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*Edited by*

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## CENTRAL NERVOUS SYSTEM DISORDERS

The central nervous system (CNS) is comprised of the brain and the spinal cord. The spinal cord controls movement and feeling of body regions located below the brain. Because the brain and the spinal cord are connected, the brain also plays a role in movement and feeling. However, the brain controls complex psychological processes such as attention, perception, motivation, emotion, language, cognition, and action. Central nervous system disorders may arise due to brain injury, or may occur as the result of congenital or degenerative neurological conditions.

When certain parts of the brain are damaged through accident or disease, specific functions may be lost. The type and extent of functional loss depends upon the location of the brain damage and the amount of brain tissue that is compromised. For example, damage to a strip of cortex in the posterior part of the frontal lobes controlling movement of parts of the body will result in paralysis of those body parts. Lesions within relay stations along the visual sensory system—from the optic nerves to the occipital lobes—will result in visual field defects such as scotomas (“blind spots”). Lesions deep in the hypothalamus may produce hunger, uncontrolled eating, and obesity. A destruction of areas involved in arousal may result in a permanent comatose state.

Damage to specific regions of the brain usually produces behavioral abnormalities that can be measured quantitatively and qualitatively by employing sensitive



tests of impaired or lost functions. For example, an analysis of specific sensory functions can reveal basic sensory defects. Cognitive and intellectual defects can be measured through the skillful administration and interpretation of tests specifically designed to elucidate organically based impairments. Descriptions of many of these tests may be found in books on neuropsychological assessments (e.g., Strauss, Sherman, & Spreen, 2006), as well as in a chapter by Oscar-Berman and Bardenhagen (1998).

Disorders of the CNS usually are classified according to lesion location (e.g., abnormalities occurring after frontal lobe damage) or according to symptomatology and functional loss (e.g., amnesia and aphasia). The following discussion focuses on specific exemplars of CNS disorders. The first, frontal system disorders, exemplifies some possible consequences of damage to the anterior regions of the frontal lobes. The others exemplify disorders recognized by their presenting symptoms and functional abnormalities: Amnesia refers to disorders of memory, including memory for recent events (anterograde amnesia) and memory for events long ago (retrograde amnesia); and aphasia refers to language disturbances. Keep in mind, however, that the distinction between structure and function is not meant to be a mutually exclusive one. The brain has many highly interconnected parts, and when one part is damaged, other parts will be affected as well.

### Frontal System Dysfunction

The frontal lobes are connected with all of the other lobes of the brain, and they receive and send fibers to numerous subcortical structures as well. While control of motor function takes place in the posterior region of the frontal lobes, the anterior region of the frontal lobes (prefrontal cortex) plays a kind of executive regulatory role within the CNS, inhibiting the occurrence of unnecessary or unwanted behaviors. Disruptions of normal inhibitory functions of frontal lobe neuronal networks often will have the interesting effect of releasing previously inhibited behaviors from frontal control. The resultant aberrant conduct of a frontal patient may be due to the freely unregulated functioning of the released brain region rather than a direct effect of a lesion within the frontal lobes, and as such may be referred to as a disinhibition syndrome (Starkstein & Kremer, 2001).

Early evidence for a role of the frontal lobes in supporting the ability to inhibit impulsivity came from the 1868 report of a physician on his patient Phineas Gage. Gage, a railway workman, survived an explosion that blasted an iron bar (about four feet long and an inch wide) through his frontal lobes. After recovering from the accident, Gage's personality changed. He became irascible, impatient, impulsive, unruly, and inappropriate. The damage had mostly been in the orbital frontal region of Gage's frontal lobes (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994).

Damage to frontal brain systems occurs in a number of CNS disorders, including stroke, brain tumors, dementing diseases (e.g., Alzheimer's), and head trauma. Patients with bilateral frontal disorders often display a pull to nearby objects (e.g., grabbing at doorknobs), as well as a remarkable tendency to imitate the actions of people nearby (echopraxia). The behaviors of frontal patients appear not to be based on rational decisions, but rather are under the control of salient objects around them, that is, objects that capture their attention. In other words, the patient's behaviors are environmentally driven rather than personally chosen. Environmental dependency and imitation behaviors can also be associated with "utilization behavior" (Archibald, Mateer, & Kerns, 2001). That is, if the examiner places a set of everyday objects in front of the patient with instructions neither to use them nor to pick them up, the patient nonetheless will do just that! If one of the objects were a comb, the patient would likely pick it up and begin combing his/her hair. Utilization behavior may even extend to dangerous objects such as hypodermic needles, with patients attempting to give themselves injections.

### Brain Mechanisms in Memory

Amnesia, especially anterograde amnesia, is an intriguing but serious disorder. When amnesia occurs as a consequence of long-term alcoholism, it is referred to as alcoholic Korsakoff's syndrome (also Alcohol-Induced Persisting Amnesic Disorder). Patients with Korsakoff's syndrome are permanently unable to remember new information for more than a few seconds. However, old memories, which were formed prior to the onset of alcohol-related brain damage, are relatively well preserved. Because new events are forgotten a few seconds after they occur, virtually nothing new is learned, and the patient with Korsakoff's syndrome lives in the past.

George Talland in his classic book, *Deranged Memory* (Talland, 1965) linked the etiology of Korsakoff's disease most frequently with the polyneuropathy of chronic alcoholism and associated malnutrition. The critical brain lesions are thought to include the mammillary bodies of the hypothalamus and/or medial thalamic nuclei. Damage to these or to other regions of the brain (hippocampus, fornix, anterior thalamus) identified with the classic interconnected circuit described by Papez (1937) has been associated with memory impairments. The impairments include severe anterograde amnesia for recent events, and some retrograde amnesia for events prior to the appearance of obvious symptomatology.

Although anterograde amnesia is the most obvious presenting symptom in Korsakoff patients, it has been suggested that, in addition to having severe memory problems, these individuals have other cognitive impairments as well. Like patients with bilateral prefrontal cortical lesions, Korsakoff patients are abnormally sensitive to



distractions (proactive interference). This sensitivity may be due to prefrontal dysfunction, which impairs the ability to counteract the effects of cognitive interruptions. Memory encoding requires the ability to resist displacement of the to-be-remembered information from ongoing memory processing. Similarly, memory retrieval requires the ability to screen out irrelevant contextual cues in order to focus on relevant cues and thus to select the target memory. In addition to their memory problems, perseverative behaviors, and distractibility, Korsakoff patients also have restricted attention, retarded perceptual processing abilities, and decreased sensitivity to reward contingencies (Oscar-Berman & Marinkovic, 2007). These additional abnormalities probably reflect widespread cerebral atrophy accompanying sustained alcohol abuse.

### Aphasia

The term aphasia literally means “no language.” More realistically, aphasic patients suffer from impairment in their previous level of ability to use language expressively or receptively, or both. For that reason, the term dysphasia (impairment in language) sometimes is used. There are many different forms of aphasia, and classification schemes can be found in Harold Goodglass’s book, *Understanding aphasia* (Goodglass, 1993). Pathology is almost always within the left hemisphere of right-handers, as well as in a majority of left-handers. Frequently the location of the brain damage is in the frontal lobe (Broca’s aphasia) or the temporal lobe (Wernicke’s aphasia), and usually the damage is the result of a cerebrovascular accident (stroke), tumor, or trauma.

There are components of aphasia that can be considered language-specific amnesias (e.g., the anomias). If a lesion is in the neighborhood of a cortical sensory projection zone, the resulting disorder may involve one or another of the sensory modalities (audition or vision). If a lesion is in a polysensory integration zone, it will cause a disorder of more highly elaborate functions (e.g., spatial recognition, language, and/or voluntary movement). It has been argued that aphasia is a mere loss of certain linguistic abilities, and that the “intellect” remains intact. Others, however, regard aphasia either as the manifestation of a primary intellectual loss or as the loss of a restricted aspect of intellect. As cautioned earlier, this problem can be oversimplified by regarding either aphasia or intellectual impairment as unitary deficits. Both are known to vary with locus and extent of lesion, and any overlap in symptoms may result from overlapping neuroanatomical representation rather than from the nature of the disorder.

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See also: Brain; Parasympathetic Nervous System;  
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