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Pathophysiology and treatment of carbon monoxide poisoning

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Abstract

Carbon monoxide poisoning is the leading cause of poisoning deaths in the US, and published reports of carbon monoxide related morbidity and mortality can vary widely. Common morbidity involves myocardial and/or neurologic injury including delayed neurologic sequelae. The pathophysiology of this entity is complex, involving hypoxic stress on the basis of interference with oxygen transport to the cells and possibly impairing electron transport. Carbon monoxide can also affect leukocytes, platelets and the endothelium, inducing a cascade of effects resulting in oxidative injury. Carboxyhemoglobin levels are valuable for confirming carbon monoxide exposure but cannot be used to stratify severity of poisoning, predict prognosis, or indicate a specific treatment plan. Oxygen therapy is the key treatment of carbon monoxide intoxication, and hyperbaric oxygen has been shown to interdict and improve clinical outcome in some patients. Immediate treatment with a high fraction of inspired oxygen and careful clinical evaluation are mandatory. Timely referral for hyperbaric oxygen is indicated for patients with any history of unconsciousness, cardiovascular instability or ischemia, and persistent mental and/or neurologic deficits. Hyperbaric oxygen should also be considered in certain other patient subsets.

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