Episodic memory in transient global amnesia

Once seen, the syndrome of transient global amnesia (TGA) is never forgotten. Patients abruptly discover that they are unable to recall recent events. Their perplexity is deepened by an inability to retain new information. This leads to repetitive but futile questioning of their companions. Attacks typically last about 4 hours. By the next day patients report that their memory is back to normal—with the exception of a dense amnesia for the attack itself. The paper by Eustache et al (this issue, pp 148–54) investigates the mechanism of this amnesia: is it due to failure to acquire, to store, or to retrieve information about events during the episode of TGA?

This work adds to an impressive body of recent research. Indeed, if TGA had been the only amnesic syndrome available for study it would have allowed us to build up a substantial understanding of the mechanisms of memory.

First and foremost, transient global amnesia teaches that memory is an independent module of neuropsychological function: attention, language, reasoning, and visuospatial abilities remain intact during attacks despite the devastation of memory.

Secondly, the characteristics of transient global amnesia indicate that short term or working memory is separable from the long term store. Digit span, for example, is usually normal or near normal in TGA. Thirdly, the nature of the amnesia in TGA hints that mechanisms of anterograde and retrograde memory may be independent: anterograde amnesia is always severe during attacks whereas the retrograde amnesia can extend from days to decades. Fourthly, TGA exemplifies Ribot’s law, that long established memories are relatively unaffected by the retrograde amnesia of TGA. Fifthly, it is now well established that procedural memories, for motor and perceptual skills, can be acquired during episodes of TGA, showing that there must be several parallel routes to memory. Finally, sensitive neuroimaging techniques have pointed the finger of guilt firmly at the medial temporal lobes, underlining the critical importance of these structures in the processing of declarative—as opposed to procedural—memories.

The retrograde amnesia of TGA clearly reflects a reversible problem with memory retrieval. Access to memory for recent events, lost during the attack, is promptly restored once it is over. The work by Eustache et al supports previous suggestions that the anterograde amnesia of TGA manifests a different problem, with memory acquisition and/or storage.

The authors make use of a verbal memory test designed to distinguish three processes involved in handling new information: encoding, storage, and retrieval. The underlying logic of the study is that defects in encoding will be apparent in any subsequent test of declarative memory; defects in storage will affect recall and recognition in equal measure, whereas defects of retrieval should be more evident in tests of recall than in tests of recognition. Of three patients tested during an attack of TGA, one performed poorly on a test of immediate cued recall, indicating a defect in memory encoding, whereas the others performed poorly on tests of delayed recall and recognition, suggesting a defect of storage.

These results from a small number of patients are of interest but do not yet allow firm conclusions. They raise several questions: why should encoding be affected in one case, storage in the others? Does this reflect genuine heterogeneity in the condition? Might differential impairment of retrieval and encoding/storage reflect degrees of dysfunction in a single mechanism—or are memory retrieval and memory acquisition genuinely independent processes? Further work with patients in the midst of their attacks of TGA should help to clarify the neuropsychology of this fertile experiment of nature.

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