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Hyperbaric oxygen therapy and mediators of inflammatory and immunological response: A concept for mechanisms of action

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The pathophysiology of response following trauma involves various chemical mediators, which include cytokines, prostaglandins and nitric oxide (NO). There is increasing interest in the beneficial role of HBO₂ in wound healing. However, the exact mechanism of action is still poorly understood and this may limit its clinical application. Results of this review of literature indicate that HBO₂ has important effects on the biology of cytokines and mediators of inflammation. Studies have showed that HBO₂ exposure transiently suppressed stimulus-induced proinflammatory cytokine production. In addition, HBO₂ attenuated the cytokine induction after hemorrhage. HBO₂ caused a marked decrease in IL-1 production. HBO₂ suppressed lipopolysaccharide-, lipid A- and PHA-induced tumor necrosis factor- α . Following HBO₂ exposure, stimulated lymphocytes released 51% less interferon- γ than cells obtained before the exposure. HBO₂ in healthy volunteers induced liberation of tumor necrosis factor α and endothelins. On the contrary, HBO₂ could attenuate the increase in the tumor necrosis factor- α and this reduction might be attributed to its beneficial effects. Vascular endothelial growth factor levels significantly increased with HBO₂, which seems to explain in part its angiogenic action. Concerning prostaglandins, it was shown that the value of prostaglandin E₂ in splenic macrophages, alveolar bone and gingiva reduced markedly after HBO₂ exposure. Moreover, HBO₂ was accompanied by significant reduction of prostaglandin E₂ generation in experimental colitis. In duodenal ulcer, HBO₂ raised prostaglandin E levels close to normal values. This might encourage the use of HBO₂ in patients with duodenal or gastric ulcer. In addition, HBO₂ decreased COX-2 mRNA. Therefore, these effects might stimulate the use of HBO₂ in acute or chronic inflammatory diseases as well as renal diseases in which large amounts of prostaglandin are produced. The effect of HBO₂ on NO production is not clear. Some studies showed that HBO₂ increased NO production whereas other studies showed the opposite effect. These discrepancies might result from different species of animals used in the studies and/or various combinations of pressure and oxygen concentrations employed. Therefore, more studies are required to demonstrate the actual effect of HBO₂ on NO production. Apparently, results from previous studies indicate that further extensive investigations are required, which could pave the way for new clinical applications of HBO₂.