

Critical Evaluation of the Evidence Derived from the Single Case Studies of Cognitively or Neurologically Impaired Individuals in the Concept of Memory Impairment

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Abstract

Cognitive dysfunction is a wide concept to explain a broad range of brain dysfunctions associating to the capability of an individual to react to emotions, think, concentrate, problem solve, reason, formulate ideas, remember etc. The basic aim of this article is the critical evaluation of the evidence resulting from cognitively damaged patients by single case studies especially on memory impairment.

Keywords: Cognitive; Memory Impairment; Dysfunction

Introduction

The cognitive psychological studies on the base of memory task in individuals with injuries restricted to the medial temporal lobes especially indicate a disproportionate damage in autobiographical episodic recall over semantic memory. There is a number of evidence that explained where lesion is relatively restricted to more lateral temporal cortex (e.g. progression of semantic dementia), the repeat pattern of dysfunction is noticed [1]. As an example of herpes encephalitis, where in most of the cases medial temporal lobe structures are mainly affected, typically also implicates basal forebrain structures and orbitofrontal - although the degree to which these areas intensify the level of memory dysfunction is not clear [2].

However, since last 20 years there has been the clinical view that episodic memory processing is comparatively intact in the frontotemporal dementia syndrome. Particularly, patients with the subcategories of behavioural form frontotemporal dementia and reformist non-fluent aphasia are stated to operate within normal range on standard memory tests. The third clinical presentation of frontotemporal dementia explained that, semantic dementia was comparatively intact episodic memory against a considerably impaired semantic memory was considered as the trademark [3]. Therefore, the comparative intactness of episodic memory came out to be a utilitarian diagnostic marker to differentiate between early frontotemporal dementia and Alzheimer's disease, in which early episodic memory dysfunction continues the most common clinical feature [4]. A complex feature of semantic dementia appears with conservation of some elements of episodic memory, particularly recall of recent autobiographical events and recognition-based visual memory. Current neuropsychological facts of retrograde and anterograde memory in light of neuro pathological and neuroimaging findings, supporting participation of medial temporal structures in frontotemporal dementia, structures known to be vital for episodic memory processing [3].

Scoville and Milner [5] explained anterograde memory in the notorious case of HM, which showed clear differences between the other intellectual functions and memory. The results demonstrated the severe anterograde amnesia, for material come across post injury, could arise in spite of relatively preserved intelligence, reasoning ability and language. A sharp division was drawn between short and long term memory which stated that: dense amnesia for on-going events could happen irrespective of normal capability to hold in mind, and make use of, information associated in on-going activity. The final conclusion drawn was that not all expressions of long-term memory were

damaged in HM. HM was able to learning skills and obtaining perceptual information unconsciously from the situations in which he was unaware he had learned something. This kind of pattern of selective memory dysfunction was principally notable in that it appeared as a result of a comparatively circumscribed lesion of the medial temporal lobes [5].

Rosenbaum., *et al.* [6] involved in a research of amnesic patients. Rosenbaum explained that the amnesic patient KC, due to a motorcycle accident had large bilateral hippocampal lesions. Patient KC had preserved good general information about world, including self-knowledge, but was not capable of recognising any incident he had personally experienced. This kind of exhibition of `episodic amnesia` by patient KC, which include a whole life time of personal knowledge, measured the prospect of amnesic patients learning knowledge that is more difficult than knowledge obtained with priming. One study explained the ability of KC to learn vocabulary related with performing a computer while further studies, involving computer use, showed that KC could actually learn commands to a level where he was able to operate the basic programming job. However, when enquired some general questions about these jobs patient KC was not able to give details about the reason behind them or the new knowledge obtained about computers. Likewise KC could not operate the same programming jobs if there were slightly change in the command instructions from the original instructions which he had learned. These results described the detail that only a simple stimulus response association had been made and that KC did not actually acquire the reasoning behind the knowledge he had attained. Knowledge loss and dysfunction of the processes that permit the combination of knowledge to establish an interrelated memory were both ended to be associated in the amnesia of patient KC. Despite this KC's case explained that acute anterograde amnesia does not restrict individuals from maintaining information that is more difficult than the knowledge learned from priming. When impaired performance is showed by patient KC it is hard to draw any fixed statement about certain impaired regions that may be responsible for the dysfunction. Conversely, when KC shows conserved memory on a specific task then it is plausible to infer that none of the impaired regions are necessary for the functioning of that task. Thus conservation of memory in KC is likely to be more enlightening from a neurological viewpoint than impairment of memory.

In addition, a latest case study suggests that an impairment of person-specific semantics may be noticeable comparatively early in dementia of Alzheimer's type (DAT). This brings the question that whether this damage may also typify those patients belonging to the not well defined mild cognitive impairment group (MCI) [7,8]. Transient epileptic amnesia is a type of temporal lobe epilepsy in which patients mostly complain about irrevocable loss of remote memories. One study explained that by using a wide range of memory tests to illuminate the nature and extent of the remote memory dysfunctions in transient epileptic amnesia patients. Standard tests of anterograde memory showed normal performance. On the contrary, there was a severe memory impairment extend for autobiographical events expanding across the entire lifespan, supporting the occurrence of `focal retrograde amnesia` in transient epileptic amnesia. There was mild personal semantic memory impairment, mostly pronounced in midlife years. In recent decades there were very limited public semantic memory impairments. These findings may explain that subtle structural pathology in the medial temporal lobes or the effects of the propagation of epileptiform activity through the network of brain regions accountable for long-term memory, or the interconnections of these two mechanisms [9].

Moreover, Down syndrome patients (DS) tend to have damaged short-term verbal memory (STM), which continues after providing the visual support for verbal tasks. In this kind of memory dysfunction case study explained that visuospatial support progress verbal STM in DS patients [10]. Latest case studies showed that this might indeed be the case. According to Laws [11] visual STM is only minimally conserved in DS patients, and conversed how the unimpaired Corsi span functioning found among the patients. It might be the reason of the spatial element of the task, rather than the visual one. In addition, there are signs that the visuospatial ability conserved in DS patients is their capability of spatial sequencing [12].

Facts for the dissociability of semantic memory and episodic memory have been explained by neuropsychological case studies of brain damage patients and functional neuroimaging studies of normal individuals [1]. According to Hirst., *et al.* [13] normal individuals and amnesia patients' memory functioning is qualitatively different and with the facts of this study it can be stated that in amnesia, functioning

relies not only on true recollection but most importantly on familiarity judgements. Gamaldo [14] studied whether changeability of time in cognitive functioning differs between cognitively healthy participants and patients who gradually develop dementia. The findings showed that in comparison to healthy participants, patients diagnosed with dementia had greater inconsistency on measures of language, executive function, attention, and semantic memory minimum 5 years before the approximated onset of cognitive dysfunction, which may be a sign of maladaptive cognitive performance. However, in dementia cases had less inconsistency on visual memory than the healthy group which may indicate that these cases had more difficult to learning [14].

Moreover a functional neuroimaging time course (MRI scans- T1, T2, T3) study with two patients of semantic dementia described about recurrence behavioural functioning, locked to scans, time, across a various language, background and semantic cognitive test [15]. Findings from patient EK revealed that there was restriction by the atrophy at T1 to those regions mainly related with object processing. The following scans also revealed gradual association of inferior frontal regions and posterior perisylvian, insular commonly related with language performance. Patient BS however, showed a restriction by the atrophy at T1 to the right hemisphere's object processing areas. When compare to EK, a less and slower wide development of atrophy was noticed across following time points. At T3, the posterior perisylvian language areas as well as the inferior frontal cortex remained comparatively intact, though there was inadequate connection of posterior insular cortex bilaterally. This described the detail that the disorder in EK was in more advance stage at onset of the study (T1) or by distinctions in amount of atrophy among patients [15].

A vigorous relation between neocortical brain regions and medial-temporal lobe has been suggested to account for memory's outstanding functioning with time in those are developing dementia. Woodard [16], demonstrates by an fMRI study, robust and temporally graded signal variations in posterior cingulate, right fusiform, left middle frontal, and right middle temporal regions in normal participants during familiar name recognition from two unequal time epochs. Especially, no neocortical brain regions demonstrated greater reaction to older than to recent stimuli. The findings of the study suggests that a possible involvement of these neocortical brain regions in temporally dating items in memory and in maintaining and establishing memory traces throughout the lifespan [16].

In addition, neurologically healthy individuals by functional neuroimaging studies showed a bilateral network or left adjacent neural participation during the recall of autobiographical event [2]. According to Aggleton and Shaw (1996) recall memory terribly affected by the impairment of the 'extended hippocampal circuit' but it does not damage recognition memory. Conversely, intermix impairment to parahippocampal gyrus and hippocampal is essential to create familiarity insufficiency (recognition). Squire [17] demonstrated that even after restricted hippocampal lesions, recognition damages were regularly noticed,

Kopelman, *et al.* [18] explained that there is no evidence for inconsistent recall dysfunction in the patients with memory dysfunctions following focal diencephalic or temporal and focal frontal lobe lesions. Conversely, in their MRI study they noticed that hippocampal volumes are the crucial volume involves recognition and recall functioning. Literature on patients with Korsakoff syndrome specifies a further important part for the frontal cortex and diencephalic structures in amnesia. Few facts showed that they have unbalanced retrieval comparative to storage arrears, indicating that memories were stored reasonably well but retrieval functioning were impaired [19].

Conclusion

The current essay discussed the features of deficits and lesion profile of various patients with memory impairment. The above discussed studies suggest that frequent description of comparative sparing for early memories. However, it is improbable that temporal gradient purely attributed to semantic memory and there's a greater need for control over neuroimaging techniques and methods for better understanding. So, it can be stated that the detailed analysis of various cases of behavioural discrepancy and development of disorders can provide a strong structure for integration challenging claims about the crucial and vital dysfunctions in memory.

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