Atherosclerosis

Hyperbaric oxygen reduces the progression and accelerates the regression of atherosclerosis in rabbits.

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We studied the effect of hyperbaric oxygen (HBO) treatment on the extent of diet-induced accumulation of lipid oxidation products in rabbit plasma and tissues, on plasma paraoxonase activity, and on the extent of progression and regression of atherosclerotic lesions in the rabbit aorta. HBO treatment of cholesterol-fed rabbits dramatically reduces the development of arterial lesions despite having little or no effect on plasma or individual lipoprotein cholesterol concentrations. Compared with no treatment in cholesterol-fed animals, HBO treatment also substantially reduces the accumulation of lipid oxidation products (conjugated dienes, trienes, and thiobarbituric acid-reactive substances) in plasma, in the low density lipoprotein and high density lipoprotein fractions of plasma, in the liver, and in the aortic tissues. In addition, HBO treatment prevents the decrease in plasma paraoxonase activity observed in rabbits fed cholesterol-rich diets. Similarly, in regression studies, HBO treatment has no effect on the rate of plasma (or lipoprotein) cholesterol decline but significantly accelerates aortic lesion regression compared with no treatment. Direct measures of aortic cholesterol content support these morphological observations. On the basis of these results, we conclude that repeated, but relatively short, exposure to HBO induces an antioxidant defense mechanism(s) that is responsible for retarding the development or accelerating the regression of atherosclerotic lesions.

Non-ischemic hypoxia of the arterial wall is a primary cause of atherosclerosis.

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The response-to-injury hypothesis has been the dominant model of atherosclerosis for 20 years. However, it does not explain the experimental role of oxygen in atherogenesis, does not explain many of the clinical features of atherosclerosis, and has failed to provide useful countermeasures. I propose that arterial wall hypoxia results from risk factors for atherosclerosis. The primary mechanism is decreased oxygen delivery by a microcirculatory derangement resulting from impaired erythrocyte deformability. As in a healing wound, hypoxia causes growth factor release within the arterial media. Diffusion of these factors causes intimal proliferation and atheroma formation. This hypothesis implies that simple inexpensive oxygenation regimens might prevent the morbidity and mortality of atherosclerosis. Despite demonstrated effectiveness in experimental models, such treatments have not been extensively studied in clinical atherosclerosis because they conflict with the dominant model. This dogma needs to be re-examined.

Use of hyperbaric oxygenation in the treatment of acute cerebrovascular disorders in ischemic heart disease

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Altogether 22 patients with concomitant cardio-cerebral acute pathology underwent all-round examinations during and after sessions of hyperbaric oxygenation (HBO). The highest clinical effect was produced by minor, substituting doses of HBO. The revealed phasic nature of the action of HBO in the treatment of patients with acute large-focal myocardial infarction and acute cerebrovascular disorders was found to be due to changes in the function of diencephalic and brain stem formations, determining the decrease of adaptation potentialities in this patients' group. HBO was found to be clinically effective in the treatment of such patients. In order to raise the treatment efficacy, it is necessary that an individual approach be exercised in the choice of therapeutic HBO sessions.