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Visual memory-deficit amnesia: A distinct amnesic presentation and etiology

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ABSTRACT **We describe a form of amnesia, which we have called visual memory-deficit amnesia, that is caused by damage to areas of the visual system that store visual information. Because it is caused by a deficit in access to stored visual material and not by an impaired ability to encode or retrieve new material, it has the otherwise infrequent properties of a more severe retrograde than anterograde amnesia with no temporal gradient in the retrograde amnesia. Of the 11 cases of long-term visual memory loss found in the literature, all had amnesia extending beyond a loss of visual memory, often including a near total loss of pretraumatic episodic memory. Of the 6 cases in which both the severity of retrograde and anterograde amnesia and the temporal gradient of the retrograde amnesia were noted, 4 had a more severe retrograde amnesia with no temporal gradient and 2 had a less severe retrograde amnesia with a temporal gradient.**

According to the consensus view of the neural mechanisms supporting autobiographical, or episodic (1), memory, the ability to recollect past events consciously is mediated by multiple systems distributed throughout the cortex (2–11). Encoding new experiences for later recollection is mediated by areas in the medial temporal lobes, especially the hippocampus and surrounding areas (9, 10), and the diencephalon, which includes the mammillary bodies, and dorsomedial thalamus. The actual long-term storage of information for conscious recall is mediated by these encoding centers but is in sense-specific locations in cortex and in cortical and subcortical areas that support emotion (3). Retrieval of this information involves the frontal lobes, but like the role of the diencephalon and medial temporal lobes in encoding, the frontal lobes are not thought to contribute most of the information but rather are thought to contribute the retrieval strategies needed to access it (6, 11). For some time after encoding, retrieval or reactivation also requires the involvement of the hippocampal region. Over time, however, a consolidation process may take place, allowing that reactivation to occur without medial temporal or diencephalic support (refs. 9, 12, and 13, but see ref. 14). When an autobiographical memory is recalled, information in widely separated areas of cortex are excited in time-locked circuits or coactivation similar to those that were active at encoding (2, 3, 12, 13). For example, a familiar sound (such as the whistle of a train) might lead to activation in the auditory cortex, which, either directly or via hippocampal mediation, might activate a pattern of firing in visual cortex (perhaps stimulating a visual image of meeting someone at the train station). This activity in visual cortex might in turn stimulate new activity in visual, auditory, and other cortices (stimulating visual images, sounds, smells, and other sensory components that are associated with the meeting) while feeding back upon the original pattern of firing in auditory cortex. This cascade of activation continues, ultimately producing a pattern of firing similar to that present during the original experience.

In its most common presentation, amnesia occurs when structures in the diencephalon or the medial temporal cortex are damaged. Amnesia involves an impaired ability both in the encoding and retention of new posttraumatic autobiographical memories (termed anterograde amnesia) and in the retrieval of pretraumatic memories (termed retrograde amnesia). The anterograde amnesia is typically more severe than the retrograde amnesia, in that the patient can remember some pretraumatic events but cannot generally encode or retain any posttraumatic memories. The opposite pattern, termed isolated or focal retrograde amnesia, is extremely rare; it is thought to result from damage to anterior temporal structures (15–17). In both presentations, retrograde amnesia is usually temporally graded so that older memories are more likely to be spared than recent memories (18). The explanation for this temporal gradient involves the phenomenon of consolidation; though the basic data in the posterior neocortex still exist for both recent and older memories, the damaged hippocampal region can no longer mediate the pattern of firing required for recollection of more recent memories.

Although theories of the neural basis of memory attribute recollection to this cascade of activation in sensory and association cortices, they make few if any claims about the relative contribution of different areas of the posterior neocortex to memory in general. The behavioral data, however, suggest that vision and visual imagery play a central role in a variety of memory tasks (19). Vivid visual images have been discussed as a major component of the flashbulb memory phenomenon (20, 21). Although visual images do not guarantee accuracy (22, 23), the successful retrieval of a visual image may lead a subject to classify a memory as being from a real event that actually occurred rather than from an event that did not occur but was just thought about (24). A broad base of psychological data and philosophical analysis suggests that the experience of recollecting an autobiographical memory coincides with the successful retrieval of a visual image (25); people tend to report recollecting or reliving an event if and only if they have a visual image of that event. Thus, autobiographical memory appears to rely upon visual imagery to a much greater extent than other sensory modalities, making vision the primary modality for recollection.

Herein, we ask what new testable hypotheses follow if we consider damage to the posterior neocortical areas where the information is stored. On the basis of a logical extension of the consensus views of autobiographical memory, the neural basis of most amnesias, and the behavioral data on the role of imagery in memory, we predict that damage to the store of visual information should lead to a global impairment of autobiographical memory that extends beyond a simple loss of visual memories. With the loss of access to this vital store of information, the coactivation in neural circuits that underlie

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recollection would be difficult to initiate or maintain; steps in the cascade of activation would be lost. In our earlier example, the sound of the train whistle would no longer stimulate the visual image of the train station, in turn preventing the other auditory, visual, and nonvisual activation in posterior neocortex that underlies the rest of the memory. Thus, an inability to initiate and maintain a pattern of firing in damaged visual cortex would prevent activation in nonvisual and visual posterior neocortices, even if those nonvisual cortices remained intact.

Damasio's theory of memory function suggests another interpretation (2). According to his theory, convergence zones serve two main functions: they activate areas of a particular sensory cortex and they feed forward to the next level of convergence that coordinates activation in multiple sensory cortices. For example, on stimulation by the hippocampal region or by a small portion of sensory cortex, local convergence zones in the visual system would coordinate the activation of all the patterns of firing necessary to form a single visual image. Damage to the convergence zones would have several effects: (*i*) it would prevent one visual stimulus from activating other areas of visual cortex; (*ii*) it would prevent visual stimuli from activating areas of other sensory cortices; (*iii*) it would prevent stimuli in other sensory cortices from activating regions in visual cortex.

In addition, we predict that these patients should suffer from only mild anterograde impairments, provided that their brain damage did not disrupt other functioning. Because the neural structures that encode information remain undamaged in this hypothesized form of amnesia, these patients could gradually compensate for their deficits, allowing emotion and nonvisual sensory data to play a greater role (26, 27). This compensation may be seen as analogous to the lack of amnesia in the congenitally blind, who have no visual input but, with minor exceptions, can use other sensory data to perform tasks usually thought to depend on visual imagery (28, 29). In practice, however, the retrograde amnesia need not be complete, because patients might be able to reactivate isolated bits of information from other areas of posterior neocortex; and the anterograde amnesia may be moderate, because the process of compensation would take some time to develop and might never totally make up for the inability to store new visual information.

We further predict that in these amnesic patients, there should be no sparing of childhood memories that characterizes almost all other forms of retrograde amnesia. The temporal gradient in more common forms of amnesia is thought to arise because consolidation is arrested at various stages of completion leading to a failure of retention of the most recent memories. In our predicted form of amnesia, though, the deficit arises not from a loss of retention of incompletely consolidated information but rather from a loss of the visual information itself, so that even older consolidated memories would be left with substantial gaps. The prediction of an absence of a temporal gradient is particularly unusual; aside from cases of psychogenic amnesias or malingering patients, early memories are almost always spared. The low base rate for a lack of a temporal gradient, combined with the low base rate for a severe retrograde amnesia with minimal anterograde deficits (15, 16), dramatically lowers the probability of seeing our predicted pattern from other etiologies.

On the basis of a synthesis and extension of current theory, we have predicted a specific form of amnesia, visual memory-deficit amnesia, one not previously noted as a general syndrome in the literature on amnesia or agnosia (30, 31). The predictions we have made, or very similar ones, are required by the consensus view of the neural basis of amnesia presented herein. They are also consistent with mathematical models of hippocampal function (12, 13). Every modern theorist of amnesia explicitly states that the perceptual and conceptual information that is the basis of autobiographical memories is not stored primarily in the medial temporal, diencephalic, or frontal regions but in posterior cortices. Removing such cortices must cause a loss in the basic information used to form autobiographical memories. Every modern theorist evokes some form of a coactivation mechanism that at recollection has a pattern of firing similar to the pattern that occurred at the original event. Removing a large portion of that feedback circuit must block such patterns of firing. Others have proposed similar ideas in the context of their individual case studies, suggesting that trauma to posterior cortices could cause amnesia (26, 27, 32), with one paper (17) formulating and then rejecting the notion. Herein, from an extensive investigation of the literature and a theoretical refinement based on Farah's analysis of visual imagery, we contribute a more detailed description of the characteristics of this syndrome.

To test our prediction, we needed a way to locate patients who had lost visual memory. Farah (33) investigated cases of visual imagery loss. From Kosslyn's (34) detailed analysis, she divided imagery into component processes, including a long-term visual memory that stores visual information. Patients with a loss of long-term visual memory cannot access stored visual information at all, so according to the consensus models of memory and amnesia, they should not be able to support the coactivation underlying memory. In Farah's model, a patient must satisfy three criteria to be classified as a case of long-term visual memory loss. (*i*) The patient must be able to detect, draw, or describe the visual properties of an object that is present, which demonstrates that the deficit could not have arisen from motor, perceptual, or linguistic impairment. (*ii*) The patient must not be able to recognize an object on sight alone, either by indicating its name or its function. The first two criteria define the patient as an associative visual agnosic (30, 31). (*iii*) The patient should not be able to draw an object from memory (though some simplistic drawings are allowed), describe its visual characteristics from memory, or detect its visual image upon introspection. The third criterion helps isolate the deficit as one of long-term visual memory. Because such patients have lost the use of stored visual information in a variety of tasks, it is likely that they have lost the use of such information in the coactivation needed for recollection. They are therefore the most likely identifiable group to have amnesia caused by a visual deficit.

We therefore searched the literature for cases of visual imagery impairments, specifically for cases of long-term visual memory impairments. We started with all 37 cases of imagery deficits in Farah's review (33), and we added 11 additional cases, mostly from the subsequent literature. Of these 48 cases, 11 cases met our three criteria for long-term visual memory loss (Table 1; refs. 26, 35–44). In sorting the data, we used a conservative threshold and did not include cases classified as imagery loss based solely on the patient's introspective report. As Farah *et al.* (45) note, brain-damaged patients' introspective reports of visual imagery may be unreliable, and many individuals with no known neurological damage report that they do not experience visual images while awake (46). Although we included the describe option in our use of the first and third criteria, we added no cases because of it, though Farah did. Case studies do not usually note that objects present can be described visually, and determining that a description is based on visual information is not easy. Thus, our cases of long-term visual memory were all determined by using the draw option in the first and third criteria. As with neurological cases in general, classification was at times difficult. We disagreed with Farah (33) on one case determined by drawing. Farah included the case as a loss of long-term visual memory, but although the patient had trouble drawing multiple shapes from memory, he had "an excellent ability to sketch an outline of his motor car, home, etc." from memory (47) and so did not fit our criteria.

Table 1. All 11 cases of long-term visual memory loss found in the literature

Reference	Etiology	Damage	Amnesia	RA > AA	Gradient
Brown and Chobor (37)	CHI	Bil. occ., R. front.	Yes	Yes	No
O'Connor *et al*. (39)	Encephalitis	R. occ., par., front., MT, IT, AT	Yes	Yes	No
Ogden (26)	CHI	Bil. occ.	Yes	Yes	No
Trojano and Grossi (43)	CHI	Bil. occ., front, temp, MT, ~IT	Yes	Yes	No
Ratcliff and Newcombe (40)	Encephalitis or CVA	Bil. occ., par., MT, IT	Yes	Yes	?
Albert *et al*. (35)	CVA	Bil. occ.	Yes	No	Yes
Gomori and Hawryluk (38)	Cyst	Bil. occ., IT	Yes	No	Yes
Beyn and Knyazeva (36)	CVA	?	Yes	?	?
Shuttleworth *et al*. (41), case 2	CHI	Bil. occ., MT	Yes	?	?
Taylor and Warrington (42)	Atrophy	Bil. occ., ~IT	Yes	?	?
Wapner *et al*. (44)	CVA	L. occ., temp., MT	Yes	?	?

The 11 cases listed met the three criteria for visual memory deficit. The five cases with RA > AA are listed first. CHI, closed-head injury; CVA, cerebrovascular accident; occ., occipital; par, parietal; front, frontal; temp, temporal; MT, possible medial temporal damage; IT, inferotemporal damage; AT, anterior temporal damage; RA > AA, is the retrograde amnesia reported as more severe than the anterograde amnesia; gradient, is there a temporal gradient; ?, case study provides no information; ~, deficit is marginal or report is equivocal.

The etiology in the 11 patients with long-term visual memory loss is mixed with four closed-head injuries (26, 37, 41, 43), three cerebrovascular accidents (35, 36, 44), one encephalitis (39), one encephalitis or cerebrovascular accident (40), one colloid cyst (38), and one cerebral atrophy aggravated by alcoholism (42). As would be expected from the presence of visual deficits, the damage in these 11 patients tended to include the occipital lobes. Of the 7 cases that based their lesion information on a brain scan, 6 have bilateral occipital damage (26, 37, 38, 40, 42, 43) and 1 has right occipital damage (39). Three of the 7 cases with brain scans reported trauma to the inferotemporal region (38–40), which may contain convergence zones for the visual system; two additional cases reported damage to both the occipital and temporal lobes, suggesting that inferotemporal trauma might exist (42, 43).

All 11 patients with long-term visual memory loss had amnesia. In addition to the occipital damage, 5 of the 11 patients showed some evidence of possible damage to the medial temporal region (39–41, 43, 44), by using a liberal criterion that considered any unspecified temporal lobe damage to be possible evidence of medial temporal trauma. However, the pattern of anterograde and retrograde amnesia suggests that this medial temporal damage was not the main cause of the observed memory deficits. There were 7 cases that reported on both anterograde and retrograde amnesia. Two of these had more severe anterograde than retrograde amnesia (35, 38), and 5 fit our predicted pattern, manifesting a severe retrograde amnesia with only mild to moderate anterograde deficits (26, 37, 39, 40, 43). This particular pattern suggests focal retrograde amnesia (15–17); however, by using a liberal criterion, only 1 of these 5 cases (39) manifested the anterior temporal lobe damage that has been hypothesized for focal retrograde amnesia. For the 4 of these 5 cases in which enough information was provided to make a judgment (26, 37, 39, 43), all 4 had dense near-complete retrograde amnesia, and none had a temporal gradient. Two showed no evidence of medial temporal damage (26, 37).

Two of the patients (41, 43) showed marked improvement in their long-term visual memory over the course of testing described. Our analysis herein is based on a time close to the onset of their deficit, when it was most severe. Although no causal link can be established, the improvement in long-term visual memory came with an improvement in autobiographical memory. In another case (36), the patient had clear prosopagnosia but marginal agnosia by our criteria in other areas and could have been excluded. The amnesia in this case was also marginal, further suggesting that the degree of severity of the two conditions may be related. Another suggestive observation is that 2 cases with severe amnesia (26, 37) mention that memory in the retrograde period was preserved for songs, though it is not clear that these memories included specific episodes. Nonetheless, consistent with the theory proposed, strong organized auditory memory seems to be able to cause coactivation in other circuits, overcoming the loss of coactivation caused by visual memory loss.

Loss of long-term visual memory may not be the only imagery deficit associated with amnesia. Our search uncovered 7 other patients with both amnesia and other forms of visual imagery deficits (Table 2; refs. 27, 32, 41, and 48–52). Three of these patients could not copy objects, although they met our other criteria (41, 49, 51); these patients might therefore suffer from perceptual problems coupled with a long-term visual memory deficit. One patient (32, 52) suffered from severe amnesia combined with only mild agnosia; although the deficits in this case fit our criteria overall, the patient also manifested extensive anterograde deficits coupled with medial temporal damage, and we excluded this case because the patient's recovery from agnosia did not coincide with a recovery from retrograde amnesia. The three remaining patients had amnesia without visual agnosia (27, 48, 50), but we

Table 2. Cases of imagery loss that did not meet criteria of visual memory deficit but that had amnesia

Reference	Etiology	Damage	Copy	Recognize	Draw	RA > AA	Gradient
Boyle and Nielsen (49)	Neoplasm/surgery	R. occ., MT	No	No	No	No	Yes
Levine (51)	Neoplasm	R. occ., par., MT	No	No	No	Yes	?
Shuttleworth *et al*. (41)	CVA and CHI	Bil. occ. temp., MT, ~IT	No	No	No	?	?
Arbuse (48)	Neoplasm	L. occ., par.	No	Yes	?	?	?
Grossi *et al*. (50)	CVA	L. occ., MT, ~IT	Yes	Yes	No	?	Yes
Hunkin *et al*. (27)	CHI	Bil. occ., IT	Yes	Yes	No	Yes	No
Schnider *et al*. (32, 52)	CVA	L. occ., MT, IT	Yes	Yes	No	?	No

Three cases that would have met our criteria for visual memory deficit if they could have drawn a object that was present are listed first. CHI, closed-head injury; CVA, cerebrovascular accident; occ., occipital; par, parietal; temp, temporal; MT, possible medial temporal damage; IT, inferotemporal damage; copy, patient can copy drawings or draw from a model; draw, patient can draw an object from memory; RA > AA, is the retrograde amnesia reported as more severe than the anterograde amnesia, ?, case study provides no information; ~, deficit is marginal or report is equivocal.

could not discern any pattern to the damage locations or the imagery deficits in these cases.

Our investigation was hindered by the lack of detail of some of the older reports. In some of these cases, the description of amnesia was limited to one word or phrase, such as ''global amnesia.'' Although this brevity might seem odd in light of our attempt to integrate multiple neural systems, many neuropsychological case reports concentrate on the exploration of one deficit, viewing a patient's other deficits as tangential to the investigation at hand.

When data were available, they fit our predictions of the three properties of visual memory deficit amnesia with only four exceptions, even though when based on all cases of amnesia our predictions are of extremely low probability events. All 11 cases of long-term visual memory loss had amnesia, though two cases had reversals of both the retrograde more severe than anterograde amnesia prediction and the lack of temporal gradient prediction. Thus, people who had a visual memory deficit also had amnesia that extended well beyond a deficit in visual memory; an amnesia that often had retrograde amnesia more severe than anterograde amnesia and no sparing of early memories. Visual memory-deficit amnesia is a new classification of amnesia that, unlike most forms of amnesia, is caused by a loss of posterior neocortex rather than damage to the medial temporal or diencephalic region. Though rare, it is frequent enough to establish that its properties distinguish it from other types of amnesia.

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