Global Amnesia: Organic and Functional Considerations

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I. The Concept of Amnesia

In its most general sense, amnesia is defined as failure of the memory. In clinical usage, the term is used in a more circumspect sense: amnesia is defined as an inability to recollect information in a verbal fashion, although the ability to speak is retained. Such conditions as alexias (memory loss for reading) or apraxias (memory loss for certain motor activities) are not formally considered to be amnesias.

Since amnesias are disorders of (verbal) memory, some knowledge of the memory process is necessary before one can study or treat the disorder. The search for a wholly adequate definition for "memory" has bedevilled psychologists for generations. For our purposes, Bartlett’s definition will suffice: “memory is an imaginative reconstruction built out of our attitudes towards a whole mass of organized past reactions to experience.”1 The establishment of a memory for an object is commonly thought to be the result of a three-step process: 1) registration—the object is perceived through the senses and attention is paid to the object; 2) retention (storage)—the physical substrate of the idea is established, and the idea is integrated in some way with older memories; 3) recall (retrieval)—the idea (now a memory) can be elicited anew through association with some new perception. Different amnesias are thought to act at different steps of this process.

In addition, memories are said to have two important subjective properties: 1) Time ordering: one perceives some events as having happened before other events. All events are perceived in a time dimension. 2) The sense of personal identity and personal identification: an individual can distinguish past events which are part of his immediate personal experience from past experiences and events conveyed to him from secondary sources. Different amnesias can also interfere with these general properties of memory.

A. Diagnostic Considerations

The various amnesias can be most simply and clearly described with the aid of some basic parameters. Firstly, the most obvious question: what is forgotten? In temporal amnesias, all verbal memories are abolished for a certain period of the patient's life. By contrast, in categorical amnesia, all verbal memories are abolished for a category of events or things; the category is banished from the patient's memory without regard for when it was placed in time.2 A special case of categorical amnesia is the amnesia of personal identity, in which the patient has no recollection or sense of self-identification with his past, although he might remember such non-personal information as historical facts. In some cases elements of both temporal and categorical amnesia can be combined. An example is global amnesia, in which all verbal memories appear to be abolished, including the sense of personal identity.

The second question we can ask about amnesia is: through what period in memory-

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time does the amnesia extend? The most common amnesia is retrograde amnesia (RA), which is a failure of memory for events preceding the amnesic trauma or disease. The time scale of RA can vary from just a few minutes before the trauma in some individuals to the entire lifespan of other individuals. Somewhat less common is anterograde amnesia (AA) or post-traumatic amnesia (PTA) which is a failure of memory for experiences subsequent to the trauma.

Thirdly, we can ask what are the precipitating and predisposing causes of the amnesia? The precipitating cause of an amnesia can be either traumatic (a physical injury or emotional shock) or non-traumatic (the result of a disease process). For certain amnesias, a cluster of personality attributes is thought to be predisposing.

Lastly, we must ask how long does it take the patient to recover his memory, and how much of the memory is recovered? Attention to these four parameters helps to distinguish the various amnesias and affords some insight into their etiology.

B. Organic vs. Psychogenic

The amnesias are conventionally divided into the organic versus the psychogenic. This distinction has been clouded somewhat by the realization that an otherwise “psychogenic” amnesia can be precipitated by a physical trauma which has caused a mild degree of organic damage. Furthermore, emotionally charged memories show more lability to physical traumas (such as electroshock) than do neutral memories. Nevertheless, if we adhere to our established parameters, we can make some distinctions between amnesias labelled as predominantly organic and amnesias labeled predominantly psychogenic:

1) With organic amnesias there is a general disorientation and memory failure (i.e., a temporal amnesia). With psychogenic amnesias the memory loss is often more acute for personal events than for items of general knowledge and information. 2) With organic amnesias which result from trauma, the RA does not extend beyond one to two weeks, and the AA does not extend beyond four weeks. With psychogenic amnesias, the RA and AA are much less likely to adhere to such a time scale. With the organic amnesias, the length of the RA and time spent in amnesic coma are directly related to the severity of the injury; no such simple relationship can be established for psychogenic amnesias.

3) As mentioned, both organic and psychogenic amnesias can be triggered by a physical trauma. A much less severe trauma can be adequate to precipitate a psychogenic amnesia. Furthermore, the psychogenic amnesiac frequently is depressed, with an unstable, poorly integrated personality in the pre-amnesic state. Severe marital or financial problems often precede the amnesic episode. Such predisposing factors are not found for predominantly organic amnesias.

4) Lastly, the prognosis differs markedly for organic and psychogenic amnesias. The organic amnesiac regains his memory only gradually following organic treatment. However, he never regains memory of the trauma itself. Prior to remission, there is usually a period of disorientation in time and place. The psychogenic amnesiac frequently will show an abrupt remission, sometimes within 24 hours; with the help of sodium amytal or hypnosis, recollection of the traumatic episode itself may be achieved.

On the basis of these observations, it has been presumed that organic amnesias interfere with the registration and/or retention phases of memory, while psychogenic amnesias interfere with recall only. Organicity and psychogenicity may well define a continuum, with any individual case lying somewhere between these poles.

a. Traumatic and Non-Traumatic

The organic amnesias can be divided into the traumatic and the non-traumatic. The traumatic amnesias are characterized by an abrupt onset and a transient duration: they include temporal lobe seizures, electroshock, and the post-concussive states. The non-
Traumatic amnesias have a gradual onset and have a more extensive duration; they include Korsakoff’s syndrome and degenerative cerebral and vascular diseases.\textsuperscript{12} Behaviorally, patients suffering from both traumatic and non-traumatic amnesia display an impairment of recent memory and immediate recall, as demonstrated in both clinical interviews and standardized tests. Patients suffering from the non-traumatic amnesias display gross impairments of remote memory, while the traumatic amnesics generally do not.\textsuperscript{13} With respect to etiology, the non-traumatic amnesias have been ascribed to degenerative neuropathies of the limbic system, usually the result of a nutritional or vascular insufficiency.\textsuperscript{14} The etiology of traumatic amnesia is more subtle, and it is the immediate focus of our attention.

The triggering cause of a traumatic amnesia is usually a blow to the head, electroshock, or a penetrating wound to the brain. The closed head injuries (cerebral concussion and electroshock) are immediately followed by a period of confusion and impairment of consciousness.\textsuperscript{15} The penetrating wounds (such as a bullet through the temporal lobes) can cause a traumatic amnesia without any intervening impairment of consciousness. The closed head injuries reflect a widespread disruption of normal brain physiology that is both instantaneous and transient. The traumatic event itself is never recalled and it seems fair to assume that it was never registered or retained due to direct interference with the mechanism which elaborates memory traces.

A 24-year-old male medical student was learning how to draw blood. After having his own blood drawn by another student, he felt a little weak and sat down on a stool. He slipped off the stool and bumped the back of his head on the floor. He lost consciousness for thirty seconds. Upon regaining consciousness, he did not know where he was; he thought it was early morning and he was still in bed, and he could not understand why people were standing over him. He couldn’t recognize the people around him (although they were his classmates). He regained his memory and his orientation in time within ten minutes. The remission was gradual but complete. He recalled the post-traumatic episode in detail but he never remembered the traumatic head injury (how he “ended up on the floor”).\textsuperscript{16}

The effects of concussion being both widespread and reversible, no specific neuropathology has been detected. In contrast, numerous anatomical studies have localized Korsakoff’s amnesia to lesions of the medio dorsal nucleus of the thalamus, the mammillary bodies and the fornix.\textsuperscript{17} Penetrating brain injuries have been localized in an adjacent region, the floor and walls of the third ventricle and the infero-mesial portions of both temporal lobes.\textsuperscript{18} The effects of concussion and electroshock being more widespread, it seems quite possible that they disrupt the neurophysiology of the reticular formation and the neocortex, as well as hippocampal-diencephalic structures, and this could explain the disruption of consciousness, the confusion, and the amnesia for the traumatic event \textit{per se}. In other words, the closed-head injuries could disrupt the registration phase of memory-trace formation by interfering with attention.

\textbf{b. Psychogenic}

Patients displaying psychogenic amnesias are usually categorized as hysterics or malingerers. The hysterics are considered repressors (amnesia due to an unconscious process) while malingerers are considered suppressors (amnesia due to a conscious process).\textsuperscript{19} A further distinction is that the hysterical forgets an experience for internal reasons (it threatens his self-image) while the malingerer forgets an experience for external reasons (to avoid punishment by society).\textsuperscript{20} Let us concentrate on hysterical amnesia.

In studying an hysterical amnesia, the crucial question is to determine exactly what is forgotten. Most likely, the patient's non-personal memory is relatively adequate and the amnesia is for some incident in which the patient was involved. The logical extreme is illustrated by the hysteric who presents a complete amnesia of personal identity. Pre-
disposing factors for hysterical amnesia include an unstable, poorly integrated personality,
nevagistic, with severe neurotic traits. The precipitating crisis usually involves problems
in marriage or a severe financial difficulty. An etiology for hysterical amnesia was
presented by Freud, and most current authors concur to this day. The hysteric represses
a memory because it threatens his self-image. The repression successfully splits the
emotion from the event, so even if the event is recalled, it is less threatening and the
hysteric feels no personal identification with it. In review, the observation of Abelas
and Schilder is worth repeating. “On the whole, (hysterical) amnesia is a weak attempt
of a weak personality to escape conflicts which are chiefly conflicts of actual life.”

Two examples of hysterical amnesia will suffice as illustrations:

(1) A 43-year-old male patient suffered from a loss of personal memory which had
persisted for 3 years. During these 3 years a localized personal memory had built up;
all previous information concerning his private life in the first 40 years of his life
had been lost. Under barbiturate narcosis he reenacted a war episode. Upon emerging
from narcosis the memory of his previous life was restored.

(2) A 26-year-old male was admitted to a hospital displaying total amnesia for his
entire personal life although his fund of general knowledge and information appeared
normal. Four days after admission he was hypnotized. Under hypnosis he recalled
having a few drinks at a bar, getting involved in a fight, attempting to strangle his
opponent, and then being knocked to the floor by two men. Upon emerging from
hypnosis his memory returned to normal. (We note that an hysterical amnesia episode
may or may not begin with a physical trauma.)

c. Transient Global Amnesia (TGA)

A most fascinating and complex amnesic syndrome is transient global amnesia (TGA). No amnesic syndrome is more completely categorized, and no amnesic syndrome is
more incompletely understood. TGA appears to strike only the middle-aged and the
elderly; it involves a total failure of memory for all events in the present and recent
past; it lasts for a period of hours only, during which the patient is bewildered and
confused, but is nonetheless fully conscious, rational, and maintains a sense of personal
identity; there is no defect of remote memory; onset is abrupt and recovery spontaneous,
with no recollection of the events that occurred during the attack.

Is TGA principally an organic or psychogenic amnesia? Is it traumatic or non-
traumatic? Despite the presence of about 50 accounts of TGA in the literature, no de-
finite answer can be given to these questions. The TGA patient usually presents quite
normal neurological signs: there is a tendency toward hypertension, but this is not
remarkable considering that all TGA patients have been middle-aged or elderly. A
number of cases of TGA have followed swimming exercise, and at least two have
followed sexual intercourse. These two bits of information have led to the suggestion
that TGA may be the result of a reversible vascular insufficiency in the region of the
hippocampus. However, a recent report challenges these assumptions. The case is
distinctive in that a physician was present during the acute attack and that an extensive
work-up was performed.

The patient is a 54-year-old male electrical technician, free of neurological complaints
prior to the event. The TGA apparently began at home in the evening after work at
5 p.m. According to his wife, the episode began when she noticed him drinking a
second glass of bourbon (the patient usually had only a single glass). The patient
“looked paralyzed” and said it was the only drink he had poured. After a succession
of similar episodes the physician next door was summoned and the patient eventually
was admitted for neurological and psychiatric exams at the Columbia-Presbyterian
Medical Center. The neurological exam was normal and the vital signs were normal
(except for bilateral arcus senilis). The patient was amnesic for present events and
for all events of the last four years. He was rational, concerned about his situation,
and retained his sense of personal identity. The patient experienced a complete remission at 2 a.m., with no RA except for the period of 5 p.m. to 2 a.m. (A subsequent alcohol tolerance test proved normal.)

This careful study lends scant support to the hypothesis that TGA is a traumatic amnesia involving a transient vascular insufficiency. No trauma (either swimming or sex) preceded the episode, and no hypertension was found (although undetectable vascular irregularities cannot be ruled out). The EEG was normal, including hyperventilation and photic stimulation tests. Furthermore, the TGA syndrome is virtually never known to recur, and this further frustrates theorizing on its etiology.

d. Global Amnesia

More severe than TGA is total or global amnesia. Global amnesia differs from TGA in at least three important respects. 1) The retrograde aspect of global amnesia is more severe. During the acute phase of TGA, memory may be lost for the present and for the last few years. In global amnesia, memory is lost for all events preceding the trauma, but a post-traumatic memory may be gradually established. 2) TGA is readily and spontaneously reversible, while global amnesia is not. 3) Global amnesia can involve an amnesia of personal identity, while personal identity is maintained in TGA.

Global amnesia per se has received little attention in the literature. This is because global amnesia has often not been distinguished from its close analog, the amnesia of personal identity. The loss of the sense of personal identity is such a cardinal symptom that a secondary problem such as the loss of impersonal memory and general information is often ignored. Since the global amnesic syndrome and the amnesia of personal identity have not been clearly distinguished in the past, it is not now possible to compare and contrast the two syndromes. A further clarification of the nature of global amnesia must await more information on the syndrome, and the following case is presented in that context.

II. A Case History

The patient was a forty-year-old married man referred for psychiatric evaluation by his attorney. He was involved as a plaintiff in a Workman's Compensation action against his employer, alleging head trauma many months prior and subsequent deficits. The existing medical file was not very helpful.

Present Situation: The history when first obtained was somewhat unclear and incomplete because of the patient's lack of total memory for both the injury and anything prior to it. As best he could reconstruct the day of the accident, he had supposedly punched in at the regular time of 8:00 p.m. at his work and had then been noted several hours later wandering disoriented and with a big bump on his forehead. He was eventually found by somebody walking around aimlessly near the bakery where he worked. He was taken to the hospital, where he remained for over three weeks. For the immediate period after the injury, i.e. two or three days, he was totally disoriented, incoherent and very negativistic toward any of the facts with which he was presented. Because his memory lack persisted, he had to be reintroduced to his family and friends and also to his home and other activities.

His current symptoms were as follows. Global memory deficit for everything up to and including the accident, and this included any and all aspects of his life. What he now knew, several months after the accident, he maintained was through relearning and being re-exposed to things, rather than a direct memory of anything. Such memory deficit was total, and there were not even glimpses of any memory of anything previous. In addition, his recent memory and orientation were not as good as usual, in that he forgot what he had recently done or lost track of time. He also suffered from continuous and terrific headaches which fluctuated in severity and were also associated with throbbing.
involving the posterior aspect of his head and neck. He also suffered from heartburn with occasional vomiting at night. He reported that a diagnostic evaluation of this had revealed nothing. His wife reported that he was much more temperamental and irritable than usual, and that he tended to sleep for long periods of time, often 14 to 16 hours at a time.

**Background:** Most of the history was obtained originally from his wife, and it was remarkable that she knew and could report little prior to their marriage some eight years previously. The patient was born in California. The wife reported that his father had never been close to him, and that his mother had died some 20 years previously. He had minimal contact with any of his siblings. The mother was described as a martyr type. Because of the father's bad disposition, the patient left home when he was very young, and worked and traveled all over the country. He had supposedly worked as a cook in one city and in a lumber yard in another state. His first marriage was not a happy one for the patient, and it ended when the older son fell to his death from a window. The patient had worked in his last occupation for the preceding five years, regularly up to the time of the accident. The wife described him as a terrific worker, very interested in coins, fishing and camping, and fond of all sports. He was also a good card player, but was socially very withdrawn and had few friends.

**Mental Status:** The patient was poorly oriented, and his memory was impaired for current activities, beyond the already mentioned total amnesia for the period prior to the accident. His speech was hesitant and at times vague. His affect was rather flat and mechanical, and did not show the distress expected from one who had suddenly lost all his memory. In describing himself, he claimed that he knew nothing prior to the accident, and that now he was hot-headed and difficult to get along with. He denied strange ideas or suspiciousness. He did not perform mathematics well, and there was some evidence of perseveration. He did a fair job of reciting digits forwards and backwards. There was definite concreteness as well as looseness and inappropriateness in his handling of proverbs. His intelligence was estimated clinically to be clearly impaired at the time of the examination.

**Neurological Evaluation:** The neurologist found no evidence of organic neurological disease at the time of the examination. He considered the symptoms as "quite bizarre" and not having a "ring of organicity." The neurological exam was completely within normal limits, and an electroencephalogram was interpreted as normal.

**Psychological Testing:** The full scale I.Q. was 104 (performance 113, verbal 96), with considerable variance from task to task. There was marked impairment in his attention, concentration and memory. Judgment and reasoning were remarkably intact. He was also found to be depressed and obsessively preoccupied with his somatic disturbances. He relied heavily on denial and seemed to protect himself through withdrawal. The findings were interpreted as most likely due to brain injury.

**Sodium Amytal Interview:** After the above evaluations were completed, it was decided to conduct such an interview with the hope both of obtaining more details surrounding the accident itself, and also elucidating, if possible, the causes and sources of his impaired memory currently. The procedure was very successful technically, as well as in terms of results. The patient was found to be able to tolerate very high dose levels of the barbiturate drug. For quite a while he resisted any breakthrough and continued to maintain that whatever he was telling us was from material relearned rather than direct memory.

Then at one point he began to describe the type of work he did at the bakery, and it became gradually apparent that he was recounting from direct memory and not from anything he had relearned since. From this he proceeded to give us a very detailed account of the day in question, as well as a lot of other material about his current and previous life. On the evening in question, he left his house normally, riding on his motorcycle, and had a near accident when a car backed out of a driveway. He
fell slightly from his motorcycle, but did not injure himself. He picked himself up and after he got the motorcycle going again went on to work, where he punched in normally and had coffee with his colleagues. Some two and one half hours later, after he had delivered some bread to the lockers, as he walked back, he bumped his head against a metal object, and as he fell backwards also injured his neck. He was dazed and disoriented, and told one of his colleagues what happened. He still remained uncomfortable and decided to walk out of the job and just to walk through the streets, for reasons that are unclear to him. It was at this point that he was found. The patient went on to tell us a little about his previous life, including the fact that as a young teenager his father had placed him in an institution, and that he had been bounced from one institution to another over the next many years. He then talked a little about his job experience leading up to his current marriage. About the latter, he expressed a great deal of dissatisfaction in that both his wife and his mother-in-law were very domineering and difficult, and had placed him and his children in a very unpleasant situation. He talked about how he didn’t dare to oppose or stand up to his wife, because doing so just led to her being more domineering and difficult. It became apparent that his amnesia was a primitive way to deal with the situation, about which he was both at a loss and desperate.

Epilogue: The legal case was settled less than a year after the evaluation. The patient did not follow through with the recommended therapy. Occasional subsequent contacts revealed that the situation remained unchanged. A year or so later, immediately following a cerebral angiogram (performed because of persistent headaches) he “came to” dramatically and remembered his original accident and other aspects of his life. This return of memory was neither complete nor persistent. The level of amnesia has subsequently fluctuated, and has included variable “blackouts.” The elements of the organic syndrome have otherwise persisted, and he has not managed to return to any type of work.

Discussion: The case presented represents a pure example of global amnesia, rendered even more unusual by the total length of the syndrome. It was triggered and terminated by physical trauma. The psychological dynamics were clear and fascinating. There has also been fair evidence, continuing to date, of diffuse organic damage. This case illustrates the complexity of the process called amnesia, and supports the notion of an elaborate admixture of organic and psychogenic factors.

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