

Is focal retrograde
amnesia is an actual
neurological
disorder?



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Wheeler & McMillan (2001) describe Focal Retrograde Amnesia (FRA) as a rare but recently recognized condition. According to Sehm et al (2011), FRA is characterized by a substantial loss of retrograde memory, often contrasted with the normal acquisition of new information. Several case studies have attributed Focal Retrograde Amnesia to various neurological disorders such as encephalitis, traumatic brain injury, hypoxia and epilepsy (Milton et al, 2010). Milton et al (2010) note that the size and localization of lesions associated with the syndrome do vary, and include neocortical, limbic and brainstem structures. However, considerable scientific evidence has shown that frontal, medial temporal and temporopolar cortices play a major role in remote memory functions and should, therefore, be involved in the condition's pathophysiology (Sehm et al (2011). Based on scientific studies, controversy has existed regarding as to whether Focal Retrograde Amnesia (FRA) can be classified as a pure neurological disorder. This paper seeks to determine whether FRA is an actual neurological disorder.

FRA was initially considered a trifling psychiatric disorder and, hence barely researched from a neurological perspective. The first comprehensive empirical study of a patient with the condition was published in 1980. This particular study described the case of a 64-year-old man who manifested with a protracted transient global amnesia (TGA)-like episode, with a lasting focal retrograde amnesia. It is such a monumental observation that triggered a search for similar cases in an attempt to define the neuropsychological profile of FRA as well as to ascertain its clinical-anatomical correlations. Subsequently, FRA aroused some considerable interest of neurologists and neuropsychologists, particularly when it became apparent that it was

associated with overt brain damage, including severe head injury, cerebrovascular complications, encephalitis, and severe cerebral hypoxia (Stracciari, Fonti & Guarino, 2008). Several studies have also associated FRA with a diversity of heterogeneous conditions, including seizures, cerebral headache, trivial head trauma and drug intoxication (Manes et al, 2001). Such observations have favoured the idea that FRA is a syndrome. However, cases of The observation of Focal Retrograde Amnesia being witnessed in the progression of acute emotional stress, as well as in the absence of obvious causal or triggering factors, always associated with normal ancillary investigations, has led some to reconsider a psychogenic explanation – at least for such cases (Brandt & Van Gorp, 2006). This has further complicated the pathophysiological classification of the condition. Following these observations, Kopelman (2000) is known to have suggested that both organic and psychogenic factors interactively contribute in the manifestation of FRA.

According to Kopelman (2000), classifying focal retrograde amnesia as a neurological phenomenon is founded on some shaky grounds. In addition, the manner in which the idea that FRA is an actual neurological disorder has become so commonly accepted shows potential vulnerabilities both in the interpretation of individual patients' clinical histories and in the ascription of causality in neuropsychology. There are, however, some aspects of retrograde amnesia (RA) on which there appears to be general agreement that focal retrograde amnesia is both attributable to organic and psychogenic factors and, thus not purely a neurological disorder.

For example, according to Kopelman (2000), various studies have consistently shown that relative sparing of early memories take place in organic amnesia and dementia and that there is inconsistency in the slopes of the ensuing temporal gradient curves both between individual patients and between patient groups. However, the debate concerning the underlying basis of these temporal gradients still persists, with many scholars arguing either that early memories may have gained a more semantic structure or that they have become fully consolidated, hence providing relative protection against the negative effects of focal retrograde amnesia. In addition, recent memories may have been scantily programmed during the progressive onset of the condition.

Secondly, various studies have agreed that there can be a discrepancy on the involvement of autobiographical and more semantic elements of remote memory. For example, studies have cited various cases of patients with left temporal lobe pathology, who present with acute loss of semantic knowledge, as measured using such aspects as semantic classification, and memory of news events and famous faces from the recent and more distant past, but still with memory for autobiographical information. On the contrary, some studies have described patients with right-hemisphere pathology, who present with disproportionate loss of autobiographical memories, relative to semantic knowledge. In addition, inconsistent impairment of autobiographical information and other sporadic memories in right- as compared to left-sided lesions has also been established in various studies (Kopelman, 2000).

Similarly, most studies have agreed that pathology in either the frontal lobes or the temporal lobes can contribute to focal retrograde amnesia. Some of the patients classified as focal retrograde amnesia patients have either presented with either left or right temporal lobe pathology, making it difficult to establish with precision whether the condition is as a result of organic or psychogenic factors. In addition, various studies have described patients with focal frontal lesions, who presented with remote memory impairment (Kapur, 2000).

Still, on the same note, studies have shown a poor connection between scores on anterograde and retrograde memory tests. This observation has been illustrated in Korsakoff patients, in a mixed group of amnesic patients, and in Alzheimer patients. Furthermore, it is universally accepted that people can present with anterograde amnesia with minimal or no retrograde amnesia, especially in some cases of transient global amnesia, basal forebrain lesions, mild head injury, and in some cases of deep midline tumours (Kopelman, 2000). However, the debatable subject has been whether retrograde amnesia can occur in the absence of anterograde amnesia, and if so, under what situation.

To determine whether focal retrograde amnesia is an actual neurological disorder, it is advisable to look at the mechanisms and outcomes associated with the condition. It is of particular significance to distinguish pragmatic outcome statements from assumed mechanism statements, whether this is the role of retrieval processes in semantic dementia patients, or differential rates of recovery after severe head injury (Kopelman, 2000). The principal issue of discussion here is whether certain patients meet the criteria for FRA. <https://assignbuster.com/is-focal-retrograde-amnesia-is-an-actual-neurological-disorder/>

Kopelman's main claim is that different accounts may be possible for the patterns of memory performance that arise in individuals who present with semantic dementia. According to Kapur (2000), while such varied accounts are possible, this does not detract from the fundamental pattern of more consolidated recent memories as compared to remote memories that have been established in some studies. Some studies have provided autobiographical memory data on patients, showing clear time-specific discrepancies in retrieving autobiographical memories, using the same set of prompts, which makes it unlikely that the better retrieval of recent and less consolidated memories are primarily due to poor conceptual skills. While Kopelman has shown that differential recovery mechanisms are critical, current studies with FRA patients have shown that such a claim confuses pragmatic outcome with the assumed mechanism. Kopelman (2000) however notes that such claims should be supported by empirical data on particular patients in order to prove that he/she really has a focal retrograde amnesia (Kapur, 2000).

Another way of determining whether focal retrograde amnesia is an actual neurological disorder is to evaluate its causality. Kopelman (2000) has dealt with scientific and theoretical issues that relate to the attribution of causality in organic and psychogenic amnesia, comprehensively analyzing the complex interfaces that occur between the two. He has, however, cautioned against the adoption of the psychogenic-organic distinction, arguing that such an approach is largely redundant. Consequently, Kopelman proposes the adoption of another neutral approach of functional amnesia. However, with his proposition, studies have noted a considerable overlap between the

areas of pathology as established in various studies of focal retrograde amnesia, as well as the similar areas of anomalous activation in functional imaging of functional or psychogenic memory loss. Based on such evidence, it, therefore, remains plausible that in some cases of FRA there is a possibility of dual contribution to the retrograde memory loss. It also further shows that such retrograde memory deficits are as a result of overlapping brain regions and that the metabolic element of the brain disorder arising from psychosocial factors may interact with the structural lesion to lead to apparently irreversible changes to the overall brain integrity in the affected region (Kapur, 2000).

Studies have shown that some, but not all, cases of severe autobiographical amnesia are associated with very poor anterograde memory and that such cases cannot be classified as cases of focal retrograde amnesia. While this is true, it is salient to note that verbal anterograde memory is typically not “very poor” and contradicts autobiographical memory, which could be termed as “very poor”. He also seems to conclude that not all forms of focal retrograde amnesia are similar and that different explanations should be considered before attributing focal retrograde amnesia to particular sites of pathology (Kapur, 2000). The point has also been made in various studies that because a focal retrograde amnesia may be subsequent to a certain cause, the outcome is by some means faulted (Kopelman, 2000).

Based on the studies related to focal retrograde amnesia, it can now be concluded that answering the question as to whether focal retrograde is an actual neurological disorder should revolve around how one defines the condition. The ultimate contradictions are, however, those where <https://assignbuster.com/is-focal-retrograde-amnesia-is-an-actual-neurological-disorder/>

assessments of retrograde and postgrade memory performance are founded on tasks that are strongly matched on all relevant variables such as memory domains, the complexity of a task, response demands, et cetera.

When focal retrograde amnesia is defined as noticeably abnormal functioning on normal retrograde memory tests, including such items like public events, regular faces and comparatively compromised performance on normal anterograde memory tests, it would be acceptable to conclude that FRA is actually a neurological condition (Kapur, 2000). This conclusion allows for the possibility that that in certain situations, enduring anterograde memory deficits may be impaired. Similarly, when focal retrograde amnesia is defined as a period of autobiographical memory deficit for a duration ranging from months to a few years, accompanied with excellent recovery of anterograde memory, it would also be concluded that FRA is an actual neurological disorder. Still, if the condition was defined as momentary loss of knowledge, accompanied with relatively consistent and continuous episodic memory, it will be argued that FRA is a true neurological disorder.

However, when focal retrograde focal amnesia is defined as a severe, temporally ungraded and temporally extensive autobiographical retrograde amnesia, in the context of standard or near-standard functioning on normal anterograde memory tests, it will be difficult to establish with certainty whether or not the condition is actually a neurological disorder (Kapur, 2000). While there are various case scenarios with such pattern of memory performance that have been diagnosed as exclusively neurological but which may be either fully or partly psychological in origin, there exist no well-documented cases satisfying the strict psychiatric criteria for the focal

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retrograde amnesia to be classified as an actual neurological disorder. Similarly, it is difficult to ascertain whether focal retrograde amnesia is an actual neurological disorder when it is defined as uneven retrograde to postgrade memory functioning, where the spheres of memory performance, testing methods, time periods sampled, and all other crucial variables are as similar as it is practically possible. This is in consideration that various studies have postulated that there exist distinctive neural networks that play a role in memory formation and retrieval for certain memories that are otherwise comparable apart from varying in terms of their temporal association to the onset of cerebral pathology. Considerable evidence also exists, suggesting that the brain has the capacity to incorporate adequate flexibility to allow for comparable patterns of memory functioning to arise, further complicating the classification of focal retrograde amnesia as either due to organic or psychogenic factors.

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