# 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 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.

**Implicit memory** In contrast to explicit memory, which entails conscious recall or recognition of past events, implicit memory refers to any effect of a past experience on subsequent experience, thought, or action – for example, priming effects.

**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.

**Priming** A phenomenon where an event facilitates (positive priming) or impairs (negative priming) performance on another, later task.

**Source amnesia** A phenomenon where a subject retains access to new information acquired during a learning experience, but forgets the learning experience itself; it is commonly reflected in *cryptomnesia*, or unconscious plagiarism.

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

**Trauma-memory argument** The notion, rooted in the nineteenth century ideas of Sigmund Freud and Pierre Janet, that psychological trauma causes amnesia by virtue of a process of repression or dissociation.

**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

*Amnesia* may be defined as a special case of forgetting, where 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 personal identity as well as her fund of autobiographical memories. Amnesia includes frank pathologies encountered in neurological and psychiatric clinics, such as the amnesic syndrome, Alzheimer's disease (AD), traumatic retrograde amnesia (RA), and the interpersonality amnesia characteristic of multiple personality disorder (MPD). But it also includes abnormalities of memory observed ubiquitously, such as infantile and childhood amnesia, the exaggerated forgetfulness associated with normal aging, and the memory failures associated with sleep and general anesthesia. These naturally occurring failures of memory have their counterparts in amnestic states induced in otherwise normal, intact individuals by means of experimental techniques, such as electroconvulsive shock (ECS) in laboratory rats and posthypnotic amnesia in college sophomores.

Experimental research on memory began with the publication of Ebbinghaus' *Uber das Gedachtniss* (*On Memory*) in 1885, but the clinical description of amnesia dates from even earlier. Korsakoff first described the amnesic syndrome that bears his name in 1854. And in 1882, Ribot published *Les Maladies de la memoire* (*Diseases of Memory*), with a detailed description of the consequences for memory of brain insult, injury, and disease, as well as a unified theory of memory and amnesia. On the basis of his observations, and Hughlings Jackson's principle that ontogeny recapitulates phylogeny, Ribot concluded that brain disorder produces a progressive loss of memory that affects memories in the reverse order of their development. Thus, in traumatic RA, memories for events occurring immediately before the accident are most likely to be lost. This principle, now known as *Ribot's law*, does not always hold, but it was an important first step in the journey from clinical description to scientific theory.

For reasons that are not completely clear, clinical and experimental study of amnesia languished for the first half of the twentieth century, but was revived by observations of a patient, H.M. (full name: Henry Molaison), who became amnesic following surgical resection of portions of his medial–temporal lobes, including the hippocampus, in a desperate attempt to relieve intractable epileptic seizures. From the time of his operation in 1953, at the age of 27, until his death in 2009 at the

age of 82, he had no conscious recollection of any episode in his life. In addition, George Talland's 1965 pioneering monograph, *Deranged Memory*, reported an extensive psychometric and experimental study of amnesic patients with Korsakoff syndrome. These studies ushered in a 'golden age' of amnesia research in which clinicians and experimentalists joined forces under the banner of *cognitive neuropsychology* (later, renamed cognitive neuroscience) – a discipline that attempts to integrate evidence obtained from the intensive study of brain-damaged patients with theories of normal cognitive function.

## The Amnesic Syndrome

The amnesic syndrome represents a profound deficit in learning and memory; it is by far the most commonly studied pathology of memory. Its most characteristic feature is a gross anterograde amnesia (AA), meaning that the person cannot remember events that have occurred since the time of the brain damage. Short-term memory (as measured by digit span, for example) is unimpaired; but after even a few moments' distraction, these patients cannot remember what they have said or done, or what has been said or done to them, just recently. In the classic case, the patient's cognitive deficits are specific to long-term memory: general intelligence, perception, reasoning, and language functions are spared. But this AA is associated with several different etiologies, and these disparate origins are associated with somewhat different patterns of memory and cognitive deficit. For more details, see 'Amnesia and the Brain' by Race and Verfaellie, following this article.

One form of the amnesic syndrome, now known as *diencephalic amnesia*, was first described by Sergei Korsakoff in association with alcoholism. These patients typically show RA as well as AA, meaning that they also have difficulties in remembering events from their premorbid life, especially those from the years immediately preceding their disease. Remote memory, such as for childhood events, is apparently preserved. Note that such a pattern conforms to Ribot's law. Chronic alcoholics often suffer from a deficiency of vitamin $B_1$ (thiamine) which results in bilateral damage to structures of the diencephalon, including the upper portion of the brainstem, the mammillary bodies, the dorsomedial nucleus of the thalamus, and the mammillothalamic tract. The acute phase of illness, known as Wernicke's encephalopathy, is characterized by confusion, disorders of visual function, and ataxia; Korsakoff's syndrome emerges as the chronic phase (the entire course of illness is sometimes known as Wernicke–Korsakoff syndrome). Although this disease is now typically prevented by the introduction of vitamin-enriched commercial foods, other etiologies, including vascular insufficiencies, tumors, and frontotemporal brain damage can have similar effects.

Another form of amnesic syndrome, known as *temporal lobe amnesia*, stems from bilateral lesions in the medial portion of the temporal lobe, and especially the hippocampus, entorhinal cortex, and surrounding structures (again, there are also material-specific amnesias resulting from unilateral damage to these structures). The most famous case is patient H.M., but there are many other famous cases. Other cases have been caused by brain tumors, ischemic episodes, head trauma, and herpes encephalitis. Temporal-lobe amnesics show AA by definition. The occurrence of RA in these patients is somewhat controversial, and may vary if damage extends beyond the hippocampus itself. H.M. showed little RA; another patient, R.B., showed an RA covering only about 1 or 2 years prior to his surgery; yet another, K.C., displayed an RA covering his entire premorbid life.

There is also *frontal-lobe amnesia*, which is qualitatively different from the amnesic syndrome. Frontal-lobe patients are not globally amnesic, but they frequently show deficits on tasks requiring memory for temporal order, as well as memory for the source of newly acquired knowledge. They also lack *metamemory* capabilities – that is, they have little appreciation of the contents stored in their own memories, or in the availability of appropriate memory strategies. Patients who have frontal damage in addition to diencephalic or medial–temporal lobe damage experience their greatest difficulties on memory tasks requiring strategic planning and organization, suggesting that frontal damage may impair executive function, rather than memory per se.

Finally, *transient global amnesia* is a temporary (typically lasting several hours) condition characterized by sudden onset. It closely resembles the permanent diencephalic and medial–temporal lobe amnesias, in that it involves both AA and RA, but – as its name implies – it is brief. The condition, while frightening, is benign: after remission, there are no signs of permanent brain damage (and little risk of another episode in the future). Transient global amnesia appears to be caused by temporary vascular insufficiency affecting brain tissue; interestingly, many cases appear in association with physical exertion or mental stress.

In the absence of permanent brain damage, something akin to the amnesic syndrome may be observed in cases of *alcoholic blackout*. Blackout involves amnesia without loss of consciousness. The intoxicated individual may engage in conversation or perform other actions normally; but after regaining sobriety, he or she will have no memory for the episode. Blackouts are most commonly observed in chronic alcoholics, though they do occur to nonalcoholics who are severely intoxicated. In any case, blackout is most likely to occur when the person ingests large quantities of alcohol rapidly, especially when fatigued or hungry. Alcohol folklore suggests that the amnesia is an instance of state-dependent retrieval – that is, that the memories return when the person resumes drinking. However, laboratory research clearly indicates that the memories covered by blackout are unrecoverable, and thus that the amnesia reflects an encoding deficit. Sedative drugs, such as barbiturates and benzodiazepines, also produce irreversible AA.

The different patterns of task performance offer clues about the nature of the memory deficit in the amnesic syndrome. In principle, any instance of forgetting may be attributed to a failure at one or more of three stages of memory processing: encoding (the creation of a memory trace of a new experience), storage (the retention of trace information over time), and retrieval (the recovery of trace information for use in ongoing experience, thought, and action). Logically, a syndrome that affects memory for postmorbid but not premorbid events is most likely due to encoding failure. And, in fact, it has been suggested on the basis of laboratory experiments with lesioned rats and monkeys that the hippocampus and other structures in the medial–temporal lobe mediate the consolidation and storage of new memories. An alternative formulation assumes

that representations of the various elements of an event are distributed widely in the cortex, and that the hippocampus creates a kind of index and binds the elements together. In either case, the occurrence of AA means that the hippocampus is crucial for memory formation, even though the memories themselves are not stored there.

What about the RA? Some degree of RA is usually, but not always, observed in the amnesic syndrome. Logically, damage to a structure that consolidates and organizes new memories should have no effect on old ones. In some cases, RA may reflect the disruption of premorbid memories that were incompletely consolidated at the time of disease onset; this would produce a temporal gradient, but the extent of RA observed would seem to imply that proper consolidation requires weeks, months, or years instead of seconds, minutes, or hours. On the other hand, if the hippocampus serves a binding and indexing function, its destruction will create an RA by effectively preventing the retrieval of memories that remain available in storage; this would produce an amnesia for remote as well as recent memories and would not necessarily produce a temporal gradient. In some cases, what appears as RA may in fact be AA, reflecting the slow onset of an insidious disease process – and producing the appearance of a temporal gradient; this suggestion is particularly plausible in the case of diencephalic amnesia associated with chronic alcohol abuse but cannot account for amnesias of sudden onset. It has also been suggested that the RA may reflect the fact that, once activated and retrieved, premorbid memories are then *re*-encoded: as a result, the same encoding failure that produces the AA for postmorbid events results in a progressive loss of premorbid memories as well, and emergence of RA.

Even conclusions about encoding deficits must be qualified to some extent. At first glance, the AA observed in the amnesic syndrome appears to be a complete inability to acquire new information. However, closer examination indicates that certain aspects of learning and memory are spared even in the densest cases of amnesia. Thus, the patient H.M. learned to solve the Tower of Hanoi puzzle, but he did not recognize the puzzle as familiar. Amnesic patients who study the word *ELATED* do not remember it just minutes later; but when presented the stem *ELA* and asked to complete it with the first word that comes to mind, they are more likely to produce *ELATED* (as opposed to *ELASTIC* or *ELABORATE*) than would be expected by chance. The ability of amnesic patients to acquire cognitive and motor skills, and to show priming effects in word-stem completion and other tasks, shows that they are able to acquire new information through experience – although, somewhat paradoxically, they do not remember these experiences and may not be aware that they possess this knowledge.

The limits of such learning are still being studied, but already they have motivated a distinction between two expressions of episodic memory, *explicit* and *implicit*. Explicit memory (EM) refers to the conscious recollection of a previous episode, as in recall or recognition. By contrast, implicit memory (IM) refers to any change in experience, thought, or action that is attributable to such an episode, such as skill learning or priming. The dissociation between EM and IM in amnesic patients indicates that some forms of learning and memory are preserved. According to one view, amnesics suffer from a specific inability to encode declarative knowledge about specific events, but retain an ability to acquire procedural knowledge. This would account for their ability to acquire new cognitive and motor skills. Preserved priming has been attributed to the automatic activation of declarative knowledge structures that were stored prior to the brain damage, or to the encoding of new episodic representations in a primitive perceptual memory system that lacks the kinds of information (e.g., about the meaning of an event, or its spatiotemporal context) that would support EM.

## Traumatic RA

Another form of amnesia occurs as a consequence of head trauma. A very severe blow to the head can bruise gray matter and shear white matter, producing both cortical and subcortical damage that may result in AA and RA similar to that observed in the amnesic syndrome. Even in the absence of such damage, a blow to the head can result in a concussion, or temporary cessation of electrical activity in the cortex and loss of consciousness. The recovery of consciousness begins with the return of simple reflexes, then the gradual return of purposeful movement, and then speech (another pattern predicted by Ribot's law). After the patient appears fully oriented, he or she will display an AA for some time, as well as an RA for the accident itself and the events leading up to the accident. Typically, the AA is immediate, that is, it will start at the time of the trauma. But if the loss of consciousness is delayed, the onset of the AA will be delayed as well. Such *lucid intervals* suggest that the AA is a result of vascular complications that may take some time to develop.

The RA in traumatic amnesia is characterized by a temporal gradient, meaning that it is densest for events nearest the time of the accident – yet another example of Ribot's law at work. However, the gradient is broken by *islands of memory* consisting of isolated events, not necessarily personally important, that are remembered relatively well. The extent of the RA is correlated with the extent of the AA. Although the memories covered by the AA are permanently lost, apparently reflecting an encoding deficit, the RA gradually remits. It was once thought that this recovery began with the earliest memories and proceeded forward, which again would be predicted by Ribot's law. Although the most recent events are generally recovered last, more careful studies show that the shrinkage of amnesia is accomplished by filling in the gaps that surround the islands of memory, leaving a *final RA* covering the accident itself and the moments or minutes leading up to it, and perhaps a few *islands of amnesia*. The shrinkage of amnesia clearly indicates that traumatic RA is a disorder of retrieval, and that the islands of memory act as anchors to support the recovery process. However, the final RA may reflect either a loss of memory from storage or, more likely, a disruption of consolidation.

A nontraumatic form of RA is observed in psychiatric patients who are administered *electroconvulsive therapy* (ECT) for acute affective disorder. In ECT, electrical stimulation (e.g., 100 V, 500 mA for 500 ms), delivered from surface electrodes applied over the temporal lobe, induces a convulsive, tonic–clonic seizure not unlike those of *grand mal* epilepsy; after a short series of such treatments (e.g., 6–10 sessions over 2–3 weeks), patients often experience a rapid return to their normal mood state (ECT is not a cure, as episodes of

depression or mania may recur). Because they are anesthetized when the treatment is delivered, patients experience no pain or distress from the convulsions themselves; because they receive muscle relaxants, the convulsions do not result in bone trauma. However, the seizure does produce both AA and RA as adventitious consequences (i.e., unrelated to treatment success). The RA shows the same sort of temporal gradient observed following concussive blows to the head. Because there is less memory impairment (though no difference in treatment outcome) with unilateral than with bilateral electrode placement, ECT is usually delivered to the nondominant hemisphere. The RA gradually clears up (except for the moments before ECT is actually delivered), but memories affected by the AA cannot be recovered.

The amnesia induced by ECT shows a dissociation between EM and IM similar to that observed in the amnesic syndrome. In one experiment, patients who studied a list of words within 90 min following administration of ECT showed a deficit in recognition, but no deficit in priming on a word-stem completion test. In another study, patients who read word strings presented in mirror-reversed fashion before delivery of ECT later showed an advantage in reading those words, even though they failed to recognize these words as familiar.

What about the memories covered by the final RA? Although ECS may disrupt encoding processes, it does not appear to remove the memory traces from storage. The relevant evidence comes from studies of the effects on memory of ECS administered to animals. A common research paradigm is called one-trial, step-down, passive avoidance learning. A rat is placed on a shelf above an electrified floor. If the animal steps down, it receives a footshock, and jumps back up on the shelf. Under ordinary circumstances, the animal will not return to the floor: it learns in one trial to avoid the shock by doing nothing. But if the animal received a dose of ECS, after it recovers, it steps down onto the floor after being placed on the shelf. It is as if it has forgotten all about the shock.

ECS-induced amnesia shows a temporal gradient similar to that observed in other forms of traumatic RA. If the ECS is delayed from the time of the original learning experience, there is less amnesia than if it is administered immediately afterward. But the extent of amnesia also depends on how memory is measured. The same time at which the amnesic animal steps down (as if the footshock never happened), it shows a marked increase in heart rate (as if it is afraid). Moreover, if the animal receives *reminder treatments* such as tail shock in another environment or immersion in circulating ice water, it will remain on the shelf and avoid the floor. The *desynchrony* between behavioral and psychophysiological indices of fear is analogous to the dissociation between EM and IM observed in human amnesic patients; and the effectiveness of reminder treatments shows that at least some aspects of the forgotten event have been preserved. Memories covered by the final RA may never be accessible to conscious recollection, but they may nonetheless be expressed as implicit memories.

## Functional Amnesia

Clinically significant amnesias are not confined to cases of organic brain syndrome. Psychiatrists and clinical psychologists sometimes encounter forms of *functional amnesia* in a group of mental illnesses known as the *dissociative disorders*. Functional amnesia may be defined as a loss of memory that is attributable to an instigating event that does not result in insult, injury, or disease to the brain. Because there is no evidence of head injury, such memory failures are also labeled as *psychogenic amnesia*. In the classic formulation, which owes much to Sigmund Freud and Pierre Janet, traumatic stress causes amnesia via a psychological process variously labeled as *repression* or *dissociation*. Several forms of functional amnesia are listed in the *Diagnostic and Statistical Manual of Mental Disorders* under the category of the *dissociative disorders*, all of which entail a disruption of conscious memory and identity.

In the twentieth century, the *trauma-memory argument* became an important fixture of popular culture as well as clinical folklore, but empirical evidence in its favor has been surprisingly lacking. Careful prospective analyses indicate that the vast majority of victims of documented trauma, including childhood sexual abuse, remember their experiences perfectly well. Any forgetting that is observed appears most likely due to infantile and childhood amnesia (see later paragraphs), the mere passage of time, or a failure to appreciate the nature of the experience at the time it occurred (meaning that the 'trauma' was not experienced as such). Moreover, both human and animal studies indicate that high levels of physiological arousal, such as would accompany traumatic stress, enhance rather than impair memory – probably due to the activation of β-adrenergic receptors. The problem with patients suffering from posttraumatic stress disorder is not that they cannot remember their trauma; it is that they cannot forget it.

In *dissociative amnesia*, the patient cannot remember specific events, usually covering a continuous period of time, resembling RA; there is no AA. As in the amnesic syndrome and traumatic RA, the patient's fund of semantic and procedural knowledge remain intact. Compared to traumatic RA, dissociative amnesia appears to be more extensive and longer lasting. Clinical lore holds that dissociative amnesia can be reversed by hypnosis or barbiturate sedation, but evidence for the reliability of recollections produced by these techniques is largely lacking. Because of concerns about the inherently suggestive nature of hypnosis, most American legal jurisdictions forbid witnesses from offering testimony based solely on hypnotically 'refreshed' memories.

*Dissociative fugue* entails a more extensive loss of autobiographical memory, covering the whole of the person's life, a loss and/or change in identity, and sometimes physical relocation (from which symptom the syndrome derives its name). Such cases often come to the attention of police and health providers when a person cannot identify himself; or when she comes to herself in a strange place and does not know how she got there. Interestingly, fugue patients lose self-knowledge and autobiographical memory, but they do not seem to lose their fund of semantic memory, or their repertoire of procedural knowledge. Upon recovery the patient is left with an amnesia covering the events of the fugue state itself, and retains no knowledge of whatever identity he or she may have adopted in that state. Examination of such cases after they are resolved sometimes reveals an instigating episode of psychological stress.

In *dissociative identity disorder* (DID; formerly known as MPD), two or more personalities appear to inhabit a single body, alternating control over experience and action. 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 'alter egos' 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. The most widely accepted theory of DID holds that it develops in defense against abuse, trauma, or deprivation in early childhood – but again, actual evidence for a causal link between trauma and any form of amnesia is very weak.

Reports of DID were relatively common in the clinical literature before 1920, and then virtually disappeared. There was a resurgence of DID, bordering on epidemic, beginning in the 1970s, but it is not clear how many of these cases – and there were hundreds if not thousands of them tabulated in the literature – were iatrogenic in nature, or simply misdiagnosed. Where the alternate personalities were initially elicited through hypnosis or other special techniques, or when an amnesic barrier was absent, the case is especially suspect.

DID is sometimes offered as an insanity defense, claiming that a second personality is actually responsible for crimes of which the first personality is accused. DID does raise 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 his or her defense; and techniques intended to elicit testimony from a personality may violate constitutional safeguards against self-incrimination. However, DID has rarely proved successful as a defense against criminal charges.

Several experimental studies confirm the existence of interpersonality amnesia in DID. 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 RA in ECT and ECS, the functional amnesias seen in the dissociative disorders 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.

Like the organic amnesias, posthypnotic amnesia selectively affects episodic as opposed to semantic or procedural memory. 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 being 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 and elsewhere. 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 may be important in the organic amnesias, but its role is magnified in their functional counterparts.

## Amnesia Through the Life Span

Some forms of amnesia occur naturally in the course of psychological development. For example, adults rarely remember much from early childhood: 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 and childhood amnesia* affects 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 the dissociation between EM and IM similar to that observed in source amnesia, is not clear.

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) the young child's entire mental life is concerned with these topics, all memories of early childhood are 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, Ernst Schachtel proposed that memories encoded by preoedipal, 'primary-process' modes of thought cannot be retrieved by postoedipal, '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' (so named because of the major cognitive change occurring between these ages) between preoperational thought and concrete operations. 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.

Although infantile and childhood amnesia are often attributed to autochthonous aspects of cognitive and neural development, it is clear that the child's interactions with other people are extremely important determinants of whether he or she will remember some past event. After all, as Ulric Neisser has pointed out, the 'five-to-seven year shift' is not simply a matter of moving from the preoperational period to concrete operations (or, for that matter, from before to after the acquisition of a theory of mind). It is also when the child first goes to school, and moves into an environment that is more structured with respect to time and place – thus affording the child an opportunity to distinguish one event from another. Even before the child enters school, research by Katherine Nelson, Robyn Fivush, Judith Hudson, and others underscores the important role played by joint reminiscence between child and parent in shaping the child's appreciation of narrative structure, including the causal as well as temporal relations among events, thus strengthening individual memories and connecting them both with each other and with the present.

At the other end of the life cycle, it appears that even the healthy aged have difficulty learning new information and remembering recent events. *Normal 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 become slower on such semantic-memory 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 the type of memory with retention interval. Memories of 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 of older individuals to learn new vocabulary or acquire new world knowledge. The aged do show impairment in episodic memory for remote events, but it is not clear whether this reflects age differences in retrieval processes, or simply the effects of the retention interval and opportunities for proactive and retroactive interference.

A relatively recent topic in research on aging memory compares EM and IM. Compared to the young, the aged show definite impairments on EM (especially free recall, less so on recognition); but they show less deficit, or none at all, on IM tasks such as stem completion. Part of the reason for their problems with EM may lie in the difficulty that the elderly have in processing contextual information. Spatial context, temporal context, and source are necessary for distinguishing one event from another, and thus crucial to conscious recollection. Whether this difficulty is specific to contextual features of events, or merely a reflection of a more general limitation on cognitive resources, is unclear.

Memory problems are confounded in the *dementing illnesses* often associated with aging – for example, AD. The severe memory problems associated with AD are likely related to the increase of neuritic plaques and neurofibrillary tangles, particularly in medial–temporal regions of the brain. These changes, as well as neuronal loss and depletion of neurotransmitters in other cortical and subcortical areas, especially the hippocampus and other medial–temporal lobe structures, contribute to the extensiveness of the disease process. Both AA and RA emerge early in the course of these diseases, and progressively worsen. In contrast to the amnesic syndrome, however, the memory deficit in dementia affects 'short-term' as well as 'long-term' memory, and forms part of a larger cluster of deficits affecting a broad swath of cognitive and emotional life, including impairments in semantic and procedural memory as well as episodic memory. In the latter stages of their illness, demented patients may show *anosognosia*, or a lack of awareness of their deficits.

Does the abnormal forgetting observed in aging and dementia extend to IM as well as EM? Research on this question is still at a very early stage, but already it seems fairly clear that IM is relatively spared in normal aging. Thus, elderly subjects fail to recognize studied words, but show priming effects on word-fragment completion. With respect to AD and other forms of dementia, however, some controversy remains. There is some evidence of intact motor-skill learning in AD patients, but there is also evidence of impaired performance on priming tasks. The issue is complicated by the fact that AD is

a progressive illness. Although impairments in EM may be observed quite early in the course of the disease, deterioration of IM may wait until later stages.

## Amnesias of Everyday Experience

Amnesia is a symptom of neurological or psychiatric disorder, but it is also something that occurs in the ordinary course of everyday living. The most familiar example is *sleep*. A great deal transpires while we are asleep, including events in the external environment and endogenous activity such as dreams, nightmares, and (in some cases) episodes of somnambulism (sleepwalking) and somniloquy (sleeptalking), but virtually none of this is remembered in the morning. In fact, our inability to remember what has been happening is often the phenomenological basis for inferring that we have been asleep. Similarly, attempts at sleep learning have been almost uniformly unsuccessful, leading investigators to conclude that we are able to learn during sleep only to the extent that we stay awake. A paradox here is that a large body of evidence now indicates that sleep plays an important role in the consolidation of *presleep* memories.

Most investigators explain sleep-induced amnesia in terms of an encoding deficit or consolidation failure. According to this view, the low levels of cortical arousal characteristic of sleep effectively impair complex information-processing functions. Thus, events in the environment are not noticed, relevant information in memory is not retrieved, and traces of new experiences are not encoded in retrievable form. Some evidence favoring this view comes from studies of memory for dreams. Sleepers who are awakened during REM sleep almost invariably report a dream, apparently by virtue of retrieval from primary memory; but dreams are rarely reported upon awakening in the morning, which requires access to trace information in secondary memory. However, subjects will remember a dream in the morning if they awaken directly out of REM sleep. And dreams reported during REM awakenings will be accessible in the morning, provided that the dreamer has remained awake long enough to rehearse the dream before returning to sleep.

Most evidence of sleep-induced amnesia comes from studies of EM, leading to speculation that evidence of memory for sleep events, including successful sleep learning, might be obtained with measures of IM. Research on this topic has only just begun, but the available evidence is negative. When care is taken to insure that there is no evidence of cortical arousal indicative of awakening, subjects show neither EM nor IM for events that occurred while they were sleeping. Even if positive evidence for sleep learning were obtained, it would almost certainly not be as efficient as learning in the normal waking state.

Amnesia is also an important component of *general anesthesia* induced in surgical patients. Clinically, the success of general anesthesia is indicated by the patient's lack of response to instructions, suppression of autonomic and skeletal responses to incisions and other surgical stimuli, and absence of retrospective awareness of pain and other events occurring during surgery. Thus, by definition, amnesia is a consequence of adequate general anesthesia. But, as with sleep, the amnesia is always assessed in terms of EM, leaving open the possibility that even adequately anesthetized patients might show IM for surgical events. Some anecdotal evidence favoring this proposition is provided by occasional cases in which patients awaken from surgery with an inexplicable dislike of their surgeon – an attitudinal change which is plausibly traced to unkind remarks made about the patient by members of the surgical team.

In recent years, this question has been the object of considerable investigation, and in fact research employing paradigms derived from studies of the amnesic syndrome has sometimes, but not always, provided evidence of spared IM. Thus, patients who are presented a list of words during surgery, sometimes show significant priming effects. Such effects are not always obtained, however; and even when they are obtained, they are relatively small. Certainly the scope of information processing during general anesthesia cannot compare to what is possible when the patient is awake and properly oriented; for example, IM after anesthesia may well be limited to the processing of the physical properties of stimuli, but not their meaning. What accounts for the different outcomes across the available research is not clear. Perhaps some anesthetic agents impair EM but spare IM, while others impair both. Such a result might yield interesting insights about the biological foundations of memory.

## Theoretical and Practical Implications

Research on amnesia is intrinsically interesting, but it also has theoretical and pragmatic implications. At the theoretical level, amnesia engages our attention because it seems to carve nature at its joints. Amnesia is selective, and the difference between those aspects of memory that are impaired in some form of amnesia, and those that are spared, promises to provide information about the processes underlying memory functioning and the organization of memory into different systems. Such conclusions are based on the *logic of dissociation*. In single dissociations, Variable A affects performance on Task Y but not Task Z; in double dissociations, Variable A affects Y but not Z, while Variable B affects Z but not Y; in reversed associations, changes in A increase Y and decrease Z, while changes in B decrease Y and increase Z; in stochastic independence, performance on Task Y is uncorrelated with performance on Task Z. All other things being equal, differences such as these suggest that the tasks in question differ in qualitative terms. If they were only quantitatively different, they would be correlated with each other, and influenced by the same variables.

Such dissociations are commonly observed in amnesia. For example, the fact that the amnesic syndrome affects the recency portion of the serial-position curve, but not the primacy component, has been cited as evidence that primary (short-term) and secondary (long-term) memory are qualitatively different memory systems, perhaps with different biological substrates (one affected by the brain lesion, the other not). Evidence from amnesia also has been used to support other structural distinctions: between declarative and procedural knowledge and – within the domain of declarative knowledge – between episodic and semantic memory. Thus, amnesic patients have

difficulty learning new factual information, but retain an ability to acquire new cognitive and motor skills; and if they do retain new factual knowledge, they display amnesia for the circumstances in which this information was acquired. Logic and experience tell us that when something breaks, it does so along natural boundaries, which form lines of least resistance. When a disorder of memory separates past memory from new learning, procedural and declarative knowledge, or episodic and semantic memory, it tells us that these distinctions, conjured in the minds of theorists, actually mean something in the real world. The fact that these kinds of dissociations are observed in all sorts of amnesia – not just the amnesic syndrome, but in traumatic RA, 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 preexisting 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 that 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 EM. 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. And, of course, the hippocampus itself is a complex structure, with many elements that may each play a special role in memory processing. As research continues, investigation of amnesia will make a unique and valuable contribution to understanding the relation between EM and IM, and the biological foundations of each.

At the same time, evidence of preserved memory functioning offers new insights concerning amelioration and rehabilitation in cases of amnesia. Loss of EM has debilitating consequences for afflicted individuals in everyday life. Amnesic patients 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 EM 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 hand-held computer, potentially a powerful prosthetic for people with memory impairments, has yet to be extensively used for this purpose probably because of the problems in teaching amnesic individuals how to use such a device.

The finding that IM and procedural knowledge 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.

*See also:* Aging and Cognition; Alzheimer's Disease; Amnesia and the Brain; Dissociative Disorders; Episodic Memory; Hippocampal Formation; Hypnosis; Memory; Memory, Neural Substrates; Posttraumatic Stress Disorder.

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