Amnesia

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I. Organic Amnesia

II. Stress Related and Transient Amnesic States

episodic memory store of personally experienced events from the past, including details not only of the content of the episode but also of its context — when and where it occurred. **semantic memory** our general knowledge about the world, such as information about words and their meanings, facts, concepts, objects and people; typically retrieved without recollection of where and when the information was initially acquired.

hippocampal complex structures located in the medial temporal lobe of the human brain (e.g., the hippocampus, subiculum, parahippocampal gyrus, and entorhinal cortex); implicated in the learning and temporary storage of recent memories.

temporal lobe/neocortex the area of the brain considered the permanent store of our episodic and semantic memories.

The term *amnesia* refers to a partial or total loss of memory and can be due to organic brain damage and/or some forms of psychological stress. The loss of memory can affect events or experiences occurring either subsequent to the onset of the disorder (anterograde amnesia) or those that took place prior to its appearance (retrograde amnesia). Furthermore, while amnesia most often occurs for personal autobiographical events (*episodic memory*), some patients also show a loss of *semantic memory*, our factual knowledge about the world.

I. Organic Amnesia

The most well studied case of organic amnesia in the literature is that of HM, who became amnesic in his mid-20s after an operation to relieve symptoms of severe epilepsy. HM's operation involved surgery to the *hippocampal complex* and parts of the temporal lobe in both brain hemispheres. Although HM's epilepsy improved following surgery, he became profoundly amnesic, unable to remember events just after they had happened (anterograde amnesia), or those that had occurred up to a few years prior to his operation (retrograde amnesia). There are now over fifty reported patients who, like HM, have exhibited an impairment to new learning after damage to the hippocampal complex. It is thought, therefore, that this region in the human brain is critically involved in the acquisition of new episodic and semantic memories.

As well as being unable to remember events from moment to moment, HM's memory impairment extended back in time for a number of years prior to his surgery. This pattern of memory loss, in which memories from the distant past (e.g., childhood, early adulthood, etc.) are remembered better than more recently experienced events (e.g., from the day, week or year before), is a phenomenon that has fascinated memory researchers for over 100 years. The fact that memory loss can be so dramatically affected by the age of the memory suggests that the system that is critical for the acquisition of human memories, the hippocampal complex, is not necessarily involved in the permanent storage of all our episodic and semantic memories. It has been suggested that other areas of the brain, such as the temporal lobes, are the location for our enduring stores of human memory. Support for this hypothesis comes from patients who show the opposite pattern of memory impairment to HM and other patients with damage to the hippocampal complex: better recall of recent memories compared to those from childhood, early

2

adulthood, etc. Crucially, these patients typically present with selective damage to the temporal neocortex with sparing of structures in the medial temporal lobe. Current theories of human memory propose, therefore, that the hippocampal complex and the temporal neocortex play distinct, but complementary, roles in the acquisition and storage of human memory.

II. Stress Related and Transient Amnesic States

Compared to instances of organic amnesia, patients with stress related impairments to memory are rare. One of the main types of non-organic memory loss is called psychogenic or dissociative amnesia. The memory impairment is characterized by a loss of one's identity and of personal autobiographical memories from the past (retrograde amnesia). It is commonly transient in nature and patients often show a full recovery after the appropriate treatment or therapy. The cause and nature of the amnesia varies from case to case, but in many circumstances the memory loss can be linked to a precipitating stressor in the person's life (e.g., financial and health worries, accidents, violence, natural disasters, etc.). For example, studies of soldiers following World War II suggested that up to 5% had no memory for the traumatic combat events they had just experienced.

Although one of the diagnostic criteria for psychogenic amnesia is that there is no evidence of structural brain damage, the increasing use of sophisticated neuroimaging techniques that measure levels of blood flow in the brain suggest that there can be altered brain function in some cases, although the reasons for this are currently unclear. For example, it has been shown that a patient with a persistent psychogenic amnesia for the whole of his past life processed his autobiographical memories in the same way that control subjects processed nonpersonal memories. This study suggests that some patients with psychogenic amnesia may treat previously personally salient episodic memories in a neutral, 'semantic' way as if they no longer belong to them, in order to escape the emotional associations of the traumatic episode(s) that triggered the amnesia.

The important link between stress and memory can be further revealed in a syndrome called Transient Global Amnesia (TGA). Although not termed a psychogenic amnesia (patients are rarely confused about their own identity) the disorder is characterized by abrupt confusion and a complete anterograde amnesia, which eventually shrinks after some hours (typically 4-6).

3

While there are known risk factors for TGA, such as a history of epilepsy or migraine, in as many as 30% of cases attacks can be directly linked to precipitating stresses, such as strenuous exertion, pain, immersion in water, and emotional events. Recent neuroimaging studies have revealed physiological changes (low blood flow) in the hippocampal complex during an attack. Extreme stress may result in memory impairment because the hippocampal complex is part of a complicated network in the human brain that mediates fear-related behavior and stress responses, as well as aspects of memory function. Highly stressful situations may overstimulate the hippocampus and related structures leading to decreased blood flow in this region of the brain and subsequent memory loss.

Further Reading

- Baddeley, A.D., Wilson, B.A., & Watts, F.N. (1995). *Handbook of Memory Disorders*. Wiley, New York.
- Cohen, N.J. & Eichenbaum, H.B. (1993). *Memory, Amnesia, and the Hippocampal System*. MIT Press, Cambridge, MA.
- Parkin, A.J. (1998). *Case Studies in the Neuropsychology of Memory*. Psychology Press, Hove, East Sussex, UK.

Squire, L.R. (1987). Memory and Brain. Oxford University Press, Oxford, UK.