

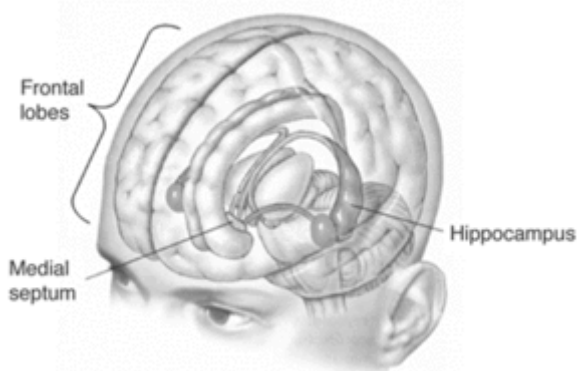
Henry Molaison

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Henry Gustav Molaison (February 26, 1926 – December 2, 2008), known widely as **H.M.**, was an American memory disorder patient who had a bilateral medial temporal lobectomy to surgically resect the anterior two thirds of his hippocampi, parahippocampal cortices, entorhinal cortices, piriform cortices, and amygdalae in an attempt to cure his epilepsy. He was widely studied from late 1957 until his death in 2008.^{[1][2]} His case played a very important role in the development of theories that explain the link between brain function and memory, and in the development of cognitive neuropsychology, a branch of psychology that aims to understand how the structure and function of the brain relates to specific psychological processes. He resided in a care institute located in Windsor Locks, Connecticut, where he was the subject of ongoing investigation.^[3]

Molaison's brain is kept at UC San Diego where it was sliced into histological sections on December 4, 2009.^[4] The corresponding data was then made available to researchers.^[5]

Biography



Most of Molaison's two hippocampi were removed bilaterally.

Henry Molaison was born on February 26, 1926, and experienced intractable epilepsy that has sometimes been attributed to a bicycle accident at the age of seven. (This accident was initially reported to have occurred at age nine, but was corrected by the patient's mother at a later stage.^[6]) He had partial seizures for many years, and then several tonic-clonic seizures following his sixteenth birthday. In 1953 he was referred to William Beecher Scoville, a neurosurgeon at Hartford Hospital, for treatment.^[2]

Scoville localized Molaison's epilepsy to his left and right medial temporal lobes (MTLs) and suggested surgical resection of the MTLs as a treatment. On August 25, 1953, at the age of 27, Molaison's bilateral medial temporal lobe resection included the hippocampal formation and adjacent structures, including most of the amygdaloid complex and

entorhinal cortex, was removed. His hippocampi appeared entirely nonfunctional because the remaining 2 cm of hippocampal tissue appeared atrophic and because the entire entorhinal cortex, which forms the major sensory input to the hippocampus, was destroyed. Some of his anterolateral temporal cortex was also destroyed.

After the surgery was successful in its primary goal of controlling his epilepsy, Molaison developed severe anterograde amnesia: although his working memory and procedural memory were intact, he could not commit new events to his explicit memory. According to some scientists, he was impaired in his ability to form new semantic knowledge,^[7] but researchers argue over the extent of this impairment. He also had moderate retrograde amnesia, and could not remember most events in the 1–2-year period before surgery, nor some events up to 11 years before, meaning that his amnesia was temporally graded. However, his ability to form long-term procedural memories was intact; thus he could, for example, learn new motor skills, despite not being able to remember learning them.

The case was first reported in a paper by Scoville and Brenda Milner in 1957.^[8] Near the end of his life, Molaison regularly filled in crossword puzzles.^[9] He was able to fill in answers to clues that referred to pre-1953 knowledge. For post-1953 information he was able to modify old memories with new information. For instance, he could add a memory about Jonas Salk by modifying his memory of polio.^[11]

He died on December 2, 2008.^[12]

Insights into memory formation

Molaison was influential not only for the knowledge he provided about memory impairment and amnesia, but also because it was thought his exact brain surgery allowed a good understanding of how particular areas of the brain may be linked to specific processes hypothesized to occur in memory formation. In this way, his case was taken to provide information about brain pathology, and helped to form theories of normal memory function.

In particular, his apparent ability to complete tasks that require recall from short-term memory and procedural memory but not long-term episodic memory suggests that recall from these memory systems may be mediated, at least in part, by different areas of the brain. Similarly, his ability to recall long-term memories that existed well before his surgery, but inability to create new long-term memories, suggests that encoding and retrieval of long-term memory information may also be mediated by distinct systems.

Nevertheless, imaging of Molaison's brain in the late 1990s revealed the extent of damage was more widespread than previous theories had accounted for, making it very hard to identify any one particular region or even isolated set of regions that were responsible for HM's deficits.^[10]

Contribution to science

The study of Molaison revolutionized the understanding of the organization of human memory. It has provided broad evidence for the rejection of old theories and the formation of new theories on human memory, in particular about its processes and the underlying neural structures (cf. Kolb & Whishaw, 1996). In the following, some of the major insights are outlined.

Molaison's brain is the subject of an unprecedented anatomical study funded by the Dana Foundation and the National Science Foundation. The project, headed by Jacopo Annese, the Director of The Brain Observatory at UC San Diego, will provide a complete microscopic survey of the entire brain and reveal the neurological basis of Molaison's historical memory impairment at cellular resolution. On December 4, 2009, Annese's group acquired 2401 brain slices with only two damaged slices and 16 potentially problematic slices. The group is currently planning the second phase of the project.^{[4][11]} The digital 3D reconstruction of his brain was finished at the beginning of 2014.^[12]

Amnesia

Molaison's general condition has been described as heavy anterograde amnesia, as well as temporally graded retrograde amnesia. Since HM did not show any memory impairment before the surgery, the removal of the medial temporal lobes can be held responsible for his memory disorder. Consequently, the medial temporal lobes can be assumed to be a major component involved in the formation of semantic and episodic long-term memories (cf. medial temporal lobes described as a convergence zone for episodic encoding in Smith & Kosslyn, 2007). Further evidence for this assumption has been gained by studies of other patients with lesions of their medial temporal lobe structures.^[8]

Despite his amnesic symptoms, Molaison performed quite normally in tests of intellectual ability, indicating that some memory functions (e.g., short-term memories, stores for words, phonemes, etc.) were not impaired by the surgery. However, for sentence-level language comprehension and production, Molaison exhibited the same deficits and sparing as in memory . Molaison was able to remember information over short intervals of time. This was tested in a working memory experiment involving the recall of previously presented numbers; in fact, his performance was no worse than that of control subjects (Smith & Kosslyn, 2007). This finding provides evidence that working memory does not rely on medial temporal structures. It further supports the general distinction between short-term and long-term stores of memory). Molaison's largely intact word retrieval provides evidence that lexical memory is independent of the medial temporal structures .

Motor skill learning

In addition to his intact working memory and intellectual abilities, studies of Molaison's ability to acquire new motor skills contributed to a demonstrated preserved motor learning (Corkin, 2002). In a study conducted by Milner in the early 1960s, Molaison

acquired the new skill of drawing a figure by looking at its reflection in a mirror (Corkin, 2002). Further evidence for intact motor learning was provided in a study carried out by Corkin (1968). In this study, Molaison was tested on three motor learning tasks and demonstrated full motor learning abilities in all of them.

Experiments involving repetition priming underscored Molaison's ability to acquire implicit (non-conscious) memories, in contrast to his inability to acquire new explicit semantic and episodic memories (Corkin, 2002). These findings provide evidence that memory of skills and repetition priming rely on different neural structures than memories of episodes and facts; whereas procedural memory and repetition priming do not rely on the medial temporal structures removed from Molaison, semantic and episodic memory do (cf. Corkin, 1984).

The dissociation of Molaison's implicit and explicit learning abilities along their underlying neural structures has served as an important contribution to our understanding of human memory: Long-term memories are not unitary and can be differentiated as being either declarative or non-declarative (Smith & Kosslyn, 2007).

Spatial memory

According to Corkin (2002), studies of Molaison's memory abilities have also provided insights regarding the neural structures responsible for spatial memory and processing of spatial information. Despite his general inability to form new episodic or factual long-term memories, as well as his heavy impairment on certain spatial memory tests, Molaison was able to draw a quite detailed map of the topographical layout of his residence. This finding is remarkable since Molaison had moved to the house five years after his surgery and hence, given his severe anterograde amnesia and insights from other cases, the common expectation was that the acquisition of topographical memories would have been impaired as well. Corkin (2002) hypothesized that Molaison “was able to construct a cognitive map of the spatial layout of his house as the result of daily locomotion from room to room” (p. 156).

Regarding the underlying neural structures, Corkin (2002) argues that Molaison's ability to acquire the floor plan is due to partly intact structures of his spatial processing network (e.g., the posterior part of his parahippocampal gyrus). In addition to his topographical memory, Molaison showed some learning in a picture memorization-recognition task, as well as in a famous faces recognition test, but in the latter only when he was provided with a phonemic cue. Molaison's positive performance in the picture recognition task might be due to spared parts of his ventral perirhinal cortex.

Furthermore, Corkin (2002) argues that despite Molaison's general inability to form new declarative memories, he seemed to be able to acquire small and impoverished pieces of information regarding public life (e.g., cued retrieval of celebrities' names). These findings underscore the importance of Molaison's spared extrahippocampal sites in semantic and recognition memory and enhance our understanding of the interrelations between the different medial temporal lobe structures. Molaison's heavy impairment in

certain spatial tasks provides further evidence for the association of the hippocampus with spatial memory (Kolb & Whishaw, 1996).

Memory consolidation

Another contribution of Molaison to understanding of human memory regards the neural structures of the memory consolidation process, which is responsible for forming stable long-term memories (Eysenck & Keane, 2005). Molaison displayed a temporally graded retrograde amnesia in the way that he “could still recall childhood memories, but he had difficulty remembering events that happened during the years immediately preceding the surgery” (Smith & Kosslyn, 2007, p. 214). His old memories were not impaired, whereas the ones relatively close to the surgery were. This is evidence that the older childhood memories do not rely on the medial temporal lobe, whereas the more recent long-term memories seem to do so (Smith & Kosslyn, 2007). The medial temporal structures, which were removed in the surgery, are hypothesized to be involved in the consolidation of memories in the way that “interactions between the medial temporal lobe and various lateral cortical regions are thought to store memories outside the medial temporal lobes by slowly forming direct links between the cortical representations of the experience” (Smith & Kosslyn, 2007, p. 214).