

# Hypoxic coma

Anoxic [encephalopathy](#) may be due to anoxicemic anoxia (drop-in pO<sub>2</sub>) or anemic anoxia (following exsanguination or [cardiac arrest](#)). [Myoclonus](#) is common.

Vulnerable cells:

1. cerebral gray matter: lesions predominate in 3rd cortical layer (white matter is usually better preserved due to lower O<sub>2</sub> requirements)
2. Ammon's horn is also vulnerable, especially the Sommer section
3. in the basal ganglia (BG):
  - a) anoxicemic anoxia severely affects globus pallidus
  - b) anemic anoxia affects the caudate nucleus and putamen
4. in the cerebellum: Purkinje cells, dentate nuclei, and inferior olives are affected

A multivariate analysis yields outcome prognosticators:

This analysis applies only to hypoxic-ischemic coma; and is based retrospectively on 210 patients, most S/P cardiac arrest with many medical complications <sup>1)</sup>.

More recent studies confirm the poor prognosis of unreactive pupils and lack of motor response to pain <sup>2)</sup> if either of these findings is seen within a few hours after cardiac arrest there is an 80% risk of death or permanent vegetative state, and if present at 3 days, this rate rose to 100%.

Glucocorticoids (steroids) have been shown to have no beneficial effect on survival rate or neurological recovery rate after cardiac arrest <sup>3)</sup>.

<sup>1)</sup>

Levy DE, Caronna JJ, Singer BH, et al. Predicting Outcome from Hypoxic-Ischemic Coma. JAMA. 1985; 253:1420-1426

<sup>2)</sup>

Zandbergen EGJ, de Haan RJ, Stoutenbeek CP, et al. Systematic Review of Early Prediction of Poor Outcome in Anoxic-Ischemic Coma. Lancet. 1998; 352:1808-1812

<sup>3)</sup>

Jastremski M, Sutton-Tyrell K, Vaagenes P, et al. Glucocorticoid Treatment Does Not Improve Neurological Recovery Following Cardiac Arrest. JAMA. 1989; 262:3427-3430

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