Mechanisms of hyperbaric oxygen and neuroprotection in stroke.

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Cerebral vascular diseases, such as neonatal encephalopathy and focal or global cerebral ischemia, all result in reduction of blood flow to the affected regions, and cause hypoxia-ischemia, disorder of energy metabolism, activation of pathogenic cascades, and eventual cell death. Due to a narrow therapeutic window for neuroprotection, few effective therapies are available, and prognosis for patients with these neurological injuries remains poor. Hyperbaric oxygen (HBO) has been used as a primary or adjunctive therapy over the last 50 years with controversial results, both in experimental and clinical studies. In addition, the mechanisms of HBO on neuroprotection remain elusive. Early applications of HBO within a therapeutic window of 3-6h or delayed but repeated administration of HBO can either salvage injured neuronal tissues or promote neurobehavioral functional recovery. This review explores the discrepancies between experimental and clinical observations of HBO, focusing on its therapeutic window in brain injuries, and discusses the potential mechanisms of HBO neuroprotection.

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