

CARDIOLOGY LITERATURE

Tohoku J Exp Med. 2008 May;215(1):113-7.

Hyperbaric oxygen therapy decreases QT dispersion in diabetic patients.

Kardesoglu E, Aparci M, Uzun G, Suleymanoglu S, Uz O, Onem Y, Ay H, Kucukardali Y, Ozkan S.

Diabetes mellitus is frequently associated with the malignant ventricular arrhythmias and sudden death. The QT dispersion is the difference between the longest and shortest QT interval calculated from the standard 12-lead electrocardiogram. The QT dispersion is suggested as an index of myocardial electrical activity. An increase in QT dispersion is associated with the malignant ventricular arrhythmias and sudden cardiac death. Diabetic patients receive hyperbaric oxygen (HBO) therapy for non-healing lower extremity ulcers. The aim of this study was to determine the effect of HBO therapy on QT dispersion in diabetic patients. Thirty diabetic patients (18 male and 12 female, 59.9 +/- 10 years), who were planning to undergo ten sessions of HBO therapy in two weeks for non-healing lower extremity ulcers, were consecutively enrolled into the study. The 12-lead resting electrocardiography recordings were taken before the first HBO therapy and after the 10th HBO-therapy session. QT intervals were measured on electrocardiogram. QT intervals were corrected for heart rate by using Bazett's formula (corrected QT [QTc] = QT/ radical R - R [seconds]). QTc dispersion was significantly decreased from 59.8 +/- 17.4 msec to 52.2 +/- 15.5 msec after ten sessions of HBO therapy (p < 0.05). However, maximum QTc, minimum QTc and mean QTc did not change significantly after HBO therapy. **We have concluded that HBO therapy may reduce the risk of malignant ventricular arrhythmia and sudden cardiac death in diabetic patients when applied repetitively.**

Kardiologija. 2007;47(12):53-6.

Long-term results of the use of hyperbaric oxygenation in patients with acute myocardial infarction

Dotsenko EA, Salivonchik DP, Kozyro VI.

Effect of hyperbaric oxygenation (HBO) on mortality and rate of development of reinfarctions during 24 month follow-up was studied in 129 otherwise conventionally treated patients with acute myocardial infarction (AMI). These patients were randomly divided into control (n=65) and intervention (n=64) groups. In the latter group treatment was supplemented with course of HBO. This course consisted of 6 HBO sessions in a single-place chamber (isopression for 40 min at working pressure 0.03 MPa o.d.) starting from day 4 - 6 of the disease. **The use of HBO in combination with traditional course of drug treatment significantly reduced rate of reinfarctions (control group - 19%, intervention group - 5.3%, p < 0.05) and increased survival (control group 86.2%, intervention group 94.7%) during 2 years after hospital discharge. Maximal effect on survival was seen during first 0.5 years (91.4 and 100% in control and intervention groups, respectively, p=0.05).**

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Adv Ther. 2007 Jan-Feb;24(1):106-18.

Can hyperbaric oxygen be used as adjunctive heart failure therapy through the induction of endogenous heat shock proteins?

Yogarathnam JZ, Laden G, Guvendik L, Cowen M, Cale A, Griffin S.

Heart failure (HF) is a chronic condition that is expected to increase in incidence along with increased life expectancy and an aging population. As the incidence of HF increases, the cost to national healthcare budgets is expected to run into the billions. The costs of lost productivity and increased social reliance on state support must also be considered. Recently, acute myocardial infarction (AMI) has come to be seen as the major contributing factor to HF. Although thrombolysis may restore coronary perfusion after an AMI, it may also introduce ischemic reperfusion injury (IRI). In an attempt to ameliorate sustained protein damage caused by IRI, endogenous chaperone proteins known as heat shock proteins (HSPs) are induced as a consequence of the stress of IRI. Recently, hyperbaric oxygen has been shown to induce the production of HSPs in noncardiac tissue, with a resultant protective effect. This current opinion review article suggests a possible role for hyperbaric oxygen, as a technologically modern drug, in augmenting the induction of endogenous HSPs to repair and improve the function of failing hearts that have been damaged by AMI and IRI. In addition, this simple, safe, noninvasive drug may prove useful in easing the economic burden of HF on already overextended health resources.

Cardiovasc Res. 2006 Oct 1;72(1):143-51. Epub 2006 Jul 21.

Hyperoxic and hyperbaric-induced cardioprotection: role of nitric oxide synthase 3.

Cabigas BP, Su J, Hutchins W, Shi Y, Schaefer RB, Recinos RF, Nilakantan V, Kindwall E, Niezgodka JA, Baker JE.

OBJECTIVE: The relative contributions of the fraction of inspired oxygen (FIO₂) and atmospheric pressure (ATM) to cardioprotection are unknown. We determined whether the product of FIO₂ x ATM (oxygen partial pressure) controls the extent of hyperoxic+hyperbaric-induced cardioprotection and involves activation of nitric oxide synthase (NOS). **METHODS:** Adult Sprague Dawley rats (n = 10/gp) were treated for 1 h with (1) normoxia+normobaria (21% O₂ at 1 ATM), (2) hyperoxia+normobaria (100% O₂ at 1 ATM), (3) normoxia+hyperbaria (21% O₂ at 2 ATM) and (4) hyperoxia+hyperbaria (100% O₂ at 2 ATM). **RESULTS:** Infarct size following 25 min ischemia and 180 min reperfusion was decreased following hyperoxia+normobaria and normoxia+hyperbaria compared with normoxia+normobaria and further decreased following hyperoxia+hyperbaria treatment. L-NAME (200 microM) reversed the cardioprotective effects of hyperoxia+hyperbaria. Nitrite plus nitrate content was increased 2.2-fold in rats treated with normoxia+hyperbaria and hyperoxia+hyperbaria. NOS3 protein increased 1.2-fold and association of hsp90 with NOS3 four-fold in hyperoxic+hyperbaric rats. **CONCLUSIONS:** Cardioprotection conferred by hyperoxia+hyperbaria is directly dependent on oxygen availability and mediated by NOS.

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J Thorac Cardiovasc Surg. 2005 Dec;130(6):1623-30. Epub 2005 Oct 26.

Pretreatment with hyperbaric oxygen and its effect on neuropsychometric dysfunction and systemic inflammatory response after cardiopulmonary bypass: a prospective randomized double-blind trial.

Alex J, Laden G, Cale AR, Bennett S, Flowers K, Madden L, Gardiner E, McCollum PT, Griffin SC.

OBJECTIVE: Animal studies have shown that pretreatment with hyperbaric oxygen can induce central nervous system ischemic tolerance and also modulate the inflammatory response. We evaluated this therapy in patients undergoing cardiopulmonary bypass. **METHODS:** Sixty-four patients were prospectively randomized to group A (n = 31; atmospheric air, 1.5 atmospheres absolute) or group B (n = 33; hyperbaric oxygen, 2.4 atmospheres absolute) before on-pump coronary artery bypass grafting. Age, sex, body mass index, diabetes, hypertension, smoking, coronary disease severity, left ventricular function, Parsonnet score, Euroscore, bypass time, myocardial ischemia time, and number of grafts were comparable in both groups. Canadian Cardiovascular Society angina, New York Heart Association dyspnea, and previous myocardial infarction were significantly higher in group B. Inflammatory markers were analyzed before surgery and 2 and 24 hours after bypass. Neuropsychometric testing was performed 48 hours before surgery and 4 months after surgery and included trail making A and B, the Rey auditory verbal learning test, grooved peg board, information processing table A, and digit span forward and backward. Neuropsychometric dysfunction was defined as more than 1 SD deterioration in more than 2 neuropsychometric tests. Chi-square tests, Fisher tests, t tests, and analysis of variance were used as appropriate for statistical analysis. **RESULTS:** Group A had a significant postoperative increase in the inflammatory markers soluble E-selectin, CD18, and heat shock protein 70. This was not observed in group B. Neuropsychometric dysfunction was also significantly higher in group A compared with group B. There was no difference in any other early postoperative clinical outcome. **CONCLUSIONS:** Our results seem to indicate that pretreatment with hyperbaric oxygen can reduce neuropsychometric dysfunction and also modulate the inflammatory response after cardiopulmonary bypass. However, further multicenter randomized trials are needed to clinically evaluate this form of therapy.

Am J Cardiol. 2004 Jun 15;93(12):1533-5.

Usefulness of hyperbaric oxygen therapy to inhibit restenosis after percutaneous coronary intervention for acute myocardial infarction or unstable angina pectoris.

Sharifi M, Fares W, Abdel-Karim I, Koch JM, Sopko J, Adler D; Hyperbaric Oxygen Therapy in Percutaneous Coronary Interventions Investigators.

The purpose of this trial was to assess whether the addition of hyperbaric oxygen to percutaneous coronary intervention can reduce clinical restenosis. Major adverse cardiac events at 8 months were found in only 1 of 24 patients (4%) who received hyperbaric oxygen compared with 13 of 37 patients (35%) who did not.