].

Characteristic symptoms of subcortical typically include forgetfulness, slowing of thought processes, mild intellectual impairment, apathy, inertia, depression (sometimes with irritability), and the inability to manipulate knowledge [3,2,4]. As a rule, this condition is associated with minimal memory loss and absence of aphasia, apraxia, or agnosia. These features lead to marked psychosocial incompetence.

On the other hand, Alzheimer's disease is held to be the classic example of a cortical dementia. The disease progresses as a picture of slow cognitive deterioration over many years. Characteristic of this disease is the diffuse cortical atrophy out of proportion of the age. Further through this review, we will focus on each of the individual neuropsychological components in a comparative analysis pattern between cortical and subcortical dementias.

## **CORTICAL VS SUBCORTICAL DEMENTIA- A SYMPTOM PROFILE**

Before we take a look at the neuropsychological differences between cortical and subcortical dementias, it is important to understand the difference in clinical presentation of these disorders. Clinically, cortical dementia syndromes exhibit symptoms suggestive of abnormalities of cortical functions including aphasia, amnesia, agnosia, and apraxia. Frontal-subcortical dementias on the other hand manifest as a very distinct clinical syndrome with predominant clinical features of bradyphrenia, problems in recall, poor abstraction and strategy formation. Additionally, subcortical dementias also have mood disorders [4,5].

Frontal-subcortical systems mediate motivation, socially responsiveness and empathic behavior, as well as a plethora of executive functions. Dysfunction of this circuitry manifests with executive impairment, apathy, and disinhibition [6,7]. Especially, noteworthy is the symptom of apathy which is evident in patients with several subcortical syndromes like progressive supranuclear palsy, corticobasal degeneration [7,8] and Huntington chorea [9]. Other important behavioral abnormalities like repetitive and compulsive behavior occur in some patients suffering from subcortical dementia, especially with progressive supranuclear palsy [10] and tauopathies [11,12].

### **Memory**

Memory has been the most studied neuropsychological domain in both cortical and subcortical dementias. Memory dysfunctions occurs in both syndromes, but studies suggest that the nature of the memory impairment differs in cortical and frontal-subcortical dementias. Several distinguishing features in the profile of memory loss in the two syndromes of cortical and subcortical dementias have been identified and elucidated [5,13,14]. In most of these descriptions, AD has been considered as the classical cortical model for dementia and for subcortical dementias Parkinsonism and Huntingtons disease have been considered.

In both disorders, spontaneous recall is impaired. However, the etiology of this symptom is different in both of these disorders. In AD, the recall abnormality is due to a failure to encode the information properly or a very rapid decay of memory consolidation.

More severe deficit in Alzheimer's Disease is on the measures of delayed recall (i.e., have abnormally rapid forgetting). Subjects show poor performance on both absolute delayed recall scores or "savings" scores (i.e., amount recalled after the delay divided by the amount recalled on the immediate learning [15]. This inability to recall information after delay persists even after the use of recognition testing [16].

They are also unable to improve episodic memory by using semantic encoding [17]. In addition AD patients exhibit attenuation of the primacy effect (i.e., recall of words from the beginning of a list) indicating towards difficulty in transferring information from primary memory to secondary memory [18]. Cortical Dementias were initially considered to be associated with accelerated forgetting [19]. However this criteria cannot be consistently used for distinction between cortical and subcortical dementias which has been proved in subsequent research [20,21]. Subcortical disorders also produce a deficit in spontaneous recall, but encoding and storage are largely preserved. Additionally, the recollection is aided by several techniques like embedding, recognition formats, and priming. Finally, the skill of procedural memory remains relatively preserved in AD as compared to the frontal-subcortical dementias.

In early AD, usually the remote memory exhibits a temporal gradient such that there is a partial sparing of early-life memories. On the other hand, subcortical dementias show a lesser impairment of remote memory with almost nil temporal gradient. On both verbal and non verbal memory tests intrusion errors (i.e. inclusion of information encoded in the past while attempting to recall freshly given facts) are often seen in Alzheimer's Disease which could be attributed to diminished inhibitory processes or enhanced vulnerability to interference [22,23]. An interesting finding in such patients is the loss of incidental memory for contextual details for the tests (eg, if the tester was a male or a female, whether the testing was done in the morning or afternoon, etc).

Further there is an evident impairment in procedural (perceptomotor) learning in Subcortical Dementias like Parkinson's Dementia and Huntington's disease as demonstrated on Tower of Hanoi task [24]. However this impairment is also not uniform across all types of perceptomotor learning. For example, subjects with Parkinson's disease may have deficits in habit/skill learning but intact learning in tasks like learning of dot pattern prototype and artificial grammar [25].

Further, a unique dissociation in procedural memory in subcortical dementia like Huntington's Disease can be found wherein the impaired sequence learning on Rotary Pursuit tasks, impaired performance on Symbol Digit Modalities Test and less affected recognition than recall on the California Verbal Learning Test may occur alongside normal perceptual-motor integration learning on Mirror Tracing tasks [26].

Among the implicit memory tasks, priming has received considerable attention for differentiating the forms of Dementia. Patients with AD do not exhibit priming effects (eg, do not produce specific responses to partial clues after prior exposure to stimuli containing the clues) and do not exhibit incidental memory for contextual details as mentioned above. Such patients show better responses in both Pictorial [27] as well as in non-pictorial word priming [28].

The pattern of memory loss is another important parameter for differentiating cortical and subcortical forms of dementias. In AD, severe retrograde amnesia with a marked temporal gradient is often found to be co existing with a general semantic memory deficit that arises from damage to cortical association areas.

Current literature indicates that the evolution of memory impairment in Alzheimer's Dementia is in the form of a temporal gradient resulting in extensive and progressive loss of remote memory [29] especially autobiographical memory rather than memory of famous events [30]. On the other hand, the pattern of memory loss in subcortical dementias is much more diverse. The subcortical dementia syndrome is characterized by a relatively mild retrograde amnesia that equally affects all time periods because here there is faulty retrieval of successfully stored information. This also explains the neuropsychological finding of preferential cueing benefit for patients with HD or HIV-associated dementia compared with patients with AD. In Huntington's Disease there may be flat uniform loss of remote memory across entire past life while in

Parkinson's Dementia there can be severe loss of remote memory, even in patients with Parkinson's Disease without dementia there is difficulty in dating past events [31]. The memory decline in Parkinson's Dementia is more rapid than Alzheimer's Disease as elicited on tasks like Boston naming test [32] and Selective Reminding test [33].

Unlike cortical dementias, learning impairment in Subcortical Dementia is amenable to correction and can be partially addressed by provision of richer, more salient cues that aid recognition [34].

### **Language**

Impairments in the domain of language are particularly seen in Cortical forms of dementia. Patients with AD exhibit several progressive language impairment, beginning with anomia and progressing to transcortical sensory aphasia before deteriorating into a terminal language syndrome dominated by logoclonia, echolalia and palilalia [35,36]. Aphasia occurs early in the course of cortical dementias, such as AD, whereas no distinctive aphasia syndromes have been delineated in the frontal-subcortical dementias. Frontal-subcortical systems mediate motivation, socially responsive and empathic behavior, and executive functions. Subcortical disorders produce a deficit in spontaneous recall, but encoding and storage are largely preserved, and recollection is aided by embedding, recognition formats, and priming. Patients in the late stages of their illnesses may exhibit difficulties with naming or following auditory commands [4,5].

Studies comparing patients with AD with equally demented Parkinsonism patients revealed that the AD patients exhibited more severe anomia and had less information content in spontaneous speech, whereas the Parkinson disease patients had grammatically simplified utterances with prominent dysarthria [37,38].

Neuropsychologically, AD patients perform worse on category related tasks as compared to letter fluency tasks, whereas Huntington chorea patients show the reverse pattern or comparable impairment on both tasks. Whereas AD patients produce more semantic pairing defects, Huntington chorea patients present with more of phonemic pairings defects, indicating selective semantic and phonemic processing deficits in AD and Huntington chorea patients, respectively [39]. Similar results were found in progressive supranuclear palsy, corticobasal degeneration, and multiple system atrophy as compared with AD by Lishman [40].

AD has also been characterized by deficits on tests of confrontation naming, verbal fluency, and semantic categorization and on tests to recall over learned facts [41, 42]. An important feature of Language deficits in Alzheimer's Dementia is that they are found almost universally in conjunction with other cognitive and visuospatial deficits and rather than in isolation. In contrast isolated language deficits are seen frequently in cortical encephalopathies like Progressive nonfluent aphasia and Semantic dementia [43]. Progressive fluent aphasia is characterized by isolated progressive decline in language production with intact comprehension while Semantic Dementia is characterized by deficit in understanding the meaning of words, and of face and object identity [44,45] which manifests as fluent, effortless and grammatically correct speech lacking any content with anomia and impaired comprehension of spoken and written words.

An interesting finding was reported by Reber & Squire [46] where they tested grammar related non-declarative functions in patients suffering from Parkinson's Disease. 13 patients with PD were tested on artificial grammar learning, artificial grammar learning with transfer to novel letter sets, and prototype learning. Patients with PD performed similarly to controls on all 3 tests. The intact learning exhibited by PD patients on these tests suggests that nondeclarative cognitive skill learning is not a single entity supported by the neostriatum.

### **Executive Functions**

Executive function involves frontal lobes and related subcortical structures. Thus it seems logical that executive function impairments should be found in both Cortical and Subcortical dementias. Alzheimer's Disease is characterized by impaired executive functions especially those responsible for concurrent mental manipulation of information, set shifting, self-monitoring, or sequencing [47] concept formation, problem solving (Tower of London puzzle) [48] and the performance on modified Wisconsin Card Sorting Task [49] and cue-directed behavior [50]. Further they also show impaired performance on tests of relational integration [51], the Porteus Maze Task, Part B of the Trail-Making Test, and the Raven Progressive Matrices Task [52].

Elementary calculation skills are impaired earlier in the course of cortical than frontal-subcortical dementias, although development of strategies for the resolution of complex problems may be affected early in the course of either disease. Tests designed to assess the integrity of functions of frontal systems indicate that these are differentially impaired in cortical and frontal-subcortical dementias. Furthermore, the patients with frontal-subcortical dementias such as progressive supranuclear palsy perform poorly on the mental control tasks, than for patients with AD even when the degrees of dementia are comparable between the two [53]. Although both AD and Parkinson disease patients perform poorly on delayed response tests, the performance on delayed alternation tasks are only impaired in the AD patients [54, 55].

### **Other cognitive functions**

The visuospatial deficits in Subcortical dementias are heterogeneous which perhaps reflects the heterogeneity of structures involved in various subcortical dementias. Bak et al [56] found that Visuospatial functions are intact in Multi System Atrophy, mildly impaired in subjects with Progressive Supranuclear Palsy and markedly impaired in subjects with cortico basilar degeneration with spatial tasks more impaired than object based tasks. Performance on object based, but not spatial, tasks was related to general cognitive status. In addition, AD patients face more difficulty performing complex constructional tasks which is an indicative of problems in coding of allocentric space (object to object distance), whereas it is the egocentric space encoding which seems to be disordered in patients with frontal-subcortical dementia syndromes (such as Huntington chorea) as evidenced by impaired performances on tasks such as Road map test (eg, the Road Map Test) [57].

Slowing of cognitive processing is difficult to quantify and distinguish from motor retardation, but bradyphrenia seems to be more prominent in the frontal-subcortical dementia syndromes than in AD [58, 59].

### **NEUROBIOLOGICAL UNDERPINNINGS OF CORTICAL AND SUBCORTICAL DEMENTIAS**

In Alzheimer's disease senile plaques and neurofibrillary tangles populate the cortex and there is generalized cortical atrophy, especially of the frontal and temporal lobes, with neuronal degeneration affecting particularly the outer three layers. The typical clinical findings include dyscalculia, dysphasias, dyspraxias and agnosias, and are said to be indicative of cortical dysfunction. However, features reflecting subcortical pathology, such as mild extrapyramidal signs, are also fairly common.

Diseases exhibiting the subcortical dementia syndrome include the degenerative extrapyramidal syndromes, subcortical infarctions, Multiple sclerosis, acquired immunodeficiency syndrome (AIDS), thalamic degenerative disorders, and subcortical trauma, inflammatory processes, and neoplasms. In the subcortical dementias, the lesions occur predominantly in the basal ganglia, the brainstem nuclei and the cerebellum [37]. The lesions producing the subcortical dementia syndrome involve a multitude of axial structures including the rostral brainstem, thalamus, basal ganglia, and frontal lobe projections of these subcortical structures. Given the caudate nucleus's intimate connections with prefrontal cortex, its atrophy could disrupt the connections between caudate-prefrontal loops and neuropsychological alterations emerge in patients with no demonstrable prefrontal lesions. Cognitive decline in such patients has been associated more closely with severity of motor symptoms than duration of the disease [63]. There have been many studies of procedural (perceptuomotor) learning in Parkinson's disease and Huntington's dementia. Saint-Cyr et al [64] found that in both conditions patients were impaired at the Tower of Hanoi task, whereas amnesic patients performed normally (on the basis of the latter's intact procedural memory).

Frontal/executive function, as evidenced by difficulties with verbal fluency, set shifting, categorisation and planning, is also disturbed in all the major subcortical diseases [65,66]. It is also impaired in early Alzheimer's dementia. However, this distinction does not hold good on detailed neuropsychological analysis of the patterns of learning and forgetting in, for example, Huntington's disease and Alzheimer's dementia [67]. and Kuzis et al [68] could not demonstrate a different profile of memory deficits between patients with Alzheimer's dementia and those with dementia consequent upon Parkinson's disease. In remote memory, there are variable patterns of impairment. In Alzheimer's dementia a gentle 'temporal gradient' (an extensive remote memory loss with some degree or relative sparing of early memories) is found in many studies [69], although there have been claims that this is specific for autobiographical memories as opposed to memories of famous events [70], whereas in Huntington's disease there is a 'flat', uniform loss of remote memories across all earlier periods.



As mentioned above Reber & Squire [25] have demonstrated that non-declarative skill learning is not a single entity: patients with Parkinson's are impaired at 'habit' learning, implicating the neostriatum. However, at the same time, they show intact learning of artificial grammars and dot pattern prototypes, which were postulated to reflect brain regions outside both the neostriatum and the medial temporal lobes.

## **CONCLUSION**

As summarized by Cummings [5], several differences exist between neuropsychological test performances of patients with cortical or subcortical dementias. These differences are seen in the domains of Memory, executive functions, language, visuosperceptive functions among others. For patients with cortical dementias, these procedural variations dont improve the performance significantly. The proposed reason for these differential effects of the test format is that in subcortical dementia the main difficulty is in retrieving information while in cortical dementia encoding or storage (or both) are thought to be defective.

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