

# Cortical blindness

Cortical [blindness](#) is the total or partial [loss of vision](#) in a normal-appearing [eye](#) caused by damage to the brain's [occipital cortex](#).

The [occipital poles](#) are particularly vulnerable to diffuse [hypoxia](#) <sup>1)</sup>; attested to by cases of [cortical blindness](#) after [cardiac arrest](#) <sup>2)</sup>. [Hypotension](#) superimposed on compromised [PCA](#) circulation (from herniation or elevated [ICP](#)) may thus increase the risk of postgeniculate [blindness](#). <sup>3)</sup>, <sup>4)</sup>.

## Classification

Cortical blindness can be acquired or congenital, and may also be transient in certain instances.

Acquired cortical blindness is most often caused by loss of blood flow to the occipital cortex from either unilateral or bilateral posterior cerebral artery blockage (ischemic stroke) and by cardiac surgery.

In most cases, the complete loss of vision is not permanent and the patient may recover some of their vision (Cortical visual impairment).

## Etiology

There have been many causes of cortical blindness reported in the medical literature. These include hypoxia/ischaemia (stroke, cardiac arrest), infections (meningitis, encephalitis), trauma, haemorrhagic shock, metabolic disturbances (uraemia, hypoglycaemia), drugs/toxins (carbon monoxide poisoning) and post-procedural (vertebral angiography, cardiac surgery)

Rarely, a patient with acquired cortical blindness may have little or no insight that they have lost vision, a phenomenon known as Anton-Babinski syndrome.

Cortical blindness and cortical visual impairment (CVI), which refers to the partial loss of vision caused by cortical damage, are both classified as subsets of neurological visual impairment (NVI). NVI and its three subtypes—cortical blindness, cortical visual impairment, and delayed visual maturation—must be distinguished from ocular visual impairment in terms of their different causes and structural foci, the brain and the eye respectively. One diagnostic marker of this distinction is that the pupils of individuals with cortical blindness will respond to light whereas those of individuals with ocular visual impairment will not.

Trauma has been known to result in cortical blindness but the exact pathophysiology remains unknown and remains a matter of continued debate.

see [Transient posttraumatic cortical blindness](#)

<sup>1)</sup>

Hoyt WF, Walsh FB. Cortical Blindness with Partial Recovery Following Cerebral Anoxia from Cardiac Arrest. Arch Ophthalmol. 1958; 60:1061-1069

<sup>2)</sup>

Weinberger HA, van der Woude R, Maier HC. Prognosis of Cortical Blindness Following Cardiac Arrest in Children. JAMA. 1962; 179:126-129

<sup>3)</sup>

Arroyo HA, Jan JE, McCormick AQ, et al. Permanent Visual Loss After Shunt Malfunction. Neurology. 1985; 35:25-29

<sup>4)</sup>

Lindenberg R, Walsh FB. Vascular Compressions Involving Intracranial Visual Pathways. Tr Am Acad Ophth Otol. 1964; 68:677-694

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