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AMNESIA

See: ALZHEIMER'S DISEASE: BEHAVIORAL ASPECTS; ALZHEIMER'S DISEASE: HUMAN DISEASE AND THE GENETICALLY ENGINEERED ANIMAL MODELS; AMNESIA, FUNCTIONAL; AMNESIA, INFANTILE; AMNESIA, ORGANIC; AMNESIA, TRANSIENT GLOBAL; HEAD INJURY; KNOWLEDGE SYSTEMS AND MATERIAL-SPECIFIC MEMORY DEFICITS; REHABILITATION OF MEMORY DISORDERS

AMNESIA, FUNCTIONAL

Analyses of learning and memory increasingly attempt to take account of clinical and experimental research on victims of amnesia. Most of this literature has focused on pathologies of memory associated with demonstrable brain lesions or the administration of centrally acting drugs. The functional amnesias are a collection of memory disorders instigated by processes that do not result in damage or injury to the brain but that do engender a marked increase in forgetting.

Amnesia in the Dissociative Disorders

One major category of functional amnesia occurs within the context of diagnosable psychopathology, especially the dramatic "Dissociative Disorders" listed in the Diagnostic and Statistical Manual (DSM) (4th edition) of the American Psychiatric Association. In current diagnostic nosology, this category includes a wide variety of syndromes whose common core is an alteration in consciousness affecting memory and identity.

In dissociative amnesia (also known as psychogenic amnesia, limited amnesia), the patient suffers a loss of autobiographical memory for specific past experiences. It sometimes occurs in cases of violent crime (interestingly, affecting either victims or perpetrators), war neurosis, and other types of posttraumatic stress disorder. Unfortunately, many reports of dissociative amnesia are anecdotal and lack independent corroboration of the purported instigating event. Moreover, it is not possible to reliably distinguish genuine cases of psychogenic amnesia from simulated cases.

In dissociative fugue (psychogenic fugue, functional retrograde amnesia), the amnesia covers the whole of the individual's past life and his or her personal identity; there may also be physical relocation (which gives the syndrome its name). The condition may go unnoticed until the patients are asked personal questions that they cannot satisfactorily answer. Recovery typically begins with the patient's recognition of loss of identity. Recovery of identity and memory per se may occur spontaneously or in response to the appearance of a relative or other salient cue. When the fugue is resolved, the patient is typically left with a limited amnesia covering the period of the fugue.

In dissociative identity disorder (multiple-personality disorder), a single individual appears to manifest two or more distinct identities, each alternating in control over conscious experience, thought, and action. Before World War II, the typical case involved only two or three such "ego states"; more recent cases have tended to present more alter egos, leading some to speculate that iatrogenic and sociocultural factors may account for much of the multiple-personality epidemic of the 1980s. In genuine cases, the personalities are separated by an amnesic barrier. The dissociation may be symmetrical, in which each ego state is ignorant of the other(s) or, more commonly, asymmetrical, in which case an ego state may be aware of some of its counterparts but ignorant of others.

In depersonalization the person believes that he or she has changed in some way or is somehow unreal; in derealization the same beliefs are held about one's

surroundings. Because these beliefs are objectively inappropriate, these experiences can be construed as disorders of memory: the person fails to recognize some object, self, or situation with which he or she is objectively quite familiar. Episodes of depersonalization and derealization frequently occur in response to stress and in association with anxiety disorders; they may also be induced by psychedelic drugs and occur spontaneously in a substantial proportion of the normal population.

Although dissociative disorders have been of interest at least since the time of Freud and Janet, they rarely have been studied with controlled experimental procedures. For example, little is known about psychogenic amnesia beyond anecdotes. A few cases of fugue and multiple personality have been studied in the laboratory, but we have no idea how representative they are. Nevertheless, the available evidence suggests a pattern of selective memory deficit in some respects resembles that observed in organic amnesia. Thus, psychogenic fugue impairs memory for past experiences and other aspects of self-knowledge but leaves the patient's repertoire of impersonal procedural and semantic knowledge largely intact. Dissociative identity disorder displays a similar pattern.

Nonpathological Amnesias

In other forms of functional amnesia, dramatic forgetting occurs in the ordinary course of everyday living, albeit with no implication of pathology. For example, people commonly fail to remember their dreams and other events of the night's sleep; attempts to demonstrate sleep learning have been almost uniformly unsuccessful. Theoretical accounts of this memory deficit usually revolve around encoding factors. For example, one hypothesis holds that sleep inhibits the higher cortical centers that support perceptual processing.

Another example of nonpathological functional amnesia is the general paucity of memory for infancy and childhood. As with sleep, most theoretical accounts of this developmental amnesia focus on encoding factors. For example, infantile amnesia (covering the first two years of life) may reflect the child's relative inability to encode symbolic and especially linguistic representations of events; even older children may lack the information-processing capacity to encode retrievable memories.

A dramatic form of forgetting known as posthypnotic amnesia occurs in some hypnotized subjects. In some respects, posthypnotic amnesia may serve as a laboratory analogue of the dissociative amnesias seen in the clinic.

Explicit and Implicit Memory

While the functional amnesias by definition impair explicit memory, some anecdotal and experimental evidence suggests that the amnesia may spare implicit memory, or the unconscious influence of past events on subsequent experience, thought, or action. In dissociative identity disorder, for example, both procedural learning and priming effects may transfer between personalities, so that one alter ego is influenced by the experiences of another even though the amnesic barrier prevents conscious recollection. The situation is complicated, however, because not all forms of implicit memory are equally spared. There has been no experimental corroboration of clinical claims that special procedures such as hypnosis and barbiturate narcosis can promote conscious access to the "lost" memories.

Among nonpathological amnesias, the dissociation between explicit and implicit memory is especially well documented in posthypnotic amnesia. Preverbal infants can show long-term retention of new learning in a manner that suggests implicit memory. However, sleep-learning procedures do not appear to leave any traces, even in implicit memory.

Trauma, Repression, and Dissociation

The lack of reliable evidence of brain damage has fostered a tendency to account for functional amnesias in purely psychological terms. Since the nineteenth century, repression and dissociation have been the favored explanations. Repression, as defined by Freud, is the motivated forgetting of material (typically, relating to sexual or aggressive ideas and impulses) that conflicts with physical reality or social sanctions. Dissociation, as discussed by Janet and Prince, is a more adventitious "splitting off" from awareness of a set of percepts, memories, thoughts, or feelings. While Freud argued that repressed contents could be known only by inference (because they were expressed only symbolically, as in dream contents), Janet argued that dissociated contents could be recovered directly, by hypnosis and other means. Given the pervasive influence of Freudian psychoanalysis in twentieth-century discourse, the repression thesis long held sway. There has been a subsequent revival of the concept of dissociation, as indicated by adoption of "Dissociative Disorder" as a category in the DSM. An eclectic combination of Freudian and Jungian theories formed the basis of clinical theories about trauma and memory that emerged in the late twentieth century. These theories, in turn, promoted the 1980s revival of therapeutic strategies based on the recovery and "working through" of traumatic memories.

Paradoxically, the idea that trauma plays a role in amnesia, while a salient aspect of clinical folklore, is not supported by solid empirical evidence. Animal studies indicate that high levels of emotional arousal stimulate the release of stress hormones that activate the amygdala, leading to enhanced memory. A review of more than sixty longitudinal studies of documented trauma survivors yielded not a single instance of amnesia that could not be accounted for by nontraumatic infantile and childhood amnesia or “organic” factors such as intoxication, anoxia, or head injury. Many clinical studies alleging “repression” of trauma also fail to rule out such factors, especially in cases of infantile and childhood amnesia that occur independently of trauma. Others fail to confirm that the ostensibly traumatic event even occurred or misinterpret subjects’ failure to disclose their trauma as a failure to remember it.

There are very few corroborated reports of the therapeutic recovery of traumatically lost memories. Most clinicians apparently assume their patients’ memories are valid or refrain from challenging them in order to protect the “therapeutic alliance.” The techniques of “memory work” used therapeutically to help patients “recover” traumatic memories also increase the risk of memory distortion and confabulation. There is no evidence that either hypnosis or sedation by barbiturate drugs facilitates the recovery of valid traumatic memories. Whenever “recovered” memories are taken as evidence, whether in the clinic or the courtroom, it is critical that they be accompanied by independent, objective, corroborative evidence.

Neuropsychology of Functional Amnesia

Beginning with the discovery of the syphilis spirochete, a major goal of psychiatry has been to transfer syndromes from the functional to the organic category as their neural bases are revealed. It seems likely that neuroscientific research will pinpoint such causal relationships for the dissociative amnesias as well. For example, a recent PET study of dissociative fugue found no sign of the activity in the right posterior frontal and anterior temporal lobes that usually accompanies recollection of emotionally salient personal experiences but did find activation in the left frontal and temporal regions. Although *functional* may eventually prove to be a misnomer, for now the term delineates a class of amnesias in which psychological processes rather than brain insult, injury, or disease are the immediate causes of the memory failure.

See also: AMNESIA, INFANTILE; AMNESIA, ORGANIC; AMNESIA, TRANSIENT GLOBAL; HYPNOSIS AND MEMORY

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AMNESIA, INFANTILE

Do you remember being born? Your first birthday party? Your first day of school? Despite the significance of these early experiences, most adults recall little or nothing about them. The absence of autobiographical memory for events that occurred during infancy and early childhood is commonly referred to as infantile (or childhood) amnesia. Sigmund Freud originally identified the phenomenon of infantile amnesia by asking his patients to describe their earliest personal memories in the course of therapy. On the basis of these patient reports, Freud argued that the period of infantile amnesia extended into the sixth or eighth year of life. Freud’s most often-cited explanation of infantile amnesia was highly influenced by his patient population. He believed that memories for our infancy and early childhood were stored in pristine condition, but were actively repressed due to their emotionally and sexually charged content. In fact, one goal of Freud’s psychoanalytic process was to “unlock” these hidden memories to allow patients to come to terms with the traumatic thoughts and experiences of their childhood.

Subsequent normative studies of adults’ earliest memories have shown that Freud probably overestimated the period of infantile amnesia. There is now general consensus that adults’ earliest autobiographical memories are for events that occurred when they were approximately three to four years of age (Bruce, Dolan, and Phillips-Grant, 2000; Dudycha and Dudycha, 1941; Mullen, 1994; Sheingold and Tenney, 1982; Waldfogel, 1948) or even slightly younger (MacDonald, Uesiliana, and Hayne, 2000; Usher and Neisser, 1993). Furthermore, normative studies of adults’ earliest memories have failed to provide any empirical evidence in support of Freud’s repression model (Pillemer and White, 1989).

Thus, the fundamental question remains: Why is it that we have little or no recollection of events that occurred during our infancy and early childhood? Although repression does not provide an adequate explanation for the phenomenon, empirical studies point to a number of other factors that might account for infantile amnesia (Howe and Courage, 1993).

The Lower Boundary for Long-Term Recall of Early Experiences

Maturation of the Central Nervous System

Maturation of the human brain begins at conception, but continues throughout childhood (and beyond). Although our understanding of the time course of human brain development is not complete, we do know that many of the brain areas that play a role in long-term memory are not fully mature during infancy and early childhood. Thus, although learning occurs rapidly during this phase of development, the ability to retain and use information over a lifetime may be precluded by the immaturity of the brain (Campbell and Spear, 1972).

Maturation of two areas of the brain—in particular, the medial temporal lobe (including the hippocampus) and the frontal cortex—is thought to play a particularly important role in the phenomenon of infantile amnesia (Bachevalier, 1992). Maturation of the hippocampus occurs relatively early in development and may be sufficient to support some of the sophisticated memory skills exhibited by infants; however, maturation of the higher-association areas of the frontal cortex continues well into childhood and may be required for the maintenance and retrieval of memories over the long term (Hayne, Boniface, and Barr, 2000; C. Nelson, 1995).

The Development of Language

When we ask adults to recall their earliest personal memories, we commonly ask them to provide a verbal report of what they can remember—both the instructions they are given (“tell me about your earliest memory”) and their response to those instructions require sophisticated language skills. Infants and children, on the other hand, typically express their memories, by necessity, through nonverbal behaviors. Even once they have acquired conversational language skills, children still rely primarily on their nonverbal skills to solve tasks that require memory. Furthermore, the ability to translate early, preverbal experiences into language is extremely limited, if not impossible (Simcock and Hayne, 2002). Although an early preverbal memory may be reflected in some aspect of an adult’s behavior (Newcombe, Drumme, Fox, Lie, and Ottinger-Alberts, 2000), he or she will be unable to provide a verbal report of the original experience. In this way, language development is another rate-limiting step in the offset of infantile amnesia.

Beyond the Basic Ingredients: The Emergence of Autobiographical Memory

The basic ingredients for long-term verbal memory are in place by the end of the second year of life.