

Effects of Hyperbaric Oxygen on Inflammatory Response to Wound and Trauma: Possible Mechanism of Action

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There is growing interest in expanding the clinical applications for HBO₂ (hyperbaric oxygen therapy) into new medical and surgical fields. The pathophysiology of response towards wounds, infection, trauma, or surgery involves various chemical mediators that include cytokines, prostaglandins (PGs), and nitric oxide (NO). The beneficial role played by HBO₂ in wound healing, carbon monoxide poisoning, decompression sickness, and other indications is well documented. However, the exact mechanism of action is still poorly understood. This review addresses the effects of HBO₂ on PGs, NO, and cytokines involved in wound pathophysiology and inflammation in particular. The results of this review indicate that HBO₂ has important effects on the biology of cytokines and other mediators of inflammation. HBO₂ causes cytokine down-regulation and growth factor upregulation. HBO₂ transiently suppresses stimulus-induced proinflammatory cytokine production and affects the liberation of TNF α (tumor necrosis factor alpha) and endothelins. VEGF (vascular endothelial growth factor) levels are significantly increased with HBO₂, whereas the value of PGE₂ and COX-2 mRNA are markedly reduced. The effect of HBO₂ on NO production is not well established and more studies are required. In conclusion, cytokines, PGs, and NO may play a major role in the mechanism of action of HBO₂ and further research could pave the way for new clinical applications for HBO₂ to be established. It could be proposed that chronic wounds persist due to an uncontrolled pathological inflammatory response in the wound bed and that HBO₂ enhances wound healing by damping pathological inflammation (anti-inflammatory effects); this hypothetical proposal remains to be substantiated with experimental results.