A review article on, memory types and general memory dysfunction

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Abstract
Memory is the part of the brain by which data or information is encoded, stored, and retrieved when needed. Memory is having a various class and its subtypes. For the memory impairment various physiological factors are affected e.g. changing lifestyle age, sex etc. Memory includes the types of memory and subtypes. Some diseases and disorders are included e.g. Parkinson’s disease, Alzheimer’s disease, Huntington’s disease, Vascular disorders and Tourette syndrome.

Keywords: Psychology, alzheimer’s, attention-deficit disorder, depression, anxiety

Introduction
Memory function is exposed to a variety of pathologic processes including neurodegenerative diseases, strokes, tumors, head trauma, hypoxia, cardiac surgery, malnutrition, attention-deficit disorder, depression, anxiety, the side effects of medication, and normal aging [1, 2]. Memory loss is often the most disabling feature of many disorders, impairing the normal daily activities of the patients and profoundly affecting their families. Some approach about memory, such as the concepts of “short-term” and “long-term,” Memory is the ability to capture externally or internally presented information, store it, and reconstruct it later. Forgetfulness is one of the most frequent symptoms in patients presenting to cognitive disorders clinics. This article approaches memory function and dysfunction from a clinical perspective [3].

Classification of memory
One widely used classification is based on the involvement of consciousness in memory use. Consciously evoked memories are declarative (explicit) while memories that do not need conscious involvement are non-declarative (implicit) [3]. Declarative memory or explicit memory is a memory system that is controlled consciously, intentionally, and flexibly. Declarative memory generally involves some effort and intention, and we can employ memory strategies such as mnemonics to recall information [1]. It is mediated by the hippocampus and frontal lobes thus the damage for those areas is known as declarative memory [9]. The definition of declarative and non-declarative memory is declarative memory and nondeclarative memory are two major classifications of long-term memory systems. Declarative memory allows us to consciously recollect events and facts. It is generally indexed by our ability to explicitly recall or recognize those events or facts. Nondeclarative memory, in contrast, is accessed without consciousness or implicitly through performance rather than recollection [10]. The various memory are existed, short term memory and long term memory. Explicit memory is conscious memory, implicit memory is unconscious memory. Explicit memory consists of episodic memory and semantic memory as well as implicit memory consists of priming and procedural memory. The main types of memory is episodic memory, semantic memory, procedural memory & working memory. This all memory types includes the specific organ, duration of storage memory, type of awareness and various examples. The episodic memory located in the medial temporal lobes, anterior thalamic nucleus, mammillary body, fornix, prefrontal cortex type of awareness is explicit and declarative e.g. is remembering a short story, what you had for dinner last night, and what you did on your last birthday. Semantic memory located in inferolateral temporal lobes.
it is type of explicit and declarative memory knowing who was the first president of the united states, the color of a lion, and how a fork differs from a comb. Procedural memory located in basal ganglia, cerebellum, Supplementary motor area duration for the procedural memory is minute to Years type of awareness is explicit or declarative driving a car with a standard transmission (explicit) and learning the sequence of numbers on a touch-tone phone without trying (implicit). Working memory phonologic: prefrontal cortex, boca’s area, Wernicke’s area spatial: prefrontal cortex, visual-association areas and type of awareness is explicit or declarative duration of memory is seconds and minutes and examples is phonologic: keeping a phone number “in your head” before dialing spatial: mentally following a route or rotating an object in your mind [4, 5, 6, 7, 8].

**Memory related clinical disease and disorders**

Memory includes various disease and disorders, disorders of grammars and procedural memory includes the developmental disorders consists, specific language impairment (Sli), Tourette syndrome, adult-onset disorders, parkinson’s disease, huntington’s disease non-fluent aphasia, korsakoff syndrome, vascular disorders severe hypoxia, head injury.

**Specific language impairment**

Special language impairment is usually defined as a developmental disorder of language that occurs in the absence of frank neurological damage, hearing deficits, severe environmental deprivation, and mental retardation [15]. The disorders is impairment is of specific grammar or processing deficit.

**Parkinson’s disorders**

Parkinson’s is chronic, progressive and neurodegenerative disorders characterized by motor and nonmotor features this disease has clinical impact on families, patient’s, care givers progressive degenerative effects on mobility and muscle control [12, 13, 14]. Parkinson’s disease (PD) is a chronic progressive neurodegenerative disorder characterized by early prominent death of dopaminergic neurons in the substantia Nigra pars compacta and wide spread presence of alpha synuclein, an intracellular protein. Dopamine deficiency in the basal ganglia leads to classical parkinsonian motor symptoms viz, bradykinesia, tremor, rigidity and later postural instability. The pathological hallmark of PD is cell loss within the substantia Nigra particularly affecting the ventral component of the pars compacta. By the time of death, this region of the brain has lost 50-70% of its neurones compared with the same region in unaffected individuals. The earliest documented pathological changes in pd3 have been observed in the medulla oblongata/pontine tegmentum and olfactory bulb. In these early stages-break stages 1 and 2-patients are pre-symptomatic. As the disease advances-break stages 3 and 4- the substantia Nigra, areas of the midbrain and basal forebrain become involved. Finally, the pathological changes appear in the neocortex and it affects the memory [16-17].

**Tourette syndrome**

Tourette syndrome is a neurodevelopment disorder characterized by both multiple motor and one or more phonetic tics [18]. Onset of Tourette syndrome occurs during childhood, and tic frequency and severity is known to wax and wane [19]. Children and adolescents are the most affected by Tourette syndrome, with an estimated population prevalence of 0.3% to 0.9% [20]. Other tic disorders, such as persistent or transient tic disorder, have a higher prevalence, especially in childhood [21]. Tourette syndrome may also cause several other functional impairments that can affect quality of life; such as physical discomfort associated with tics, poor concentration, relationship problems (e.g., stigmatization, victimization), and academic, cognitive, and emotional problems that can result in explosive outbursts [22, 23, 24, 25, 26].

**Huntington’s disease**

Huntington’s disease is a progressive brain disorder caused by a single defective gene on chromosome 4-one of the 23 human chromosomes that carry a person’s entire genetic code. The hallmark symptom of Huntington’s disease is uncontrolled movement of the arms, legs, head, face and upper body. Huntington’s also causes a decline in thinking and reasoning skills, including memory, concentration, judgment and ability to plan and organize. In addition, Huntington’s disease brain changes lead to changes in mood, especially depression, anxiety and uncharacteristic anger and irritability. Obsessive-compulsive behavior is also common, causing a person to repeat the same question or activity over and over [27].

Huntington’s disease is an autosomal dominant neurodegenerative disorder clinically characterized by progressive involuntary movements, neuropsychiatric disturbances and cognitive impairment. At a molecular level, the disease is caused by the abnormal expansion of a cag trinucleotide repeat within the it15 gene located on chromosome [28].

**Korsakoff syndrome**

Korsakoff syndrome is a chronic memory disorder caused by severe deficiency of thiamine (vitamin b1). Thiamine helps brain cells produce energy from sugar. When levels fall too low, brain cells cannot generate enough energy to function properly. Korsakoff syndrome is most commonly caused by alcohol misuse but can also be associated with aids, cancers that have spread throughout the body, chronic infections, poor nutrition and certain other conditions. It is also common in people whose bodies do not absorb food properly (malabsorption). This can sometimes occur with a chronic illness or after weight-loss (bariatric) surgery korsakoff syndrome is often-but not always-preceded by an episode of wernicke encephalopathy, which is an acute brain reaction to severe lack of thiamine. Wernicke encephalopathy is a medical emergency that causes life-threatening brain disruption, profound confusion, staggering and stumbling, lack of coordination and abnormal involuntary eye movements. The chronic memory loss of korsakoff syndrome often follows an episode of wernicke encephalopathy, the chronic disorder is sometimes known as wernicke korsakoff syndrome. But korsakoff syndrome can also develop in individuals who have not had a clear-cut prior episode of wernicke encephalopathy. Symptoms of korsakoff syndrome causes problems learning new information, inability to remember recent events and long-term memory gaps. Memory difficulties may be strikingly severe while other thinking and social skills are relatively
unaffected e.g. individuals may seem able to carry on a coherent conversation but moments later are unable to recall that the conversation took place or with whom they spoke. Those with korsakoff syndrome may “confabulate,” or make up, information they can’t remember. They are not “lying” but may actually believe their invented explanations. Scientists don’t yet understand the mechanism by which korsakoff syndrome may cause confabulation. The person may also see or hear things that are not there (hallucinations).

**Vascular disease**
Vascular risk factors including diabetes [30-34], Insulin resistance [35], Hypertension [36]. Heart disease [37]. Smoking [38, 39]. And obesity [40]. Are each independently associated with an increased risk of cognitive impairment and dementia and are frequent in the elderly. The associations between vascular risk factors and dementia may depend on the stage of the life span in which they are examined. For example, high blood pressure and obesity in middle-age have been found as risk factors for dementia in old age [41, 42]. In old age, however, these associations seem to be attenuated or inverse for reasons that are uncertain [43-44]. Cerebrovascular disease is increasingly recognized as a cause of cognitive impairment and dementia in later life either alone or in conjunction with alzheimer disease (ad) or other pathologies. Cerebrovascular disorder describes a heterogeneous group of disorders with different types of cerebrovascular lesions contributing to cognitive decline and finally to the development of dementia. Dementia related to vascular disorders, first described as “arteriosclerotic dementia” [45], was later replaced by terms like “multi-infarct dementia”, post-stroke dementia [45].

**Head injury**
Dementia is a general term for a decline in mental ability severe enough to interfere with daily life. Dementia is not a single disease; it’s the umbrella term for an individual’s changes in memory, thinking or reasoning. There are many possible causes of dementia, including Alzheimer’s. Disorders grouped under the general term “dementia” are caused by abnormal brain changes. These changes trigger a decline in thinking skills, also known as cognitive abilities, severe enough to impair daily life and independent function. They also affect behavior, feelings and relationships. Brain changes that cause dementia may be temporary, but they are most often permanent and worsen over time, leading to increasing disability and a shortened life span. Survival can vary widely, depending on factors such as the cause of the dementia, age at diagnosis and coexisting health conditions. traumatic brain injury (TBI), cognitive change TBI’S direct effects—which may be long-lasting or even permanent—can include unconsciousness, inability to recall the traumatic event, confusion, difficulty learning and remembering new information, trouble speaking coherently, unsteadiness, lack of coordination, and problems with vision or hearing. Certain types of TBI may increase the risk of developing Alzheimer’s or another type of dementia years after the injury takes place [4].

**References**


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