Matthew Q response to Week 4 discussion 1 post

Anterograde Amnesia

Introduction

What is anterograde amnesia? This week anterograde amnesia will be discussed. Included in this discussion will be a discussion defining this type of amnesia, the symptoms of Anterograde amnesia, brain mechanisms of anterograde amnesia, and the most common etiologies of anterograde amnesia. Anterograde amnesia is the most common form of amnesia when compared to retrograde amnesia (Carlson & Birkett, 2017).

Anterograde amnesia is generally, brain damage that causes the inability to learn new things (Carlson & Birkett, 2017). However, more specifically in anterograde amnesia, “basic abilities of perceptual learning, stimulus-response learning, and motor learning are intact, but that complex relational learning is gone” (Carlson & Birkett, 2017).

Symptoms of Anterograde Amnesia

The symptoms of anterograde amnesia are fairly straight forward. The symptoms include a disturbance in memory marked by an inability to learn new information (APA Dictionary of Psychology, 2014).  The symptoms are an ability to remember events that occurred in the past, from the time before the brain damage occurred, but an inability to retain information encountered after the brain damage (Carlson & Birkett, 2017).

Brain Mechanisms of Anterograde Amnesia

            There are a few brain mechanisms and brain structures involved in anterograde amnesia. Anterograde amnesia can also be caused by damage to the temporal lobes, where the hippocampus is located, (Scoville& Milne, 1957, as cited in Carlson & Birkett, 2017). Anterograde amnesia involves the role of the hippocampus as it relates to memory and learning. The hippocampus converts short-term memories into long-term memories (Carlson & Birkett, 2017). It is theorized that that, “short-term memory of an event is retained by neural activity and that long-term memories consist of relatively permanent biochemical or structural changes in neurons” (Carlson & Birkett, 2017). Therefore, a person would not be able to remember new information if there is no permanent record made of it.

            There has been evidence found that damage restricted to the hippocampal formation produces anterograde amnesia, including deficits in consolidation (Zola-Morgan et al., 1986 as cited in Carlson & Birkett, 2017).  There have been several studies that reported damage to the region of the hippocampal formation called field CA1 caused anterograde amnesia (Kartsounis et al., 1995; Rempel-Clower et al., 1996; Victor and Agamanolis, 1990 as cited in Carlson & Birkett). The field CA1 of the hippocampus is sensitive to anoxia. This is because of the fact that this area of the brain is especially rich in NMDA receptors (Carlson &Brikett, 2017). Metabolic disturbances of disparate types—including, “seizures, anoxia, or hypoglycemia—cause glutamatergic terminal buttons to release glutamate at abnormally high levels. The effect of this glutamate release is to stimulate NMDA receptors, which permit the entry of calcium. Within a few minutes, excessive amounts of intracellular calcium begin to destroy the neurons” (Carlson &Brikett, 2017).

            In addition to the CA1 field of the hippocampus, there have been researchers that have found other areas that are involved with anterograde amnesia. Researchers have found “growing clinical evidence that retrosplenial cortex dysfunctions contribute to both anterograde amnesia” (Aggleton, 2014).

Most Common Etiologies of Anterograde Amnesia

The most common etiology of anterograde amnesia is brain damage to the region of the hippocampal formation called field CA1by metabolic disturbances of disparate types—including, “seizures, anoxia, or hypoglycemia (Carlson &Brikett, 2017).

Conclusion

This discussion has shown that anterograde amnesia is an inability of a person to learn new information but can still remember old information from before the incidents causing brain damage to the hippocampal formation in field CA1. The symptoms, brain structures involved, and the etiologies of anterograde amnesia have been examined in detail.

References

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