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Visual memory loss and autobiographical amnesia: a case study

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Abstract

Amnesia typically results from trauma to the medial temporal regions that coordinate activation among the disparate areas of cortex that represent the information that make up autobiographical memories. We proposed that amnesia should also result from damage to these regions, particularly regions that subserve long-term visual memory [Rubin, D. C., & Greenberg, D. L. (1998). Visual memory-deficit amnesia: A distinct amnesic presentation and etiology. *Proceedings of the National Academy of Sciences of the USA, 95*, 5413–5416]. We previously found 11 such cases in the literature, and all 11 had amnesia. We now present a detailed investigation of one of these patients. M.S. suffers from long-term visual memory loss along with some semantic deficits; he also manifests a severe retrograde amnesia and moderate anterograde amnesia. The presentation of his amnesia differs from that of the typical medial-temporal or lateral-temporal amnesic; we suggest that his visual deficits may be contributing to his autobiographical amnesia.

Keywords: Retrograde; Anterograde; Medial temporal; Encephalitis; Visual memory

1. Introduction

According to the consensus theory of memory, Autobiographical Memory (AM) requires many disparate brain regions (Conway & Pleydell-Pearce, 2000; Damasio, 1989; Fuster, 1995; Kopelman, 2000; Kopelman & Kapur, 2001; Mayes & Roberts, 2001; McClelland, McNaughton, & O'Reilly, 1995; Murre, Graham, & Hodges, 2001; Shastri, 2002; Squire, 1992). The medial temporal lobe (MTL) and diencephalon coordinate encoding of experiences for later recall. They do not, however, represent a memory by themselves; they mediate its representation in sense-specific areas of cortex and areas involved in emotion. Retrieval involves the frontal lobes (Wheeler, Stuss, & Tulving, 1997) and the MTL, which coordinate activation among disparate regions and produce activation patterns similar to the patterns present during the original experience.

MTL trauma tends to cause profound, ungraded anterograde amnesia (AA). The retrograde amnesia (RA) is often temporally graded; older retrograde memories are more likely to be spared than newer retrograde memories (Squire, 1992). In this paper, rather than studying the MTL, we investigate the effects of damage to neocortical regions and the cognitive processes they subserve. Such damage could have several consequences. First, vital portions of the memory could be rendered inaccessible. Second (and more important), such damage could disrupt the cascade of activation required for retrieval. For example, impaired activation in visual cortex could impede activation in non-visual cortices even if non-visual cortices were intact (see O'Connor, Butters, Miliotis, Eslinger, & Cermak, 1992; Ogden, 1993; and Hunkin et al., 1995 for case studies that discuss this idea). Therefore, impaired retrieval of information from neocortex could result in global autobiographical amnesia, not just a simple loss of information within individual memories (Greenberg & Rubin, 2003).

AM relies heavily upon visual imagery (Rubin, 1995). (As used here, "visual imagery" means pictorial or object

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imagery, not linguistic imagery such as the shape of letters or words.) Retrieval of AMs usually coincides with retrieval of a visual image (Brewer, 1995), and the intensity of visual imagery is well correlated with feelings of recollection or reliving (Rubin, Schrauf, & Greenberg, 2003). Therefore, an impairment of visual imagery might have a significant effect on AM.

Farah (1984) suggested that patients with particular visual imagery impairment, specifically an impairment of long-term visual memory, would meet three criteria. First, the patient would be able to copy line drawings, thereby showing that other deficits are not caused by basic perceptual problems. Second, the patient would be unable to recognize objects by sight, defined as an inability to indicate either their names or their functions. Third, the patient would be unable to draw objects from memory, describe their visual properties from memory, or detect a visual image of them upon introspection. The first two criteria identify the patient as an associative visual agnosic; the third criterion demonstrates that the deficit arises from impaired access to long-term visual memory rather than difficulty generating or manipulating images (Farah, 1984). Patients who meet the third criterion but not the first two—those who cannot draw from memory but are not agnosic—have intact recognition memory for visual stimuli. Thus, patients only have a long-term memory deficit if they meet all three criteria.

We previously reviewed case studies of patients who met these criteria (Greenberg and Rubin, 2003; Rubin & Greenberg, 1998). We suggested that long-term visual memory loss should lead to a form of amnesia that would have unique properties. First, these patients should suffer from mild to moderate AA; they could compensate for their deficits by placing greater reliance on non-visual sensory data, as in a case reported by Ogden (1993). Second, these patients should not show sparing of childhood memories. In MTL amnesia, early memories are intact because they are consolidated or because traces of those memories still exist in the remaining parts of the MTL. In this form, though all retrograde memories would be affected, since memories from any age would involve visual information. This pattern of deficits would be exactly opposite to that found in cases of amnesia resulting from MTL damage (Squire, 1992; Kapur, 1997, 1999). In practice, patients might be able to retrieve some visual information, so the RA may not be complete; AA might be moderate because an increased reliance on other modalities might never fully compensate for the inability to encode new visual data. We found 11 such patients, and all 11 had amnesia. Five of eleven had some sign of MTL damage. Seven of these case studies compared the severity of RA and AA; in five of them, retrograde deficits were greater. We called this syndrome visual-memory-deficit amnesia (VMDA), meaning global amnesia arising from a deficit of visual memory.

Our investigation was hindered by the absence of detail in many of the case studies; case studies tend to focus on the exploration of one disorder and may only briefly describe comorbid deficits. Two other case studies met our criteria, but the report of the memory deficits was inconclusive (the first patient was described as being "forgetful" (Goldenberg, 1992) while the second had "memory difficulties" (Wilson & Davidoff, 1992) and a delayed Wechsler score of 0 (Davidoff & Wilson, 1985). Also, these case studies generally do not describe the phenomenological properties of patients' memories. We previously designed a test to probe these properties (Rubin et al., 2003). It uses the Galton–Crovitz cue-word technique to cue a memory and asks the subject to rate its properties (e.g., emotional valence or intensity of visual imagery) on a set of scales. These ratings can be used to see if a patient's memories differ from those of controls.

We combined this questionnaire with standard tests to investigate the memory deficits of M.S., one of the 11 patients previously identified as having VMDA. We attempted to determine whether he met the criteria for a loss of visual memory, and if so whether he suffered from the predicted form of amnesia; we also sought to rule out other causes of his amnesia. We attempted to determine whether his deficits were consistent with VMDA, and thereby examined the role of visual imagery and visual regions in AM.

2. Case history

M.S. has been tested regularly since 1971 (De Haan, Heywood, Young, Edelstyn, & Newcombe, 1995; Heywood, Cowey, & Newcombe, 1991; Mehta, Newcombe, & De Haan, 1992; Newcombe & Ratcliff, 1975; Newcombe, Young, & De Haan, 1989; Ratcliff & Newcombe, 1982; Young, Newcombe, Hellawell, & De Haan, 1989). He is a left-handed Caucasian male with no family history of sinistrality. In 1970, while a 23-year-old police cadet, he suffered a febrile illness with frontal headache and vomiting. He was diagnosed with probable herpes encephalitis; antibody tests were negative, but MRIs taken in 1989 are inconsistent with a vascular etiology (see Heywood et al., 1991). M.S. now presents with a left homonymous hemianopia, but his visual acuity is normal (6/6, N5 for near vision). He also has achromatopsia, associative visual agnosia, and amnesia (for other reports of his visual deficits, see Heywood et al., 1991; Newcombe et al., 1989; Mehta et al., 1992). His linguistic skills are generally excellent, and he has no significant aphasic symptoms; he reads a newspaper and often completes the crossword.

M.S. tends to tell stories multiple times, presumably because he forgets he has told them before. He has a striking ability to remember dates of events even when he recalls few if any details about them. He lives with his mother but can briefly function on his own and has held a job in a remploy factory since 1972.

3. Neuroimaging

MRI showed extensive damage in the occipital and temporal lobes (Heywood et al., 1991). In the left temporal lobe, the