
AMNESIA

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Glossary

Alcoholic blackout Amnesia without loss of consciousness, in which the intoxicated person retains the ability to perform certain "automatized" behaviors without any subsequent memory for the episode.

Amnesia A special case of forgetting in which the memory loss is greater than would be expected under ordinary circumstances. Anterograde amnesia affects memory for events occurring after the instigating event; retrograde amnesia affects memory for events occurring before the instigating event.

Amnesic syndrome A profound deficit in learning and memory usually associated with bilateral damage to the diencephalon or to the medial portions of the temporal lobe. It always involves an anterograde amnesia and may involve a retrograde amnesia as well.

Functional amnesia A significant loss of memory attributable to an instigating event, usually stressful, that does not result in insult, injury, or disease affecting brain tissue. Its most common forms are psychogenic amnesia, psychogenic fugue, and multiple personality disorder.

Infantile and childhood amnesia An amnesia observed in adults, affecting memory for personal experiences occurring in the first 5–7 years of life. Infantile amnesia commonly covers the period before language and speech develop.

Posthypnotic amnesia A retrograde amnesia induced by means of hypnotic suggestion; it may be canceled by a prearranged reversibility cue.

Transient global amnesia A benign and temporary amnesia characterized by sudden onset, apparently caused by momentary vascular insufficiencies affecting brain tissue.

Traumatic retrograde amnesia A retrograde amnesia resulting from a concussive blow to the head; most of the affected memories are eventually recovered, except for a "final RA" affecting the accident itself.

AMNESIA may be defined as a special case of forgetting, in which the loss of memory is greater than would be expected under ordinary circumstances. A head-injured patient is no longer able to learn things that he was once able to master easily; a patient with psychogenic fugue loses her identity as well as her fund of autobiographical memories. Amnesia includes frank pathologies encountered in neurological and psychiatric clinics, such as Korsakoff's syndrome, Alzheimer's disease, traumatic retrograde amnesia, and multiple personality disorder. But it also includes abnormalities of memory observed ubiquitously, such as infantile and childhood amnesia, the exaggerated forgetfulness associated with healthy aging, and the memory failures associated with sleep and general anesthesia. These naturally occurring pathologies of memory have their counterparts in amnesic states induced in otherwise normal, intact individuals by means of experimental techniques, such as electroconvulsive shock in laboratory rats and posthypnotic amnesia in college sophomores.

Experimental research on memory began with the publication of Ebbinghaus' *Über das Gedächtniss* in 1885, but the clinical description of amnesia dates from even earlier. Korsakoff described the amnesic syndrome that bears his name in 1854. And in 1882 Ribot published *Les Maladies de la memoire*, with a detailed description of the consequences for memory of brain insult, injury, and disease, as well as a

may have adopted in that state. Examination of such cases after they are resolved often reveals an instigating episode of psychological stress. [See AUTOBIOGRAPHICAL REMEMBERING AND SELF-KNOWLEDGE; SEMANTIC MEMORY.]

Multiple personality disorder (MPD), in which two or more personalities appear to inhabit a single body, alternating control over experience and action, also involves a disruption of memory and identity. One of these personalities is often "primary," in that it is the one that has been manifest the longest and known by most other people. Most important in the present context, the various personalities appear to be separated by an amnesic barrier that prevents one alter ego from gaining access to the memories of another. In many cases, the amnesia is asymmetrical, in that Personality A may be aware of Personality B, but not the reverse. The amnesia largely affects identity and autobiographical memory; as a rule, the various personalities share semantic memory and procedural knowledge in common. The most widely accepted theory of MPD holds that it develops in defense against abuse, trauma, or deprivation in early childhood. [See CHILD ABUSE; PERSONALITY DISORDERS.]

Reports of MPD were relatively common in the clinical literature before 1920, and then virtually disappeared. There has been a resurgence of MPD, bordering on epidemic, in recent years. However, it is not clear how many of these are iatrogenic in nature or simply misdiagnosed. Where the alternate personalities are initially elicited through hypnosis or other special techniques, or when an amnesic barrier is absent, the case is suspect. MPD is sometimes offered as an insanity defense, claiming that a second personality is actually responsible for crimes of which the first personality is accused. MPD raises interesting issues of criminal law: in principle, the actions of one personality may be outside another personality's ability to control; interpersonality amnesia may prevent the accused from assisting in the defense; and techniques intended to elicit testimony from a personality may violate constitutional safeguards against self-incrimination. However, MPD has rarely proved successful as a defense against criminal charges.

There are several experimental studies that confirm the existence of interpersonality amnesia in MPD. Thus, for example, one alter ego is often unable to recall or recognize a list of items studied by another. Interestingly, there is some evidence that IM may be spared in these cases. Thus, one alter

ego may show savings in relearning, interference, transfer of training, or priming effects involving a list studied exclusively by another one. Although the available research is somewhat ambiguous, in general it seems that the amnesic barrier is permeable in the case of implicit memories.

Just as the amnesic syndrome finds its experimental analog in drug-induced amnesia, and traumatic retrograde amnesia in ECT and ECS, the functional amnesias seen clinically have their laboratory parallel in posthypnotic amnesia. Following appropriate suggestions and the termination of hypnosis, many subjects cannot remember the events that transpired while they were hypnotized. After the hypnotist administers a prearranged cue, the critical memories become accessible again; the fact of reversibility marks posthypnotic amnesia as a disruption of memory retrieval. The amnesia does not occur unless it has been suggested (explicitly or implicitly), and memory is not reinstated merely by the reinduction of hypnosis; thus, posthypnotic amnesia is not an instance of state-dependent memory. Response to the amnesia suggestion is highly correlated with individual differences in hypnotizability: while hypnotic "virtuosos" typically show a very dense amnesia, their insusceptible counterparts show little or no forgetting. [See HYPNOSIS.]

Like the organic amnesias, posthypnotic amnesia is selective. The subject may forget which words appeared on a study list, but retains the ability to use these words in speech and writing. Skills acquired in hypnosis transfer to the posthypnotic state, and suggestions for amnesia have no impact on practice effects. Subjects who learn new factual information while hypnotized may retain it despite suggestions for amnesia, but these same subjects may well forget the circumstances in which this knowledge was acquired—a phenomenon of *source amnesia* that has also been observed in the amnesic syndrome. Finally, there is good evidence that priming effects are preserved in posthypnotic amnesia. That is, subjects who cannot remember words from a study list are more likely to use those words as free associations or category instances than would be expected by chance. Thus, posthypnotic amnesia shows the familiar dissociation between EM and IM.

Because functional amnesia occurs in the absence of brain damage, and because posthypnotic amnesia occurs in response to suggestion, questions inevitably arise about malingering, simulation, and behavioral compliance. Unfortunately, it is difficult to distinguish between genuine and simulated amnesia in

either clinical or experimental situations. Claims of amnesia are readily accepted when there is palpable evidence of brain damage. It should be understood, however, that evidence of a significant interpersonal or sociocultural component does not necessarily mean that functional amnesia is faked. Rather, it means that functional amnesia is complex. Hypnosis may be a state of altered consciousness, but it is also a social interaction; thus, it should not be surprising to discover that the subject's response to amnesia suggestions will be influenced by the precise wording of the suggestion, the discourse context in which it is embedded, the subject's interpretation of the hypnotist's words, and perceived social demands. The social context is probably important in the organic amnesias, but its role is magnified in their functional counterparts.

IV. AMNESIA THROUGH THE LIFESPAN

Some forms of amnesia occur naturally in the course of psychological development. For example, adults rarely remember much from their early childhoods; the earliest memory is typically dated between the third and fourth birthdays, and is limited to a relatively small number of isolated fragments until about 5 or 7 years of age. The appearance of childhood amnesia is not merely an artifact of the long retention interval between childhood encoding and adult retrieval; something special seems to happen to memories for childhood events. Infantile amnesia, covering the first year or two of life, may be attributed at least in part to the lack of language and to the immaturity of the neocortex and other critical brain structures. However, the exact mechanism for childhood amnesia, covering the years after the second birthday, remains uncertain.

The classic explanation for childhood amnesia was proposed by Freud. In his view, during the phallic stage of psychosexual development the child resolves the Oedipus complex by repressing infantile sexual and aggressive impulses, as well as any thoughts, images, and memories that might be related to them. Since (according to the theory) all the young child's mental life is concerned with these topics, all of early childhood is repressed—except a couple of banal *screen memories* that aid repression by giving the person something to remember. Recall that the major goal of psychoanalysis is to lift the repressive barrier so that patients can acknowledge and cope realistically with their primitive

instinctual urges. Other theories emphasize the relationship between cognitive processes employed at encoding and retrieval. For example, Schachtel proposed that memories encoded by pre-oedipal, primary-process modes of thought cannot be retrieved by post-oedipal, secondary-process schemata. A similar account can be offered from Piaget's perspective, emphasizing the incompatibility between sensory-motor and preoperational encodings and the retrieval processes characteristic of concrete and formal operations. Note that all these theories predict that memories of childhood experience should be accessible to young children, who have not undergone the "five-to-seven shift." In contrast, some theorists have argued that young children simply do not possess the information-processing capacity—specifically, the ability to pay attention to two things at once, like an event and its episodic context—required to encode retrievable memories. In this case, the prediction is that children will know little more about their childhood histories than adults do. [See COGNITIVE DEVELOPMENT.]

It should be noted that infantile and childhood amnesias affect only memories for personal experiences. Children acquire a vast fund of information, and a considerable repertoire of cognitive and motor skills, which they carry into adulthood. Whether this selectivity reflects merely the effects of constant rehearsal, or reveals a dissociation between EM and IM similar to that observed in the clinical amnesias, is not clear.

At the other end of the life cycle, it appears that even the healthy aged have difficulty learning new information and remembering recent events. Aging has little effect on primary or short-term memory, as reflected in digit span or the recency component of the serial-position curve; but it has substantial effects on secondary or long-term memory, especially after moderately long retention intervals. Again, the deficit primarily affects episodic memory: the elderly do not lose their fund of semantic information (although they may be slower on such tasks as word-finding) and their repertoire of procedural knowledge remains intact, provided that they have been able to maintain these skills through practice.

At the same time, it should be noted that episodic-semantic comparisons almost inevitably confound type of memory with retention interval. Memories for recent experiences have, by definition, been encoded recently; most semantic knowledge was acquired while the individual was relatively young. Surprisingly, little is known about the ability

these kinds of dissociations are observed in all sorts of amnesia—not just the amnesic syndrome, but in traumatic retrograde amnesia, psychogenic amnesia, and posthypnotic amnesia as well—strengthens the conclusion that the theoretical distinctions are psychologically and biologically valid.

Of particular interest in recent theory are the various dissociations between explicit and implicit expressions of episodic memory. To date, three broad classes of theories have been proposed to explain these dissociations; each has several exemplars. According to the *activation* view, the activation, by a current event, of pre-existing knowledge representations is sufficient for IM; but EM requires elaborative activity, in which individually activated structures are related to each other. According to the *processing* view, IM is an automatic consequence of environmental stimulation, while EM occurs by virtue of controlled processes that are limited by attentional resources. According to the *memory systems* view, IM reflects the activity of a perceptual representation system, which holds information about the form and structure of the objects of perception, and EM reflects the activity of an episodic memory system that represents knowledge about the meaning of events and the context in which they occur.

Research on the amnesic syndrome, including studies of both human patients and animal models, indicates that the medial-temporal lobe, including the hippocampus, entorhinal cortex, and perirhinal and parahippocampal cortex, forms the biological substrate of explicit memory. But the diencephalic form of amnesic syndrome seems to indicate that the mammillary bodies and the dorsomedial nucleus of the thalamus are also critical for memory. As research continues, investigation of amnesia will make a unique and valuable contribution to understanding the relation between explicit and implicit memory, and the biological foundations of each. [See MEMORY, NEURAL SUBSTRATES.]

At the same time, evidence of preserved memory functioning offers new insights concerning amelioration and rehabilitation in cases of amnesia. Loss of explicit memory has debilitating consequences for afflicted individuals in everyday life. They are often unable to keep track of events, remember appointments or schedules, engage in educational or vocational pursuits, or manage home activities. Attempts at rehabilitation have frequently focused on restoration of damaged explicit memory processes either through the use of repetitive drills or by teaching

patients mnemonic strategies such as visual imagery or verbal elaboration. These retraining attempts have met with limited success. There is no evidence that exercising damaged neural or cognitive mechanisms leads to positive outcomes; and although patients have sometimes been able to acquire a few pieces of information by using mnemonic techniques, they do not use the strategies spontaneously in everyday life.

On the other hand, rehabilitation strategies that have focused on providing compensatory devices designed to bypass problems in daily life have been somewhat more promising. External aids such as notebooks, diaries, alarm watches, and environmental labels have enabled some amnesic patients to function somewhat more independently, although use of such devices often requires considerable amounts of training and practice. The microcomputer, potentially a powerful prosthetic for people with memory impairments, has yet to be extensively used for this purpose.

The finding that implicit and procedural memory often remain intact even in cases of severe amnesia has recently prompted researchers to begin to explore ways in which these preserved processes might be exploited beneficially for rehabilitation purposes. Cuing techniques, which take advantage of amnesic patients' ability to respond normally to word-stem or fragment cues, have been used successfully to teach individuals new factual information such as vocabulary as well as procedural tasks such as data-entry and word-processing. Continued research in this direction, paralleling more theoretically based research concerning preserved memory functions in amnesia, should enable further progress toward improving the ability of amnesic individuals to function effectively in their everyday lives.

Acknowledgments

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