



TRAUMATIC BRAIN INJURY LITERATURE

Acta Neurochir Suppl. 2008;101:145-9.

Effect of hyperbaric oxygen on patients with traumatic brain injury.

Lin JW, Tsai JT, Lee LM, Lin CM, Hung CC, Hung KS, Chen WY, Wei L, Ko CP, Su YK, Chiu
Hyperbaric oxygen therapy (HBOT) is the medical therapeutic use of oxygen at a higher atmospheric pressure. The United States Food and Drug Administration have approved several clinical applications for HBOT, but HBOT in traumatic brain injury (TBI) patients has still remained in controversial. The purpose of our study is to evaluate the benefit of HBOT on the prognosis of subacute TBI patients. We prospectively enrolled 44 patients with TBI from November 1, 2004 to October 31, 2005. The study group randomly included 22 patients who received HBOT after the patients' condition stabilization, and the other 22 corresponding condition patients were assigned into the matched control group who were not treated with HBOT. The clinical conditions of the patients were evaluated with the Glasgow Coma Scale (GCS) and Glasgow Outcome Scale (GOS) before and 3 to 6 months after HBOT. The GCS of the HBOT group was improved from 11.1 to 13.5 in average, and from 10.4 to 11.5 ($p < 0.05$) for control group. Among those patients with GOS = 4 before the HBOT, significant GOS improvement was observed in the HBOT group 6 months after HBOT. **Based on this study, HBOT can provide some benefits for the subacute TBI patients with minimal adverse side effects.**

Brain Res. 2008 Jul 24;1221:126-33. Epub 2008 May 11.

Neuroprotective effect of hyperbaric oxygen therapy in brain injury is mediated by preservation of mitochondrial membrane properties.

Palzur E, Zaaroor M, Vlodaysky E, Milman F, Soustiel JF.

Recent experimental data have shown that hyperbaric oxygen therapy (HBOT) was associated increased Bcl-2 expression at the injury site that correlated with reduced apoptosis. We hypothesized that HBOT mediated enhancement of Bcl-2 expression and increased intracellular oxygen bio-availability may both contribute to preserve mitochondrial integrity and reduce the activation of the mitochondrial pathway of apoptosis. For this purpose, a cortical lesion was created in the parietal cortex of Sprague-Dawley rats by dynamic cortical deformation (DCD) and outcome measures in non-treated animals were compared with that of HBOT treated rats. Morphological analysis showed a profound reduction in neuronal counts in the perilesional area and a marked rarefaction of the density of the axonal-dendritic network. In treated animals, however, there was a significant attenuation of the impact of DCD over perilesional neurons, characterized by significantly higher cell counts and denser axonal network. In mitochondria isolated from injured brain tissue, there was a profound loss of mitochondrial transmembrane potential (Deltapsi(M)) that proved to be substantially reversed by HBOT. This finding correlated with a significant reduction of caspases 3 and 9 activation in HBOT treated animals but not of caspase 8, indicating a selective effect over the intrinsic pathway of apoptosis. **All together, our results indicate that the neuroprotective effect of HBOT may represent the consequence of preserved mitochondrial integrity and subsequent inhibition of the mPTP and reduction of the mitochondrial pathway of apoptosis.**



TRAUMATIC BRAIN INJURY LITERATURE

Neurol Res. 2007 Mar;29(2):162-72.

Hyperbaric oxygen in traumatic brain injury.

Rockswold SB, Rockswold GL, Defillo A.

OBJECTIVES: This critical literature review examines historical and current investigations on the efficacy and mechanisms of hyperbaric oxygen (HBO) treatment in traumatic brain injury (TBI). Potential safety risks and oxygen toxicity, as well as HBO's future potential, are also discussed. METHODS: Directed literature review. RESULTS: Historically, cerebral vasoconstriction and increased oxygen availability were seen as the primary mechanisms of HBO in TBI. HBO now appears to be improving cerebral aerobic metabolism at a cellular level, namely, by enhancing damaged mitochondrial recovery. HBO given at the ideal treatment paradigm, 1.5 ATA for 60 minutes, does not appear to produce oxygen toxicity and is relatively safe. DISCUSSION: The use of HBO in TBI remains controversial. Growing evidence, however, shows that HBO may be a potential treatment for patients with severe brain injury. Further investigations, including a multicenter prospective randomized clinical trial, will be required to definitively define the role of HBO in severe TBI.

Chin Med J (Engl). 2006 Dec 5;119(23):1978-82.

Evaluation of hyperbaric oxygen treatment of neuropsychiatric disorders following traumatic brain injury.

Shi XY, Tang ZQ, Sun D, He XJ.

BACKGROUND: Improvement of clinical symptoms following hyperbaric oxygen (HBO) treatment of neuropsychiatric disorders arising from traumatic brain injury was proved by our previous study. This study was aim to obtain the evidence of other changes. METHODS: Three hundred and ten patients with neuropsychiatric disorders arising from traumatic brain injury were treated twice with hyperbaric oxygen. Cerebral single photon emissions computed tomography (SPECT) images and computed tomography scans (CT) before and after hyperbaric oxygen treatment, were compared. RESULTS: Before treatment, the proportion of abnormal cerebral changes detected by SPECT was 81.3% but only 15.2% by CT. After HBO treatment, 70.3% of SPECT scans showed no abnormalities and these patients were clinically improved. Treatment improved regional cerebral blood flow. CONCLUSION: SPECT was much more sensitive than CT in the diagnosis of neuropsychiatric disorders following hyperbaric oxygen treatment of neuropsychiatric disorders arising from traumatic brain injury.



TRAUMATIC BRAIN INJURY LITERATURE

Neuropathol Appl Neurobiol. 2006 Feb;32(1):40-50.

Hyperbaric oxygen therapy reduces neuroinflammation and expression of matrix metalloproteinase-9 in the rat model of traumatic brain injury.

Vlodavsky E, Palzur E, Soustiel JF.

The acute inflammatory response plays an important role in secondary brain damage after traumatic brain injury (TBI). Neutrophils provide the main source of matrix metalloproteinases (MMPs) which also play a deleterious role in TBI. Numerous preclinical studies have suggested that hyperbaric oxygen therapy (HBOT) may be beneficial in various noncerebral and cerebral inflammatory diseases. The goal of this study was to evaluate the effects of HBOT on inflammatory infiltration and the expression of MMPs in correlation with secondary cell death in the rat model of dynamic cortical deformation (DCD). Twenty animals underwent DCD with subsequent HBOT (2.8 ATA, two sessions of 45 min each); 10 animals: DCD and normobaric oxygenation (1 ATA), 10 animals: not treated after DCD. Cell death was evaluated by TUNEL. Neutrophils were revealed by myeloperoxidase staining. Immunohistochemical staining for MMP-2 and -9 and tissue inhibitors of MMP-1 (TIMP-1) and -2 was also performed and the results were quantitatively evaluated by image analysis. In the animals treated by HBOT, a significant decrease in the number of TUNEL-positive cells and neutrophilic inflammatory infiltration was seen in comparison with nontreated animals and those treated by normobaric oxygen. The expression of MMP-9 was also significantly lower in the treated group. Staining for MMP-2 and TIMP-2 did not change significantly.

Our results demonstrate that HBOT decreased the extent of secondary cell death and reactive neuroinflammation in the TBI model. The decline of MMP-9 expression after HBOT may also contribute to protection of brain tissue in the perilesional area. Further research should be centred on the evaluation of long-term functional and morphological results of HBOT.



TRAUMATIC BRAIN INJURY LITERATURE

J Neurosurg. 2001 Mar;94(3):403-11

Effects of hyperbaric oxygenation therapy on cerebral metabolism and intracranial pressure in severely brain injured patients.

Rockswold SB, Rockswold GL, Vargo JM, Erickson CA, Sutton RL, Bergman TA, Biros MH.

OBJECT: Hyperbaric oxygenation (HBO) therapy has been shown to reduce mortality by 50% in a prospective randomized trial of severely brain injured patients conducted at the authors' institution. The purpose of the present study was to determine the effects of HBO on cerebral blood flow (CBF), cerebral metabolism, and intracranial pressure (ICP), and to determine the optimal HBO treatment paradigm. **METHODS:** Oxygen (100% O₂, 1.5 atm absolute) was delivered to 37 patients in a hyperbaric chamber for 60 minutes every 24 hours (maximum of seven treatments/patient). Cerebral blood flow, arteriovenous oxygen difference (AVDO₂), cerebral metabolic rate of oxygen (CMRO₂), ventricular cerebrospinal fluid (CSF) lactate, and ICP values were obtained 1 hour before and 1 hour and 6 hours after a session in an HBO chamber. Patients were assigned to one of three categories according to whether they had reduced, normal, or raised CBF before HBO. In patients in whom CBF levels were reduced before HBO sessions, both CBF and CMRO₂ levels were raised 1 hour and 6 hours after HBO ($p < 0.05$). In patients in whom CBF levels were normal before HBO sessions, both CBF and CMRO₂ levels were increased at 1 hour ($p < 0.05$), but were decreased by 6 hours after HBO. Cerebral blood flow was reduced 1 hour and 6 hours after HBO ($p < 0.05$), but CMRO₂ was unchanged in patients who had exhibited a raised CBF before an HBO session. In all patients AVDO₂ remained constant both before and after HBO. Levels of CSF lactate were consistently decreased 1 hour and 6 hours after HBO, regardless of the patient's CBF category before undergoing HBO ($p < 0.05$). Intracranial pressure values higher than 15 mm Hg before HBO were decreased 1 hour and 6 hours after HBO ($p < 0.05$). The effects of each HBO treatment did not last until the next session in the hyperbaric chamber. **CONCLUSIONS:** The increased CMRO₂ and decreased CSF lactate levels after treatment indicate that HBO may improve aerobic metabolism in severely brain injured patients. This is the first study to demonstrate a prolonged effect of HBO treatment on CBF and cerebral metabolism. On the basis of their data the authors assert that shorter, more frequent exposure to HBO may optimize treatment.