

Visual memory loss and autobiographical amnesia: a case study

Daniel L. Greenberg^{a,*}, Madeline J. Eacott^b, Don Brechin^b, David C. Rubin^a

^a Psychological and Brain Sciences, Duke University, P.O. Box 90086, Durham NC 27708, USA

^b Psychology Department, Science Laboratories, University of Durham, Durham DH1 4NB, UK

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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 subservise 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.

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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 subservise. 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

* Corresponding author. Tel.: +1 919 660 5639; fax: +1 919 660 5726.

E-mail addresses: dan.greenberg@duke.edu (D.L. Greenberg), m.j.eacott@durham.ac.uk (M.J. Eacott).

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 agnostic; 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 agnostic—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, Hallowell, & 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

temporal pole, parahippocampal gyrus, hippocampus, amygdala, and 4th temporal gyrus are destroyed; the 1st, 2nd, and 3rd temporal gyri are generally spared. In the right temporal lobe, the temporal pole is destroyed as well, along with the 2nd, 3rd, and 4th temporal gyri; the anterior parahippocampal gyrus is damaged, but the hippocampus, posterior amygdala, and posterior 1st temporal gyrus are all largely intact. In the left occipital lobe, the cortex is spared, but there is abnormal signal in white matter in infra- and supracalcarine regions. The right occipital lobe is largely destroyed along with the occipitotemporal junction. Although part of the calcarine seems spared, it may be denervated by damage to white matter. Some white matter in the right parietal lobe has been destroyed, but the cortex appears largely intact, as do the frontal lobes.

4. Neuropsychological assessment

On the Mini-Mental Status Exam (Folstein, Folstein, & McHugh, 1975), M.S. scored 29/30. He lost one point for forgetting one of the three words he was given, but correctly named a pencil and a watch. On the WAIS and WAIS-R, M.S.'s verbal IQ is average (101); his mental arithmetic score on the WAIS-R is above average at 14 (Mehta et al., 1992; Newcombe and Ratcliff, 1975). His performance IQ is low with scores of 0 for block design and object assembly (Newcombe and Ratcliff, 1975). On the logical memory part of the Wechsler Memory Scale, M.S. scored 6.5 at immediate recall and 0 after a delay (Newcombe and Ratcliff, 1975). He performs poorly on paired-associate and maze-learning tasks (Newcombe and Ratcliff, 1975). He reads single words correctly, makes correct lexical decisions (Newcombe et al., 1989), and judges absurd sentences normally (46/50; Ratcliff and Newcombe, 1982).

4.1. Visual imagery

4.1.1. Criterion 1: copying from a picture or a model

Fig. 1 shows M.S.'s copy of the drawing of the rhinoceros from the Graded Naming Test. He worked clockwise from

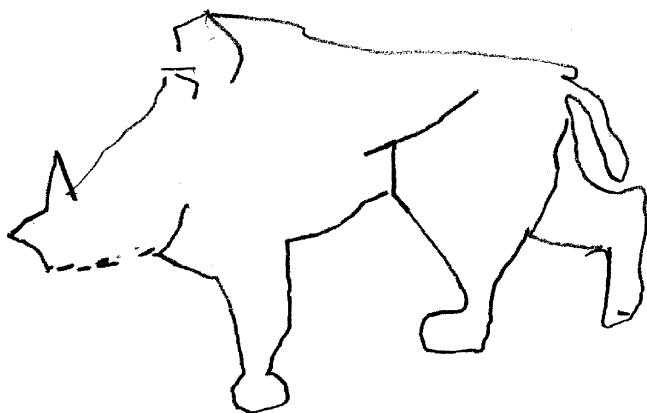


Fig. 1. M.S.'s copy of the drawing of a rhinoceros.

the ear and appeared to use a line-by-line copying strategy. When we tested him with the figure from the Adult Memory and Information Processing Battery (Coughlan & Hollows, 1985), M.S. scored 75% on the copy, 13% on the immediate recall, and 0% on the delayed recall.

4.1.2. Criterion 2: object recognition

M.S. could not recognize the rhinoceros he drew in Fig. 1; he thought it might be a dog. When we presented him with line drawings from the Category-Specific Names Test, M.S. recognized 0/30 fruits and vegetables, 0/30 animals, and 0/15 praxic objects. In past testing, he has identified at most 11 of 36 Oldfield–Wingfield drawings (Ratcliff and Newcombe, 1982). On a spontaneous naming test, we presented M.S. with a series of 30 household objects (keys, coffee cup, pencil, etc.), and could identify only 16; he could describe the function of 1 of the objects he failed to recognize. He has a greater deficit for living items, as do other encephalitis patients (see Barbarotto, Capitani, & Laiacona, 1996, for a review). On a prior forced-choice recognition task, M.S. identified 18/36 living items and 28/36 non-living items, while controls scored 36/36 (Mehta et al., 1992). In daily life, he often makes recognition errors (e.g., mistaking a tripod for a stool).

M.S. also has difficulty with face-recognition tasks. In previous tests, he has scored 33/55 on the Benton Test, 13/24 with changed orientation, and 15/24 with changed lighting, while controls scored 74.4/80 (Newcombe et al., 1989). On a forced-choice face- and name-recognition task, M.S. performed at near-normal levels for name recognition, but was at chance for face recognition (Newcombe et al., 1989).

4.1.3. Criterion 3: drawing from memory

Fig. 2 shows M.S.'s attempts to draw several objects from memory. While his drawings of a tree and a bird are perhaps passable, his drawing of a butterfly lacks wings, and his drawing of a car depicts a box on wheels with doors that are higher than they are wide. These tests also reveal some semantic deficits; on his drawing of a tree, he labeled the crown as the "ruff" [roof] and the trunk as the "stem." In prior tests, he has had trouble drawing recognizable objects (Newcombe and Ratcliff, 1975).

4.1.4. Other imagery tests

We also gave M.S. several self-report tests of visual imagery. In the vividness of visual imagery questionnaire (Marks, 1972, 1973; Richardson, 1994), participants generate images of four scenes; for each scene, they image four components and rate their vividness (5, no image; 1, as clear as normal vision). M.S. scored an average of 4.0 with eyes open (S.D. = 1.10) and 4.7 with eyes closed (S.D. = 0.60); his combined mean was 4.35 (S.D. = 0.93), 3 S.D. below published control means (Pollock & Brown, 1984). He received a score of 5 for four questions because he was unable to generate a visual image of a relative or friend (even when asked



Fig. 2. M.S.'s drawings from memory of a tree, a bird, a butterfly, and a car (l-r).

about specific relatives). The Visual Elaboration Scale asks subjects to generate images of four scenes; for each scene, it asks several yes-or-no questions about the level of detail (Slee, 1980; Richardson, 1994). M.S. scored 3/15; several control groups had means between 8 and 9 and S.D. ranging from 2.9 to 4 (Slee, 1980), so M.S.'s score ranges from 1 to 2 S.D. below the mean. M.S. failed on all questions that asked about color or motion; he also failed on 6 of 8 questions that tapped spatial imagery. The Gordon Test of visual imagery control (Gordon, 1950; Richardson, 1994) asks subjects to generate and modify a visual image of a car. M.S. received a score of 9, putting him at 3 S.D. below the mean (control mean = 20.09, S.D. = 3.72). He responded in the negative to half the questions that asked him to generate an image of movement, and he could neither manipulate a stationary image nor alter its color.

In prior testing, M.S. was verbally presented with the names of three items and was asked to judge which two were the most similar visually. For living objects, M.S. made far more errors than controls; for non-living objects, his performance was normal (Mehta et al., 1992). M.S. also states that he rarely dreams; he cannot recall if he dreamed more frequently before his illness.

4.2. Semantic deficits

M.S. may have semantic deficits in addition to his visual memory deficits. In prior testing, he could recognize only 20/36 objects from verbal descriptions of their functions (Newcombe et al., 1989). When asked to generate exemplars for living and non-living categories, he performs well below normal for living items but is at normal levels for non-living items (Young et al., 1989). He is unable to define some words (e.g. "nightingale"; Ratcliff and Newcombe, 1982). He cannot always tactually identify objects; in prior tests, he identified 6/10 objects by touch (Newcombe and Ratcliff, 1975), and in our tests, he was able to identify by touch only 4 of the 14 household objects that he could not recognize visually. His visual deficits are, however, far more severe than his semantic deficits; as a prior report concluded, "M.S. has a specific difficulty in accessing semantic information from a pictorial route and his agnosia is not totally explained by disturbance of the semantic system itself" (Ratcliff and Newcombe, 1982, p. 163).

4.3. Memory abilities

4.3.1. General world knowledge

M.S. was able to provide accurate definitions for several words that had entered British English after his illness (Euro, AIDS, channel tunnel, millennium bug, poll tax, internet, concorde, ethnic cleansing, global warming, punk, GM (genetically modified) foods). For a few terms, he gave vague answers: "Glasnost" made him think of the leader of Russia; "microwave" was used "for cooking," and "anorexia" was "an illness." He could not define "sleaze," "spin doctor," "Pokemon," or "collateral damage."

4.3.2. Autobiographical memory interview (AMI)

We tested M.S.'s AM using the AMI (Kopelman, Wilson, & Baddeley, 1990), which assesses personal semantic and autobiographical event memory from three life periods (childhood, early adult life, and recent life). The early adult period was split into pre-illness and post-illness segments since M.S.'s illness fell in the middle. On the personal semantic component, M.S. was just below normal on all periods; on the autobiographical component, he was well below normal on all periods (Table 1).

4.3.3. Rubin et al. autobiographical memory questionnaire (AMQ)

We also tested M.S.'s memory using a version of the Galton–Crovitz technique (Rubin et al., 2003). We presented him with a series of cue words and asked him to retrieve an associated memory. Thirty words came from an existing set (Rubin et al., 2003); we added new words after M.S. failed to generate memories to several words.

We also have an interest in exploring the properties of M.S.'s AMs to see if they differ from those of controls. This approach must be regarded as exploratory until it has been used on larger populations of patients. We believe, however, that it sheds light on some interesting aspects of M.S.'s dis-

Table 1
M.S.'s performance on the autobiographical memory interview

	Childhood	Pre-illness	Post-illness	Recent
Semantic	15 (16–21)	14 (17–21)	16 (17–21)	18.5 (19–21)
Autobiographical	1 (6–9)	4 (7–9)	3 (7–9)	2 (7–9)

Note: Normal ranges are listed in parentheses.

Table 2
MS's performance on the Galton–Crovitz task

Condition	Session	Anterograde	Retrograde	No memory	Total words
Unconstrained	1	16	3	11	30
	2	6	0	4	10
	3	6	0	2	8
Total		28	3	17	48
Constrained	1	–	3	17	20
	2	–	9	25	34
	3	–	5	22	27
Total		–	17	64	81

order. Moreover, phenomenological reports are the *only* way to assess properties such as a patient's belief in his memories. Therefore, after M.S. retrieved a memory, we asked him to rate its properties on a series of scales (see Appendix). We used these questions to accomplish several aims: (1) to compare the number and distribution of M.S.'s memories to those of controls; (2) to compare M.S.'s mean ratings to those of controls in an attempt to determine whether his memories have different properties; and (3) to use correlation analyses to see if M.S. bases his metacognitive judgements about his memories on different properties than controls do.

M.S. could perform this task but sometimes found it tiring and frustrating. In each session, we stopped the test when he seemed tired, so the number of trials varied across sessions. Due to time constraints and his tendency to fatigue, we could not ask him to fully describe each memory, so he may have reported a memory more than once.

We conducted three testing sessions about 1 year apart. In each session, M.S. was first asked for memories from any time in his life (unconstrained condition); he was then asked to recall only retrograde memories (constrained condition). For these memories, we used keywords tailored to his early life (e.g. "police" and "school"). Controls have no difficulty with this task; in prior testing, participants were always able to produce at least 28 memories to 30 words (Rubin et al., 2003) and 75 memories to 80 words. However, M.S.'s results indicate a severe and global AM deficit. Table 2 shows the results.

Table 3 presents means and standard deviations for each question; the standard deviations are across control participants. We conducted *t*-tests thresholded at $p < 0.05$ to compare the mean values for his anterograde and retrograde memories on each of these properties. Even using the non-conservative assumption that the memories were independent, the only statistically significant difference was in the rehearsal variable ($t(45) = -2.39$, $p < 0.02$, $\omega^2 = 0.10$). We compared M.S.'s ratings to those of 50 controls who were given 30 of the keywords we presented to M.S. He scored 2 S.D. below the control mean on several variables: mentally traveling back in time, remember-know, real-imagine, see, and setting. M.S.'s rating of importance was 2 S.D. above the mean.

The correlations among his ratings further characterize his deficit. With the exception of three variables, *see*, *talk*, and *in words*, his anterograde correlations are similar to the average correlations calculated within each of 50 undergraduates who were each given 30 cue words (Rubin et al., 2003, Table 4). Correlations with *see*, as might be expected, are generally smaller in M.S. Correlations with *talk* are often negative, while they are positive in the undergraduates. Correlations with *in words* are among M.S.'s largest, but are among the undergraduates' smallest; it is as if having the memory come in words is a substitute for it coming with a large visual component. In contrast, the correlations from before illness are very different from those of the undergraduates. Consistent with our view, the anterograde correlations, with three exceptions, are like those of a sample of undergraduates, but the retrograde correlations are not. To quantify this observation, we correlated cell by cell the lower triangular matrix of correlations from the undergraduates with the anterograde and retrograde correlations of M.S., excluding the *age of memory* variable, which was restricted in M.S.'s data. Over the 91 ($14 \times 13/2$) cells, these correlations were 0.40 and 0.13, respectively. If the *see*, *talk*, and *in words* variables are not

Table 3
Means and S.D. for controls and M.S.

Var name	Controls		M.S.	
	Means	S.D.	Means	S.D.
Reliving	4.83	0.78	4.50	1.44
Back in time	4.85	1.09	2.10	1.63
Remember/know	5.69	0.65	4.10	1.78
Real/imagine	5.75	0.72	3.29	1.52
See	5.37	0.72	1.88	1.33
Setting	5.85	0.75	4.04	1.60
Hear	4.23	0.96	4.38	1.48
Talk	4.34	0.87	2.83	1.91
In words	3.42	1.36	4.71	1.46
Story	4.49	1.06	4.04	1.62
Emotions	4.65	0.91	3.79	1.76
Importance	3.51	0.88	6.35	1.23
Rehearsal	3.41	0.81	2.17	1.59
Specific	0.69	0.15	0.40	0.50
Merged/ext.	1.40	0.27	1.00 ^a	0.00 ^a

^a M.S. never identified a memory as 'extended' and so his score on this variable was always 1.

Table 4
MS's correlations for retrograde memories

Variable name	Reliving	Back in time	Remember/know	Real/imagine	See	Setting	Hear	Talk	In words	Story	Emotions	Importance	Rehearsal	Once/many
Reliving														
Back in time	52													
Remember/know	37	37												
Real/imagine	15	30	44											
See	-01	13	23	-05										
Setting	07	11	-01	-10	38									
Hear	20	24	27	32	20	66								
Talk	-12	08	-47	01	21	-07	-11							
In words	41	71	52	55	08	-14	17	-12						
Story	-18	04	27	-34	35	18	-01	-18	15					
Emotions	21	23	23	16	-14	30	66	-34	31	01				
Importance	-18	-14	-30	-24	11	27	04	18	02	37	02			
Rehearsal	-08	-15	00	11	00	-21	20	01	04	-06	40	27		
Once/many	-40	13	03	-15	40	51	22	-10	04	47	00	22	-22	
Age of memory	-44	05	04	-12	51	37	-02	-10	-07	33	-18	05	-29	77

Note: Decimals omitted. The order of abbreviations at the top follows the order of the left column.

Table 5
MS's correlations for anterograde memories

Variable name	Reliving	Back in time	Remember/know	Real/imagine	See	Setting	Hear	Talk	In words	Story	Emotions	Importance	Rehearsal	Once/many
Reliving														
Back in time	34													
Remember/know	50	24												
Real/imagine	63	50	47											
See	32	42	11	47										
Setting	28	17	23	17	06									
Hear	49	16	23	59	04	37								
Talk	-37	28	-20	-02	27	02	-18							
In words	68	40	50	47	23	47	63	-31						
Story	33	45	25	55	21	58	54	-17	56					
Emotions	40	48	25	47	25	22	50	19	41	44				
Importance	53	23	19	43	15	36	48	-26	30	47	45			
Rehearsal	27	23	14	29	04	13	29	18	18	23	54	46		
Once/many	14	-13	11	-16	14	36	-19	-35	21	05	-26	16	-28	
Age of memory	04	32	-10	03	-04	-18	14	09	25	-04	24	-15	00	-23

Note: Decimals omitted. The order of abbreviations at the top follows the order of the left column.

included, these correlations become 0.69 and 0.25, respectively (Table 5).

5. Discussion

M.S. clearly suffers from a severe imagery deficit. He can copy a line drawing but cannot recognize what he has drawn, and he has difficulty recognizing common objects and drawing objects from memory. He also has trouble generating and manipulating visual images, particularly when they involve colors, people, or mental rotation. Overall, M.S.'s visual deficits are closest to those of M.H. (Ogden, 1993), who also suffered from achromatopsia, prosopagnosia, and agnosia without alexia; two other well-described cases (M.M., from Brown & Chobor, 1995; L.D. from O'Connor et al., 1992) had prosopagnosia and visual agnosia but not achromatopsia. He, thus, meets the three criteria for an impaired access to long-term visual memories.

Previous research has demonstrated that visual imagery plays a central role in the recall of AMs. The recollection of an AM involves the retrieval of visual and spatial imagery (Brewer, 1995; Rubin et al., 2003), and AMs that come with a strong sense of reliving almost always involve vivid visual imagery (Rubin et al., 2003). For example, a recollection of a birthday party might involve generating a visual image of the overall scene and populating it with the images of the partygoers. M.S., however, cannot perform these tasks, and one might predict that his memories would be accordingly impoverished; that is, perhaps his memories would involve sound, smells, tastes, and tactile sensations, but no images. Neurological theories of memory allow for a different prediction. The destruction of M.S.'s visual regions may disrupt the spread of activation that AM retrieval requires (Damasio, 1989). For example, visual stimuli would no longer trigger firing in non-visual regions, and non-visual stimuli could no longer trigger visual regions, thereby preventing coactivation. If so, these visual imagery deficits could account for M.S.'s

memory problems, particularly his RA for AM (Greenberg and Rubin, 2003; Rubin and Greenberg, 1998). One might expect that blind people (whether cortical, congenital, or late) would manifest similar deficits; however, Patients like M.S. have lost long-term visual memory; late-blind people (Ogden & Barker, 2001) and cortically blind people (Chatterjee & Southwood, 1995) have not, and the congenitally blind probably never relied on it at all (Ogden and Barker, 2001).

Although M.S.'s RA for events is quite severe, his retrograde memory for autobiographical facts is relatively unimpaired, perhaps because he can rely on data from other modalities. He can remember names and dates that he had learned before his illness, but can rarely remember particular episodes associated with those facts. As measured by the AMI, his personal semantic memory is almost normal. As Wheeler & McMillan (2001) noted, this pattern of deficit is seen in DH (Hunkin et al., 1995), LD (O'Connor et al., 1992), MH (Ogden, 1993), and MM (Brown and Chobor, 1995), all of whom could retrieve some retrograde personal semantic memories. This pattern is consistent with the syndrome's etiology; the retrieval of data represented amodally or in one non-visual modality need not be impaired by disruption of coactivation.

Along the same lines, M.S. is better able to retrieve memories from after his illness, though his anterograde memory is clearly not normal. This finding is also consistent with similar cases, all of whom showed moderate AA (Brown and Chobor, 1995; Hunkin et al., 1995; O'Connor et al., 1992; Ogden, 1993; for further discussion of these cases, see Conway & Fthenaki, 2000; Greenberg and Rubin, 2003; Wheeler and McMillan, 2001). While we cannot fully explain the relative preservation of M.S.'s anterograde memories, we speculate that he may compensate by relying on other modalities, as his recall of post-morbidly acquired visual information is impaired.

M.S.'s neurological damage might suggest an MTL basis for his amnesia, but his retrograde memory deficits are different from those seen in MTL amnesia (though his anterograde deficits may be in part attributable to MTL damage). On the AMI, patients with MTL trauma tend to have a temporal gradient, with poorer performance for recent retrograde memories (Kopelman, 1994), while M.S.'s memory is impaired for all periods. As others have noted (e.g. Wheeler and McMillan, 2001), extensive autobiographical RA involves damage outside the MTL. We, therefore, suggest that M.S.'s RA is not entirely due to his MTL trauma and requires further explanation.

M.S. is similar to other cases of amnesia coupled with visual imagery loss; however, other damaged regions may contribute to his RA. Extensive RA also arises from bilateral thalamic infarction (Hodges & McCarthy, 1993), frontal and fronto-temporal junction damage (Hodges & Gurd, 1994), and temporal pole damage (see Kapur, 1999 and Kopelman, 2000 for reviews). While M.S. does have bilateral temporal pole damage, his performance differs from that of other temporal pole amnesics. (Because we need to examine the

specific contribution of temporal lobe damage to M.S.'s memory, we are not comparing him to cases that had frontal as well as temporal lobe damage (e.g. Wilson, Baddeley, & Kapur, 1995).) M.S.'s personal semantic memory is slightly impaired with no gradient. By contrast, one patient (Eslinger, 1998) had a greater impairment for childhood and early-adult periods; other patients had better remote semantic memory, though their scores were only mildly abnormal (Kopelman, Stanhope, & Kingsley, 1999). Temporal pole damage seems to cause graded personal semantic amnesia; M.S.'s personal semantic deficits are ungraded.

M.S.'s autobiographical amnesia is ungraded and unusually severe. On the autobiographical incidents part of the AMI, M.S. scores below other temporal-lobe amnesics (Viskontas, McAndrews, & Moscovitch, 2000), though he is within 1 S.D. of Kopelman et al. patients. Two patients (Eslinger, 1998; Kapur et al., 1996, Case 1) had impaired AM for the early periods but normal AM for the recent period. Other patients had better remote memory (e.g. Cermak & O'Connor, 1983; Evans, Graham, Pratt, & Hodges, 2003; Hokkanen, Launes, Vataja, Valanne, & Iivanainen, 1995; Mayes et al., 2003; Oxbury, Oxbury, Renowden, Squier, & Carpenter, 1997). Some patients had near-complete, ungraded RA (Kapur, Ellison, Smith, McLellan, & Burrows, 1992; Kapur et al., 1996, Case 2; Kitchener, Hodges, & McCarthy, 1998; Tanaka, Miyazawa, Hashimoto, Nakano, & Obayashi, 1999; Lucchelli & Spinnler, 1998; Warrington & McCarthy, 1988). The cases reported by Kapur et al. had visual deficits that were not described in detail, and their case 2 had right parieto-occipital damage. Finally, RAs as long as 20 year have been reported (Fujii, Yamadori, Endo, Suzuki, & Fukatsu, 1999), so M.S.'s RA might have had a gradient if he had contracted encephalitis later in life.

M.S.'s semantic deficits may contribute to his amnesia, as they do in semantic dementia (e.g. Westmacott, Leach, Freedman, & Moscovitch, 2001; see Hodges & Graham, 2001, for a review). His semantic deficits are, however, mild compared to those of most semantic-dementia patients with amnesia. Thus, like O'Connor and colleagues (1992) patient, M.S. might have a combination of deficits from Wheeler and McMillan's (2001) first category (probable temporal pole RA) and their second category (patients with visual deficits and more severe autobiographical than semantic RA). The severe and ungraded nature of M.S.'s deficit, however, is rare in temporal-lobe amnesics and suggests that his visual deficits are the major contributor to his RA. Overall, M.S.'s case shows the importance of neocortical regions that represent the components of AMs.

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Appendix A. Appendix

For Questions 1–6, the scales ranged from 1 (not at all) to 7 (as clearly as if it were happening right now). The questions were as follows:

1. As I remember the event, I feel as though I am *reliving* the original event.
2. As I remember the event, I can *hear* it in my mind.
3. As I remember the event, I can *see* it in my mind.
4. As I remember the event, I or other people are *talking*.
5. As I remember the event, I can feel now the *emotions* that I felt then.
6. As I remember the event, I can recall the *setting* where it occurred.

For Questions 7–11, the scales ranged from 1 (not at all) to 7 (as much as any memory). The questions were as follows:

7. Sometimes people know something happened to them without being able to actually remember it. As I think about the event, I can actually *remember* it rather than just knowing that it happened.
8. As I remember the event, it comes to me *in words*.
9. As I remember the event, I feel that I travel *back to the time when it happened*, that I am a subject in it again, rather than an outside observer tied to the present.
10. As I remember the event, it comes to me in words or in pictures *as a coherent story* or episode and not as an isolated fact, observation, or scene.
11. This memory is *significant* for my life because it imparts an important message for me or represents an anchor, critical juncture, or a turning point.

The remaining questions had unique scales:

12. I believe the event in my memory *really occurred* in the way I remember it and that I have not imagined or fabricated anything that did not occur. (Scale: 1, 100% imaginary; 7, 100% real).
13. Since it happened, I have *thought* or *talked about* this event. (Scale: 1, not at all; 7, as often as any event in my life).
14. To the best of your knowledge, is the memory of an event that occurred *once* at one particular time and place, a summary or *merging* of many similar or related events, or a for events that occurred over a fairly continuous *extended* period of time lasting more than a day. (Scale: 1, once; 2, merging; 3, extended).

This question actually produced two scales. *Specific* had a value of 1 if the subject judged the memory to take place within a single day and 0 if it took longer. *Merge* had a value of 1 if the event lasted longer than a day and was extended in a fairly continuous manner over a period of time and 2 if it was the merging of many discrete events.

15. Please *date* the memory (month/day/year) as accurately as you can. Please fill in a month, day, and year even if

you must estimate. If the memory extended over a period of time, report the approximate middle of the period (scored as retention interval in days).

The italicized words were double-underlined and set in bold in the booklet.

References

- Barbarotto, R., Capitani, E., & Laiacina, M. (1996). Naming deficit in herpes simplex encephalitis. *Acta Neurologica Scandinavica*, *93*, 272–280.
- Brewer, W. F. (1995). What is recollective memory? In D. C. Rubin (Ed.), *Remembering our past: Studies in autobiographical memory* (pp. 19–66). Cambridge: Cambridge University Press.
- Brown, J. W., & Chobor, K. L. (1995). Severe retrograde amnesia. *Aphasiology*, *9*, 163–170.
- Calabrese, P., Markowitsch, H. J., Durwen, H. F., Widlitzek, H., Haupts, M., Holinka, B., et al. (1996). Right temporofrontal cortex as critical locus for the ephory of old episodic memories. *Journal of Neurology, Neurosurgery, and Psychiatry*, *61*, 304–310.
- Cermak, L. S., & O'Connor, M. (1983). The anterograde and retrograde retrieval ability of a patient with amnesia due to encephalitis. *Neuropsychologia*, *21*, 213–234.
- Chatterjee, A., & Southwood, M. H. (1995). Cortical blindness and visual imagery. *Neurology*, *45*, 2189–2195.
- Conway, M. A., & Fthenaki, A. (2000). Disruption and loss of autobiographical memory. In F. Boller & J. Grafman (Eds.), *Handbook of neuropsychology: 2* (2nd ed., pp. 281–312). Elsevier.
- Conway, M. A., & Pleydell-Pearce, C. W. (2000). The construction of autobiographical memories in the self-memory system. *Psychological Review*, *107*, 261–288.
- Coughlan, A. K., & Hollows, S. E. (1985). *The adult memory and information processing battery*. Leeds: St. James University Hospital.
- Damasio, A. R. (1989). Time-locked multiregional retroactivation: A systems-level proposal for the neural substrates of recall and recognition. *Cognition*, *33*, 25–62.
- Davidoff, J., & Wilson, B. (1985). A case of visual agnosia showing a disorder of pre-semantic visual classification. *Cortex*, *21*, 121–134.
- De Haan, E. H., Heywood, C. A., Young, A. W., Edlstein, N., & Newcombe, F. (1995). Ettlinger revisited: The relation between agnosia and sensory impairment. *Journal of Neurology, Neurosurgery, and Psychiatry*, *58*, 350–356.
- Eslinger, P. J. (1998). Autobiographical memory after temporal lobe lesions. *Neurocase*, *4*, 481–495.
- Evans, J. J., Graham, K. S., Pratt, K. H., & Hodges, J. R. (2003). The impact of disrupted cortico-cortico connectivity: A long-term follow-up of a case of focal retrograde amnesia. *Cortex*, *39*, 767–790.
- Farah, M. (1984). The neurological basis of mental imagery: A componential analysis. *Cognition*, *18*, 245–272.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-mental state: A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189–198.
- Fujii, T., Yamadori, A., Endo, K., Suzuki, K., & Fukatsu, R. (1999). Disproportionate retrograde amnesia in a patient with herpes simplex encephalitis. *Cortex*, *35*, 599–614.
- Fuster, J. (1995). *Memory in the cerebral cortex*. Cambridge, MA: MIT Press.
- Goldenberg, G. (1992). Loss of visual imagery and loss of visual knowledge—a case study. *Neuropsychologia*, *30*, 1081–1099.
- Gordon, R. (1950). An experiment correlating the nature of imagery with performance on a test of reversal of perspective. *British Journal of Psychology*, *41*, 63–67.

- Greenberg, D. L., & Rubin, D. C. (2003). The neuropsychology of autobiographical memory. *Cortex*, 39, 687–728.
- Heywood, C., Cowey, A., & Newcombe, F. (1991). Chromatic discrimination in a cortically colour blind observer. *European Journal of Neuroscience*, 3, 802–812.
- Hodges, J. R., & Graham, K. S. (2001). Episodic memory: Insights from semantic dementia. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 356, 1423–1434.
- Hodges, J. R., & Gurd, J. M. (1994). Remote memory and lexical retrieval in a case of frontal Pick's disease. *Archives of Neurology*, 51, 821–827.
- Hodges, J. R., & McCarthy, R. A. (1993). Autobiographical amnesia resulting from bilateral paramedian thalamic infarction. A case study in cognitive neurobiology. *Brain*, 116, 921–940.
- Hokkanen, L., Launes, J., Vataja, R., Valanne, L., & Iivanainen, M. (1995). Isolated retrograde amnesia for autobiographical material associated with acute left temporal lobe encephalitis. *Psychological Medicine*, 25, 203–208.
- Hunkin, N. M., Parkin, A. J., Bradley, V. A., Burrows, E. H., Aldrich, F. K., Jansari, A., et al. (1995). Focal retrograde amnesia following closed head injury: A case study and theoretical account. *Neuropsychologia*, 33, 509–523.
- Kapur, N. (1997). How can we best explain retrograde amnesia in human memory disorder? *Memory*, 5, 115–129.
- Kapur, N. (1999). Syndromes of retrograde amnesia: A conceptual and empirical synthesis. *Psychological Bulletin*, 125, 800–825.
- Kapur, N., Ellison, D., Smith, M. P., McLellan, D. L., & Burrows, E. H. (1992). Focal retrograde amnesia following bilateral temporal lobe pathology. A neuropsychological and magnetic resonance study. *Brain*, 115, 73–85.
- Kapur, N., Scholey, K., Moore, E., Barker, S., Brice, J., Thompson, S., et al. (1996). Long-term retention deficits in two cases of disproportionate retrograde amnesia. *Journal of Cognitive Neuroscience*, 8, 416–434.
- Kitchener, E. G., Hodges, J. R., & McCarthy, R. (1998). Acquisition of post-morbid vocabulary and semantic facts in the absence of episodic memory. *Brain*, 121, 1313–1327.
- Kopelman, M. D. (1994). The autobiographical memory interview (ami) in organic and psychogenic amnesia. *Memory*, 2, 211–235.
- Kopelman, M. D. (2000). Focal retrograde amnesia and the attribution of causality: An exceptionally critical review. *Cognitive Neuropsychology*, 17, 585–621.
- Kopelman, M. D., & Kapur, N. (2001). The loss of episodic memories in retrograde amnesia: Single-case and group studies. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 356, 1409–1421.
- Kopelman, M. D., Stanhope, N., & Kingsley, D. (1999). Retrograde amnesia in patients with diencephalic, temporal lobe or frontal lesions. *Neuropsychologia*, 37, 939–958.
- Kopelman, M. D., Wilson, B. A., & Baddeley, A. D. (1990). *The autobiographical memory interview*. Bury St. Edmunds: Thames Valley Test Company.
- Lucchelli, F., & Spinnler, H. (1998). Ephemeral new traces and evaporated remote engrams: A form of neocortical temporal lobe amnesia? A preliminary case report. *Neurocase*, 4, 447–459.
- Marks, D. F. (1972). Individual differences in the vividness of visual imagery and their effect on function. In P. W. Sheehan (Ed.), *The function and nature of imagery*. New York: Academic Press.
- Marks, D. F. (1973). Visual imagery differences in the recall of pictures. *British Journal of Psychology*, 64, 17–24.
- Mayes, A. R., Isaac, C. L., Holdstock, J. S., Cariga, P., Gummer, A., & Roberts, N. (2003). Long-term amnesia: A review and detailed illustrative case study. *Cortex*, 39, 567–603.
- Mayes, A. R., & Roberts, N. (2001). Theories of episodic memory. *Philosophical Transactions of the Royal Society of London B: Biological Sciences*, 356, 1395–1408.
- McClelland, J., McNaughton, B., & O'Reilly, R. (1995). Why there are complementary learning systems in the hippocampus and neocortex: Insights from the successes and failures of connectionist models of learning and memory. *Psychological Review*, 102, 419–457.
- Mehta, Z., Newcombe, F., & De Haan, E. (1992). Selective loss of imagery in a case of visual agnosia. *Neuropsychologia*, 30, 645–655.
- Murre, J. M., Graham, K. S., & Hodges, J. R. (2001). Semantic dementia: Relevance to connectionist models of long-term memory. *Brain*, 124, 647–675.
- Newcombe, F., & Ratcliff, G. (1975). Agnosia: A disorder of object recognition. In F. Michel, B. Schott (Eds.), *Les Syndromes de disconnexion calleuse chez l'homme*. Lyon, France: Hôpital neurologique de Lyon.
- Newcombe, F., Young, A. W., & De Haan, E. H. (1989). Prosopagnosia and object agnosia without covert recognition. *Neuropsychologia*, 27, 179–191.
- O'Connor, M., Butters, N., Miliotis, P., Eslinger, P., & Cermak, L. S. (1992). The dissociation of anterograde and retrograde amnesia in a patient with herpes encephalitis. *Journal of Clinical and Experimental Neuropsychology*, 14, 159–178.
- Ogden, J. A. (1993). Visual object agnosia, prosopagnosia, achromatopsia, loss of visual imagery, and autobiographical amnesia following recovery from cortical blindness: Case M.H. *Neuropsychologia*, 31, 571–589.
- Ogden, J. A., & Barker, K. (2001). Imagery used in autobiographical recall in early and late blind adults. *Journal of Mental Imagery*, 25, 135–152.
- Oxbury, S., Oxbury, J., Renowden, S., Squier, W., & Carpenter, K. (1997). Severe amnesia: An usual late complication after temporal lobectomy. *Neuropsychologia*, 35, 975–988.
- Pollock, S., & Brown, P. (1984). Individual differences in visual imagery and spatial ability. *Intelligence*, 8, 93–138.
- Ratcliff, G., & Newcombe, F. (1982). Object recognition: Some deductions from the clinical evidence. In A. W. Ellis (Ed.), *Normality and pathology in cognitive functions* (pp. 147–171). New York: Academic Press.
- Richardson, A. (1994). *Individual differences in imaging: Their measurement, origins, and consequences*. Amityville, New York: Baywood.
- Rubin, D. C. (1995). *Memory in oral traditions: The cognitive psychology of epic, ballads, and counting-out rhymes*. New York: Oxford University Press.
- 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.
- Rubin, D. C., Schrauf, R. W., & Greenberg, D. L. (2003). Belief and recollection of autobiographical memories. *Memory & Cognition*, 31, 887–901.
- Shastri, L. (2002). Episodic memory and cortico-hippocampal interactions. *Trends in Cognitive Sciences*, 6, 162–168.
- Slee, J. A. (1980). Individual differences in visual imagery ability and the retrieval of visual appearances. *Journal of Mental Imagery*, 4, 93–113.
- Squire, L. R. (1992). Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review*, 99, 195–231.
- Tanaka, Y., Miyazawa, Y., Hashimoto, R., Nakano, I., & Obayashi, T. (1999). Postencephalitic focal retrograde amnesia after bilateral anterior temporal lobe damage. *Neurology*, 53, 344–350.
- Viskontas, I. V., McAndrews, M. P., & Moscovitch, M. (2000). Remote episodic memory deficits in patients with unilateral temporal lobe epilepsy and excisions. *Journal of Neuroscience*, 20, 5853–5857.
- Warrington, E. K., & McCarthy, R. A. (1988). The fractionation of retrograde amnesia. *Brain & Cognition*, 7, 184–200.
- Westmacott, R., Leach, L., Freedman, M., & Moscovitch, M. (2001). Different patterns of autobiographical memory loss in semantic dementia and medial temporal lobe amnesia: A challenge to consolidation theory. *Neurocase*, 7, 37–55.

- Wheeler, M. A., & McMillan, C. T. (2001). Focal retrograde amnesia and the episodic-semantic distinction. *Cognitive, Affective, & Behavioral Neuroscience, 1*, 22–36.
- Wheeler, M. A., Stuss, D. T., & Tulving, E. (1997). Toward a theory of episodic memory: The frontal lobes and autonoetic consciousness. *Psychological Bulletin, 121*, 331–354.
- Wilson, B. A., Baddeley, A. D., & Kapur, N. (1995). Dense amnesia in a professional musician following herpes-simplex virus encephalitis. *Journal of Clinical and Experimental Neuropsychology, 17*, 668–681.
- Wilson, B. A., & Davidoff, J. (1992). Partial recovery from visual object agnosia: A 10 year follow-up study. *Cortex, 29*, 529–542.
- Young, A. W., Newcombe, F., Hellawell, D., & De Haan, E. (1989). Implicit access to semantic information. *Brain & Cognition, 11*, 186–209.