

Redwood Caregiver Resource Center  
1140 Sonoma Ave., Ste. 1B  
Santa Rosa, CA 95405  
(707) 542-0282 or (800) 834-1636  
Fax (707) 542-0552

Email: [rcrc@redwoodcrc.org](mailto:rcrc@redwoodcrc.org)

Web: <http://redwoodcrc.org>

Serving: Del Norte, Humboldt, Mendocino, Lake, Sonoma,  
Napa & Solano Counties



# Fact Sheet Hypoxic-Anoxic Brain Injury

## Introduction & Definition

Oxygen is required for normal brain functioning. Hypoxic-anoxic injuries result when there is a substantial (partial, or *hypoxic*) or a complete (total, or *anoxic*) lack of oxygen supplied to the brain. This diminished oxygen supply to the brain may produce profound cognitive (thinking), physical (movement), and affective (emotional) impairments which may be slow to recover. As a result, hypoxic-anoxic injury (HAI) can have a catastrophic impact, both in terms of functional (what a person can do) deficits as well as the costs involved in treatment and the disruption it can cause among associated families. One of the problems that has plagued affected individuals and their families is the relative lack of easy-to-read, accessible information to answer their questions. This fact sheet will help answer some of these questions.

## Causes of Hypoxic-anoxic Injury (Etiology)

Why is oxygen important? The brain consumes about 20% of the body's total oxygen. 90% of the brain's total energy is used to send electrochemical impulses and maintain the neurons' ability to send these impulses. Much like the base of a pyramid on which everything else rests, oxygen is necessary to metabolize glucose which is used to provide the energy for all living cells.

If oxygen is not available, a cascade-effect of problems occurs. Oxygen and glucose are re-

sponsible, either directly or indirectly, for a variety of chemical reactions which are involved in the production of important chemical-like brain neurotransmitters (e.g., dopamine, norepinephrine, and serotonin). Such neurotransmitters act to regulate the brain's many complex functions. One particular neurotransmitter, acetylcholine (Ach), seems to play a direct role in memory.

HAI can be caused by a variety of disease processes and injuries. Although there are several possible reasons for anoxic injury, hypoxic-ischemic injury (HII) is the most common.

- **Anoxic anoxia**—not enough oxygen is in the air to be absorbed by the body and used (high-altitude sickness). Unless the person has just climbed Mount Everest or is a jet pilot, this is an uncommon cause.
- **Anemic anoxia**—basically, not enough blood or hemoglobin, which is a chemical in red blood cells that carry oxygen throughout the body. Acute hemorrhage, chronic anemia, carbon monoxide poisoning are common causes of this type of injury. *Acute hemorrhages* (bleeding) can occur due to gunshot wounds. *Chronic anemia* occurs when there are persistent low red blood cells or hemoglobin. *Carbon monoxide* poisoning is seen in suicide attempts using the exhaust of automobiles, but can also occur in home or industrial accidents (e.g., malfunctioning furnace, cleaning oil tanks, working with machinery in poorly ventilated areas). Carbon monoxide poisoning also

appears to selectively damage particular areas of the brain (such as the basal ganglia, caudate nucleus, putamen, globus pallidus, and central white matter). These brain areas are important for the control of movement.

- **Stagnant (ischemic) anoxia (also called hypoxic-ischemic injury, or HII)**—not enough cerebral blood flow to carry blood to the brain. Injury can be localized (such as ischemic strokes) or generalized (circulatory collapse secondary to cardiac arrhythmias or cardiac arrest). This type of injury causes general, diffuse damage to the cerebral cortex and cerebellum. Areas of the brain that are very sensitive to lack of oxygen include the hippocampus (a region critical for memory), borderzone areas of the cerebral cortex (the parieto-occipital and frontoparietal regions), cerebellum, basal ganglia, and spinal cord (thoracic region).

Below is a summary of the more frequent causes of cardiac arrest, perhaps the most common cause of ischemic anoxia:

- Anesthesia accidents – 32%
- Atherosclerotic cardiovascular disease (“hardening of the arteries”) – 29%
- Asphyxia (drowning and suicide attempts) – 16%
- Chest trauma – about 10%
- Electrocutation – about 6.5%
- Severe bronchial asthma – 3%
- Barbiturate poisoning –3%

(Source: Groswasser, Ze'ev, Cohen, & Costeff, 1989).

## Symptoms

The symptoms of hypoxic-ischemic injury (HII) are related to the areas of the brain and spinal cord that are damaged. A brief summary of the types of symptoms and regions of the brain responsible are

described below. Symptoms will be divided into cognitive and physical deficits:

**Cognitive deficits.** The cognitive symptoms observed from most to least common are:

- *Short-term memory loss.* This is by far the most common, virtually universal, symptom of HII. The reason is that one portion of the brain believed to be critical for learning new information (called the hippocampus) has neurons which are highly sensitive to changes in chemistry or trauma.
- *Executive functioning* (reasoning/judgment, initiation, perseveration, and impulsivity).
- *Word-finding difficulties* (also called anomia).
- *Visual disturbances.* A person may have difficulty processing incoming visual information. In rare instances, a disorder called cortical blindness (Anton’s Syndrome) may be observed in HII. In this case, the area of the brain responsible for vision becomes disconnected from the rest of the brain. Because the brain cannot tell that this part of the brain is damaged or disconnected, a person may act as though he/she can see, even though he/she shows no ability to identify or discriminate objects, shapes, or colors.

**Physical deficits.** Commons symptoms may include:

- *Ataxia* (incoordination). These symptoms are similar to those seen in alcohol-intoxicated individuals. Areas of the brain that may be affected are the cerebellum, basal ganglia, and putamen.
- *Apraxia* (inability to follow a sequence of commands).
- *Spasticity, rigidity, myoclonus.* Abnormal movements.
- *Quadriparesis* (weakness of all four extremities), paresis (weakness) or paralysis (inability to move). This can also occur due to spinal cord damage.

## Predicting Outcome (Will the person recover?)

Why is the recovery of from HAI different from traumatic brain injuries or strokes? Recovery is thought to occur for a variety of reasons. One theory is that there exists a neuronal reserve which is used to compensate for lost nerve cells. Because an anoxic injury is diffuse and widespread, this may deplete the reserve capacity substantially.

Predicting outcome is much like estimating how high a rocket will go up. There are some general factors at the start of the launch which will be helpful, but the *actual* course of the rocket will also point out future progress. In other words, past and current changes will predict future recovery. Below is a list of these factors.

- **Coma duration**—as might be expected, the longer a person is in a coma, the less promising the outcome. One study suggested that if coma is less than 12 hours, there is no or little transient damage. If coma is greater than 12 hours, recovery may be slow and incomplete (lasting deficits). If coma is 24 hours or more, the person is at a greater chance of dying.

Another study showed that 21% of HAI patients who remained in coma four weeks or less had a good recovery, and 79% had a poor recovery. 100% of those patients who remained in coma more than four weeks had a poor recovery. Also, the patients whose coma durations were greater than four weeks tended to show minimal recovery (functional gains) after the first four months.

If a person does not “wake up” from a coma, then significant damage to the cerebral cortex, called laminar necrosis may have occurred. Like wood-paneling, the cerebral cortex is made up of a number of layers. If these cells die, the layers become separated from each other. This results in neocortical death while lower brain functions continue to operate (called a persistent vegetative state).

- **Pupillary reactivity**—when both eyes have “fixed”, or dilated pupils, this points to a very poor prognosis. Even survival is highly questionable as such signs point to brain-stem damage, which is a brain area responsible for keeping the body regulated (like breathing, heart-rate, etc.).
- **Oculovestibular reflexes**—one of the brain’s many reflexes that can be tested by a neurologist. If these are not present, it suggests lower brain-stem damage.
- **Age**—Some studies suggest that patients 25 years and younger have a better rate of recovery than those who are older.
- **Cause of injury**—there have been several studies which suggest that functional recovery is unrelated to the cause of the cardiopulmonary arrest.
- **Imaging studies (CT/MRI scans)**—These imaging techniques typically fail to find any recent (acute) damage. If an imaging study is done several months later, damage may include brain matter loss (atrophy) with resulting enlargement of the ventricles.
- **EEGs/Evoked Potentials (EPs)**—If EEGs show cortical activity, then it is a positive prognostic sign. EEGs/EPs may also be used to determine brain death through the lack of cortical electrical activity.

## Treatment and Course of Injury

Unfortunately, direct treatment of the anoxia is limited. Some studies have suggested the use of barbiturates, which slow down the brain’s activity, may be helpful in the first 2-3 days of injury onset. Otherwise, the general medical approach is one of maintaining the body’s general status. Once a person becomes medically stable, the next question is to what capacity can he/she recover/participate? Rehabilitation efforts may be attempted anywhere along the several stages of recovery:

- **Coma**—a condition in which the person appears to be sleeping, but is unable to be aroused

- **Persistent vegetative state (PVS)**—“wakeful unresponsiveness”; a person who is neither in a coma nor conscious.
- **Conscious**—a person is awake and able to interact with his/her environment.
- **Coma/PVS rehabilitation.** In the 1980s and early 1990s, coma or sensory stimulation was thought to help speed the person from coma or persistent vegetative state (PVS). However, there have been a number of studies which, to date, do not support these earlier claims.
- **Acute/sub-acute rehabilitation.** If the person is conscious, then active attempts will be made to assist the patient to improve cognitive and functional skills. Course of treatment may be very long.

During rehabilitation, the individual and family may interact with a variety of professionals as the need for constant medical attention (and hence for physician involvement) diminishes. Such professionals may include a *physical therapist* (who aids in improving walking/movement), an *occupational therapist* (who assists in retraining/relearning previously known daily living skills), a *speech therapist* (who helps with swallowing, speech or cognitive problems), and a *neuropsychologist* (who may assess the level and kind of cognitive impairment, collaborate on cognitive retraining and assist both the individual and family with behavioral and emotional issues).

As recovery may take months to years, rapport and a good working relationship with the rehabilitation specialists are very important. Because of extended contact, both the person in rehabilitation and his/her family may experience a variety of emotions as they learn to cope and adapt to a constantly changing condition. Because expectations may not always match the person’s current level of progress, the potential for disappointment and/or conflict may be high. Therefore, it is important to discuss such issues both early in the course of treatment as well as throughout the course of recovery.

Finally, predicting long-term recovery from hypoxic-anoxic brain injury can be difficult. Full rehabilitation potential is not always apparent

early on. Often, depending on the nature and extent of the injury, rehabilitation and improvement can continue over a period of months or years. Families should stay informed and involved. It is important that the person’s family work with the rehabilitation team to help ask and answer questions about the person, monitor care, and provide support. Working together is one way to help maximize the person’s potential and ensure that the person can continue to live in the least restrictive environment.

## Credits

Caronna, J. (1979). Diagnosis, prognosis, and treatment of hypoxic coma. *Advances in Neurology*, 26, 1-15.

Gibson, G.E., Pulsinelli, W., Blass, J.P., Duffy, T.E. (1981). Brain dysfunction in mild to moderate hypoxia. *American Journal of Medicine*, 70, 1247-1254.

Groswasser, Ze’ev, Cohen, M., & Costeff, H. (1989). Rehabilitation outcome after anoxic brain damage. *Archives of Physical Medicine & Rehabilitation*, 70, 186-188.

Myer, R.E. (1979). A unitary theory of causation of anoxic and hypoxic brain pathology. *Advances in Neurology*, 26, 195-213.

## Resources

### American Academy of Physical Medicine and Rehabilitation

IBM Plaza, Suite 2500  
Chicago, IL 60611-3604  
(312) 464-9700

### American Board of Clinical Neuropsychology

Dept. of Psychiatry  
c/o Dr. Linas Bieliauskas  
University of Michigan Medical Center  
1500 E. Medical Center Drive  
Ann Arbor, MI 48109-0704  
(313) 936-8269

### Brain Train

(computer software for cognitive retraining)  
727 Twinridge Lane  
Richmond, VA 23235  
(804) 320-0105

**National Rehabilitation Information Center**  
8455 Colesville Road, Suite 935  
Silver Spring, MD 20910-3319  
(800) 346-2742

*Prepared by Brent A. Hughey, Ph.D., Neuropsychologist, for use by Family Caregiver Alliance and California's Caregiver Resource Centers, a statewide system of resource centers serving families and caregivers of brain-impaired adults. Dr. Hughey is in private practice in Pleasanton, California. He is on staff at Golden Gate Rehabilitation Hospital in San Ramon and serves as consultant for several long-term care facilities in the San Francisco Bay Area. Fact sheet funded by California Department of Mental Health. Printed September 1996. ©All rights reserved.*