

**SOCIETA' ITALIANA
DI
MEDICINA SUBACQUEA E IPERBARICA**



**RICERCA IN MEDLINE DEI LAVORI DI
OSSIGENO TERAPIA IPERBARICA
INDICIZZATI CON PAROLA CHIAVE DI IMPORTANZA RILEVANTE**

**2004
SECONDO SEMESTRE**

a cura del
Dott. Francesco Ruocco
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Anestesia e Rianimazione del Dipartimento di Emergenza
della Azienda Ospedaliera Universitaria di Careggi

Search "**Hyperbaric Oxygenation**"[MAJR] Limits: **Publication Date from 2004/07 to 2004/12**

Search "Hyperbaric Oxygenation"[MAJR] Field: All Fields, Limits: Publication Date from 2004/07 to 2004/12

1: J Med Assoc Thai. 2004 Sep;87 Suppl 2:S218-22. Hyperbaric oxygenation and blood lactate clearance: study in sixty male naval cadets.

Sueblinvong T, Egtasaeng N, Sanguangrangsirikul S. Department of Biochemistry, Faculty of Medicine, Chulalongkorn University, Bangkok 10330, Thailand.

The research was aimed to study the effects of a 30 minute exposure to 2.5 ATA with 100% O₂-inhalation on lactate concentration after muscular fatigue from incremental exercise on a cycle ergometer. The subjects were 60 male naval cadets aged 20-23 years whose physical fitness was equivalent to the average athletes. All volunteers participated in the first VO₂ max exercise test to obtain their baseline data and randomly assigned into 3 groups of 20. The 3 groups were: Rest recovery group (RR), Oxygen recovery group (OR), and the Hyperbaric oxygenation (HBO₂) recovery group (HR). The volunteers took the incremental exercise test (Ordinary Lamp Protocol) on a cycle ergometer to exhaustion, then rested according to the above assigned groups. Blood samples were taken from each volunteer before the experiment, at exhaustion and every 5-minute intervals after the exhaustion for 30 minutes and immediately assayed for lactate concentration. The results showed significant decrease of blood lactate concentration at 15, 20 and 25 minutes intervals after the exhaustion in the HR group compared to the others. It might be initially concluded that HBO₂ enhanced the rate of lactate removal from peripheral blood vessels, therefore it shortened the recovery time.

PMID: 16083191 [PubMed - indexed for MEDLINE]

2: Arch Toxicol. 2005 May;79(5):289-93. Epub 2004 Nov 9.

Investigation of the role of hyperbaric oxygen therapy in cisplatin-induced nephrotoxicity in rats.

Atasoyu EM, Yildiz S, Bilgi O, Cermik H, Evrenkaya R, Aktas S, Gultepe M, Kandemir EG. Department of Nephrology, Gulhane Military Medical Academy, Haydarpasa Training Hospital, Kadikoy/Istanbul, Turkey.

Cisplatin (CP) is an effective chemotherapeutic agent used in the treatment of a variety of solid tumours. The most frequently observed side-effect of the use of CP is nephrotoxicity. Recently, evidence has been demonstrated that reactive oxygen species forming in the tubular epithelium play an important role in CP-linked nephrotoxicity. The aim of the study was to observe the effect of hyperbaric oxygen (HBO)

therapy on CP nephrotoxicity, a subject which has not been studied previously. Wistar rats were treated with CP (a single intraperitoneal (IP) dose of 0.6 mg/100 g) alone and in combination with HBO (60 min every day for seven days at 2.5 x atmospheric pressure). Effects of the treatment on renal function and histology were determined. In analyses at the end of the study it was observed that serum urea, creatinine, and daily urinary protein excretion levels of the CP group were higher than at the start of the study, and that the creatinine clearance level had fallen ($P < 0.05$). There was no significant difference between the CP+HBO group and HBO group serum urea, creatinine, creatinine clearance, and daily urinary protein excretion levels at the beginning and end of the study ($P > 0.05$). Histopathological examination showed that the necrosis score in the proximal tubule epithelial cells and average apoptotic cell numbers in the CP group were higher than those in the CP+HBO and HBO groups ($P < 0.05$). There was no statistical difference between the CP+HBO group and the HBO group in terms of necrosis score in the proximal tubule epithelial cells and the percentage of distal tubules containing hyaline casts in the lumen. In conclusion, in this study it was observed that in experimental study of CP nephrotoxicity the synchronous application of HBO therapy with CP prevents kidney damage.

PMID: 15902426 [PubMed - indexed for MEDLINE]

3: Amyotroph Lateral Scler Other Motor Neuron Disord. 2004 Dec;5(4):250-4.

A Phase I safety study of hyperbaric oxygen therapy for amyotrophic lateral sclerosis.

Steele J, Matos LA, Lopez EA, Perez-Pinzon MA, Prado R, Busto R, Arheart KL, Bradley WG.

Department of Neurology, University of Miami School of Medicine, Miami, FL 33136, USA.

BACKGROUND: Vascular endothelial growth factor and mitochondrial abnormalities have been described in ALS and its animal models. We have reported that hyperbaric oxygen (HBO) treatment delayed the onset of weakness in the wobbler mouse.

OBJECTIVE: To perform a Phase I safety study of HBO in patients with ALS. **METHODS:** Five patients with ALS were treated for 60min with 100% oxygen at 2 atmospheres pressure daily for five days a week for four weeks. The patients reported any deterioration in their condition after each treatment, and their neurological condition was measured serially during the four weeks of the treatment, and for four further weeks. **RESULTS:** Four patients reported decreased fatigue, while one patient dropped out at three weeks because of increased fatigue. Maximum isometric voluntary contraction (MVIC) of all muscle groups except right hand grip improved significantly by up to 97%. Most improvement occurred during the four weeks after treatment. It is possible that the improvement in muscle strength was a placebo or a learning effect, though no such effects

have been detected in prior therapeutic trials in ALS using MVIC. No change was detected in other measures of neuromuscular function. CONCLUSIONS: A longer duration, placebo controlled trial in a larger number of patients is needed to determine the safety and efficacy of HBO. Until that is completed, it is not recommended that ALS patients should be treated with HBO. Publication Types: Clinical Trial Clinical Trial, Phase I PMID: 15799556 [PubMed - indexed for MEDLINE]

4: J Investig Med. 2004 Dec;52(8):523-30. Effects of carbon monoxide poisoning on neutrophil responses in patients treated with hyperbaric oxygen. Schnittger V, Rosendahl K, Lind F, Palmblad J. Department of Anesthesia and Intensive Care Medicine, Karolinska University Hospital, Solna, Sweden. vendela.s@swipnet.se BACKGROUND: Carbon monoxide (CO) poisoning can cause tissue injury. Neutrophil granulocytes have been proposed to contribute to the injury, which may be ameliorated by hyperbaric oxygen (HBO2) treatment. We sought to assess the relationship between acute CO poisoning and blood neutrophil count, plasma cytokine, and cortisol responses, as well as the mechanism behind the observed beneficiary effects of HBO2 treatment. METHODS: Eight patients (age 26-82 years) with severe acute CO poisoning were enrolled, concomitant with eight healthy controls (age 27-42 years), in a prospective, controlled, clinical study. The patients were given three HBO2 treatments (2.8 atmospheres absolute, 100 minutes) within the first 24 hours. The controls were given identical simultaneous HBO2 treatments. Venous blood samples were taken before and after each treatment. Results: At the start of the HBO2 treatment, patients displayed significantly higher blood neutrophil counts ($p < .0001$) and plasma cortisol levels ($p = .020$) than controls, but the two groups had similar values for interleukin-8, granulocyte colony-stimulating factor (G-CSF), neutrophil H2O2 generation, and CD16 and CD18 surface expression. During the observation time, neutrophil H2O2 accumulation declined in patients and in controls ($p = .031$), whereas the up-regulation of CD18 expression increased ($p = .002$) in both groups. Moreover, G-CSF levels became significantly higher in patients than in controls ($p = .015$). G-CSF levels also correlated significantly with neutrophil counts. Conclusion: CO poisoning was associated with discrete changes of blood neutrophil counts, cortisol, and G-CSF plasma concentrations. HBO2 treatment modulated neutrophil generation of H2O2 and surface expression of CD18. These changes may be part of the cascade of events leading to the sequelae of CO poisoning and their attenuation by HBO2. Publication Types: Clinical Trial PMID: 15682684 [PubMed - indexed for MEDLINE]

5: J Womens Health (Larchmt). 2004 Nov;13(9):1008-18. Can hyperbaric oxygen therapy reduce breast cancer treatment-related lymphedema? A pilot study. Teas J, Cunningham JE, Cone L, Jansen K, Raghavan SK, Nitcheva DK, Xie D, Butler WM. Department of Health Promotion Education and Behavior, Arnold School of Public Health, University of South Carolina and the South Carolina Cancer Center, Columbia, South Carolina, USA. jane.teas@palmettohealth.org OBJECTIVE: Arm lymphedema after surgery or radiation for breast cancer is common, causing pain and limitation of activities. Previous reports of hyperbaric oxygen (HBO) therapy for breast edema led us to consider the use of HBO therapy for arm lymphedema. METHODS: Ten healthy postmenopausal women (age 58 +/- 5.7 years) with persistent (9.4 years +/- 9.1 years) arm lymphedema following breast cancer surgery and radiation ($n = 10$) plus chemotherapy ($n = 7$) received 20 HBO treatments (90 minutes at 2.0 ATA five times a week for 4 weeks). End points included changes in upper extremity volume, platelet counts, plasma levels of vascular endothelial growth factor (VEGF), and lymph angiogenic-associated vascular endothelial growth factor-C (VEGF-C). Lymphedema volume (LV) was defined as the volume of the unaffected arm subtracted from the volume of the affected arm. RESULTS: We observed a 38% average reduction in hand lymphedema (-7.4 ml, 11.6 SD, range -30+8 ml, $p = 0.076$, 95% confidence interval -15.7-0.9 ml) at the end of HBO, which was independent of changes in body weight. For those who benefited ($n = 8$), the reduction was persistent from the end of treatment to a final measurement an average of 14.2 months after the last HBO treatment. However, total LV did not change significantly. VEGF-C increased from baseline ($p = 0.004$) before treatment 20, suggesting HBO had begun to stimulate this growth factor. CONCLUSIONS: Future studies should explore the effects of a greater number of HBO treatments on lymphedema, with more patients. Publication Types: Clinical Trial PMID: 15665658 [PubMed - indexed for MEDLINE]

6: Free Radic Res. 2004 Sep;38(9):927-32. Influence of an orally effective SOD on hyperbaric oxygen-related cell damage. Muth CM, Glenz Y, Klaus M, Radermacher P, Speit G, Leverve X. Sektion Anasthesiologische Pathophysiologie und Verfahrensentwicklung, Universitätsklinikum Ulm, D-89073 Ulm, Germany. claus-martin.muth@medizin.uni-ulm.de In a prospective, double-blind, randomised placebo-controlled study, we tested the hypothesis that a new formulation consisting of wheat gliadin chemically combined with a vegetal (thus orally effective) preparation of superoxide dismutase (SOD) allows to prevent hyperbaric oxygen (HBO)-induced oxidative cell stress. Twenty healthy volunteers were exposed

to 100% oxygen breathing at 2.5 ATA for a total of 60 min. DNA strand breaks (tail moments) were determined using the alkaline version of the comet assay. Whole blood concentrations of reduced (GSH) and oxidised (GSSG) glutathione and F2-isoprostanes, SOD, glutathione peroxidase (GPx) and catalase (Cat) activities and red cell malondialdehyde (MDA) content were determined. After HBO exposure the tail moment ($p = 0.03$) and isoprostane levels ($p = 0.049$) were significantly lower in the group that received the vegetal formulation. Neither SOD and Cat nor GSH and GSSG were significantly affected by this preparation or HBO exposure. By contrast, blood GPx activity, which tended to be lower in the SOD-group already before the HBO exposure ($p = 0.076$), was significantly lower afterwards ($p = 0.045$). We conclude that an orally effective SOD-wheat gliadin mixture is able to protect against DNA damage, which coincided with reduced blood isoprostane levels, and may therefore be used as an antioxidant.

Publication Types: Clinical Trial Randomized Controlled Trial

PMID: 15621710 [PubMed - indexed for MEDLINE]

7: J Am Acad Orthop Surg. 2004 Nov-Dec;12(6):376-84.

Hyperbaric oxygen therapy in extremity trauma.

Greensmith JE.

St Elizabeth Hospital, Appleton, WI 54915, USA.

Hyperbaric oxygen therapy potentially can provide enhanced oxygen delivery to peripheral tissues affected by vascular disruption, cytogenic and vasogenic edema, and cellular hypoxia caused by extremity trauma. After appropriate resuscitation, macrovascular repair, and fracture fixation/stabilization, adjunctive hyperbaric oxygen therapy can enhance tissue oxygen content. In patients with crush injury or early compartment syndrome, hyperbaric oxygen therapy may reduce the penumbra of cells at risk for delayed necrosis and secondary ischemia. Animal experiments and human case series suggest the benefits of such therapy, and recent randomized, prospective studies on trauma patients have confirmed its efficacy in those with extremity trauma. However, more data are necessary to determine additional indications as well as optimal timing and dosing for hyperbaric oxygen therapy.

Publication Types: Review Review, Tutorial

PMID: 15615503 [PubMed - indexed for MEDLINE]

8: Arch Surg. 2004 Dec;139(12):1339-45.

Hyperbaric oxygen treatment and survival from necrotizing soft tissue infection.

Wilkinson D, Doolette D.

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HYPOTHESIS: Necrotizing soft tissue infection (NSTI) refers to a spectrum of infective diseases characterized by necrosis of the deep soft tissues.

Features of manifestation and medical management have been analyzed for association with outcome. The use of hyperbaric oxygen (HBO(2)) therapy has been recommended as an adjunctive treatment but remains controversial. DESIGN: Retrospective cohort study. SETTING: A major tertiary hospital. PATIENTS: All patients admitted with a diagnosis of NSTI across a 5-year period. INTERVENTION: Features of manifestation and medical management were analyzed for their association with survival to hospital discharge. Long-term survival was analyzed for the intervention of HBO(2) therapy. MAIN OUTCOME MEASURES: Primary outcome was survival to hospital discharge. Secondary outcomes were limb salvage and long-term survival after hospital discharge. RESULTS: Forty-four patients were reviewed, with 6 deaths (14%). Survival was less likely in those with increased age, renal dysfunction, and idiopathic etiology of infection and in those not receiving HBO(2) therapy. Logistic regression determined the strongest association with survival was the intervention of HBO(2) therapy ($P = .02$). Hyperbaric oxygen therapy increased survival with an odds ratio of 8.9 (95% confidence interval, 1.3-58.0) and a number needed to treat of 3. For NSTI involving an extremity, HBO(2) therapy significantly reduced the incidence of amputation ($P = .05$). Survival analysis revealed an improved long-term outcome for the HBO(2) group ($P = .002$). CONCLUSION: Hyperbaric oxygen therapy was associated with improved survival and limb salvage and should be considered in the setting of NSTI.

PMID: 15611459 [PubMed - indexed for MEDLINE]

9: BJU Int. 2004 Dec;94(9):1275-8.

Hyperbaric oxygen does not accelerate latent in vivo prostate cancer: implications for the treatment of radiation-induced haemorrhagic cystitis.

Chong KT, Hampson NB, Bostwick DG, Vessella RL, Corman JM.

Section of Urology and Renal Transplantation, Virginia Mason Medical Center, Seattle, WA 98111, USA.

OBJECTIVE: To assess the effects of hyperbaric oxygen (HBO2; often used to treat haemorrhagic cystitis, a known side-effect after radiation therapy for prostate cancer and with the potential to induce tumour angiogenesis and stimulate latent recurrence) on indolent in vivo prostate cancer in a murine model. MATERIALS AND METHODS: Human prostate LNCaP cells were injected into 60 severe combined-immunodeficient mice; of these 24 (40%) did not develop palpable tumours after 6 weeks. They were randomized to undergo 20 sessions of either HBO2 or normobaric air in standardized conditions, and observed for another 4 weeks before the histological assessment of any palpable tumours that developed. Analysis of developed LNCaP tumours included tumour volume, microvessel density, MIB-1, p53, p27 and racemase staining intensity. RESULTS: HBO2 was associated with less prostate tumour progression than normobaric air ($P = 0.26$).

During HBO2 therapy, 10 mice remained free of palpable tumours, compared with seven controls ($P = 0.30$). On evaluation during the 4 weeks after therapy, six mice treated with HBO2 remained free of palpable tumours, vs eight of the controls ($P = 0.17$). There was tumour invasion and necrosis in a two of six and four of the HBO2 group during and after therapy, respectively, vs five and seven of the controls. Tumour microvessel density, proliferative index, differentiation and apoptosis markers were similar in both groups. CONCLUSIONS: HBO2 does not accelerate the growth of indolent prostate cancer in a murine model, suggesting that it does not increase the risk of residual prostate cancer reactivation when it is used to manage radiation-induced haemorrhagic cystitis in patients treated by pelvic radiotherapy for prostate cancer. PMID: 15610104 [PubMed - indexed for MEDLINE]

10: Swiss Med Wkly. 2004 Oct 30;134(43-44):650-5. Visual loss as a late complication of carbon monoxide poisoning and its successful treatment with hyperbaric oxygen therapy. Ersanli D, Yildiz S, Togrol E, Ay H, Qyrdedi T. Gulhane Military Medical Academy, Haydarpasa Training Hospital, Department of Ophthalmology, Kadikoy/Istanbul, Turkey. PURPOSE: To present recovery from vision loss due to CO poisoning with Hyperbaric Oxygen (HBO) therapy in two patients. METHODS: Two female patients developed visual deterioration after carbon monoxide (CO) poisoning and were treated with hyperbaric oxygen therapy (HBO). Clinical examination, including visual acuity assessment, visual field examination and visual evoked potentials (VEPs) before and after the HBO therapy were performed. RESULTS: In Case 1 the visual loss started on the third day with visual acuity at the level of perception of hand movements at 10 cm in the right eye and finger count at 10 cm in the left eye. The visual evoked potentials (VEPs) had low amplitudes and prolonged (128 msec bilaterally) latencies (implicit times). After 48 sessions and 52 days of HBO therapy, the visual acuity became 0.2 in the right eye and 0.15 in the left eye. Visual field examination revealed homonymous right lower quadrant anopsia. The VEPs also improved. In Case 2 the visual acuity was 0.2 in the right eye and 0.1 in the left eye on the 6th day following the accident when the patient was admitted for treatment. The VEP latencies were within normal limits. After 36 days and 35 sessions of HBO therapy, the visual acuity became 0.7 on both eyes. The visual fields completely normalised. The VEP latencies in this case also became shorter. CONCLUSION: It appears that the adverse effects of CO poisoning continue to progress during the late period and we believe that HBO treatment in this period may still be effective and will prevent some of the neurological sequelae such as visual loss from becoming permanent. Clinical, neurological, neuropsychological, visual outcome seems to be favourable even if HBO

treatment started as late as 6 or 8 days after the exposure to CO.

Publication Types: Case Reports

PMID: 15609209 [PubMed - indexed for MEDLINE]

11: Eur Surg Res. 2004 Nov-Dec;36(6):350-6.

Beneficial effect of hyperbaric oxygen therapy on liver regeneration after 90% hepatectomy in rats.

Nagamine K, Kubota T, Togo S, Nagashima Y, Mori M, Shimada H.

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BACKGROUND/AIMS: Hyperbaric oxygen therapy (HBOT) has been reported to augment oxygen delivery to ischemic tissues and improve the liver dysfunction in clinical cases. HBOT was performed after 90% hepatectomy in rats to determine its effect on the regeneration of remnant liver. METHODS: After 90% hepatectomy was performed in 8-week-old male Wistar rats, the animals were subdivided into an HBOT (2 atm abs., 80% O₂, 1 h/day, 3 days) group and a non-HBOT group. Members of both groups were sacrificed, usually every 4 h until a maximum of 50 h after hepatectomy, and the liver regeneration rate, the proportion of PCNA-positive cells and the ATP volume in the remnant tissues were examined. RESULTS: In the HBOT group, the liver regeneration rate at 36 h and 50 h after operation and the proportion of PCNA positive cells at 8 h was significantly increased compared with the non-HBOT group. The ATP volume in the remnant livers in the HBOT group was also significantly increased at 12 h. CONCLUSION: HBOT augmented liver regeneration after hepatectomy by stabilization of energy metabolism induced by oxygen delivery in rats. 2004 S. Karger AG, Basel.

PMID: 15591743 [PubMed - indexed for MEDLINE]

12: South Med J. 2004 Nov;97(11):1060-5.

Ice storm-related carbon monoxide poisonings in North Carolina: a reminder.

Ghim M, Severance HW.

Duke University Medical Center, Department of Surgery, Division of Emergency Medicine, Durham, NC 27710, USA.

Severe winter weather, such as ice storms, that results in loss of electrical power, is frequently mentioned as a contributing factor in acute carbon monoxide (CO) poisoning. However, in our literature review, such events are infrequently reported. This article reports on such an event in which more than 200 patients were evaluated and treated at a single facility because of the crippling effects of an ice storm leading to prolonged loss of power and subsequent catastrophes with alternative heating and cooking sources. One hundred seventy-six patients were treated and subsequently released after Emergency Department-based treatment for CO exposure, and three patients were admitted. Eighteen patients were treated with hyperbaric treatments and discharged. Three others

left before treatment was completed. Three cases representing varying levels of severity at presentation leading to differing treatment algorithms are discussed to demonstrate a suggested clinical decision pathway in the treatment of unintentional CO poisoning.

Publication Types: Case Reports

PMID: 15586595 [PubMed - indexed for MEDLINE]

13: J Trauma. 2004 Nov;57(5):1060-4.

Effect of hyperbaric oxygen therapy on patellar tendinopathy in a rabbit model.

Hsu RW, Hsu WH, Tai CL, Lee KF.

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BACKGROUND: Hyperbaric oxygen therapy is a method for augmenting oxygen availability to tissues.

This study investigated the effect of hyperbaric oxygen therapy on the collagenase-induced tendinopathy in the rabbit patellar tendon.

METHODS: In this study, 13 rabbits were treated by ultrasound-guided injection of 0.025 mL collagenase into the patellar tendon at the right knee, with the left knee serving as a control condition. The rabbits were randomly divided into two groups. After tendinopathy had been confirmed by histologic examination 3 weeks after treatment, hyperbaric oxygen therapy was initiated for group 1. The hyperbaric oxygen therapy involved 30 daily sessions of 2.5 ATA for 120 minutes starting 6 weeks after treatment. The rabbits in group 2 were put in normobaric room air. Both groups were killed 10 weeks after treatment. Histologic examinations as well as mechanical and biochemical tests were performed after the animals were killed. **RESULTS:** The ultimate tensile load in the tendon that had hyperbaric oxygen therapy was 34.8% greater than that in the control tendon 10 weeks after treatment ($p < 0.05$). Hydroxyproline concentrations increased 82.2% simultaneously in the tendons that had hyperbaric oxygen therapy, as compared with the concentrations in the control tendons ($p < 0.05$). However, no statistical difference was found between the two groups in terms of pyridinoline concentration at the 10th week ($p > 0.05$). The histologic examination demonstrated an increase in blastlike tenocytes in group 1, with more mature phenotype, more organized collagen matrix, absence of myxoid degeneration, and increased vascularity at the 10th week, as compared with the control knee. **CONCLUSIONS:** The results validate the effectiveness of hyperbaric oxygen therapy in the treatment of tendinopathy. Hyperbaric oxygen therapy may increase collagen synthesis and collagen cross-link formation during the early healing process. PMID: 15580033 [PubMed - indexed for MEDLINE]

14: J Appl Physiol. 2005 Apr;98(4):1309-13. Epub 2004 Dec 3.

Optimal oxygen pressure and time for reduced bubble formation in the N₂-saturated decompressed prawn.

Ertracht O, Arieli R, Arieli Y, Ron R, Erlichman Z, Adir Y.

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Bubbles that grow during decompression are believed to originate from preexisting gas micronuclei. We showed that pretreatment of prawns with 203 kPa oxygen before nitrogen loading reduced the number of bubbles that evolved on decompression, presumably owing to the alteration or elimination of gas micronuclei (Arieli Y, Arieli R, and Marx A. J Appl Physiol 92: 2596-2599, 2002). The present study examines the optimal pretreatment for this assumed crushing of gas micronuclei. Transparent prawns were subjected to various exposure times (0, 5, 10, 15, and 20 min) at an oxygen pressure of 203 kPa and to 5 min at different oxygen pressures (PO₂ values of 101, 151, 203, 405, 608, and 810 kPa), before nitrogen loading at 203 kPa followed by explosive decompression. After the decompression, bubble density and total gas volume were measured with a light microscope equipped with a video camera. Five minutes at a PO₂ of 405 kPa yielded maximal reduction of bubble density and total gas volume by 52 and 71%, respectively. It has been reported that 2-3 h of hyperbaric oxygen at bottom pressure was required to protect saturation divers decompressed on oxygen against decompression sickness. If there is a shorter pretreatment that is applicable to humans, this will be of great advantage in diving and escape from submarines.

PMID: 15579569 [PubMed - indexed for MEDLINE]

15: World J Surg. 2004 Oct;28(10):1068-9; author reply 1069. Epub 2004 Sep 29.

Comment on: World J Surg. 2004 Mar;28(3):312-5.

Experiences in applying oxygen therapy in some otolaryngological diseases.

Narozny W, Kuczkowski J, Mikaszewski B.

Publication Types: Comment Letter

PMID: 15573269 [PubMed - indexed for MEDLINE]

16: Am Fam Physician. 2004 Nov 15;70(10):1860.

Comment on: Am Fam Physician. 2003 Jul 15;68(2):309-12.

Hyperbaric oxygen to treat malignant external otitis.

Narozny W, Kuczkowski J, Mikaszewski B.

Publication Types: Comment Letter

PMID: 15571053 [PubMed - indexed for MEDLINE]

17: Undersea Hyperb Med. 2004 Fall;31(3):285-9.

Hyperbaric oxygen therapy in the treatment of carbon dioxide gas embolism.

Gorji R, Camporesi EM.

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Publication Types: Case Reports

PMID: 15568416 [PubMed - indexed for MEDLINE]

18: Undersea Hyperb Med. 2004 Fall;31(3):281-4.
Emphysematous cystitis: rapid resolution of symptoms with hyperbaric treatment: a case report.
McCabe JB, Mc-Ginn Merritt W, Olsson D, Wright V, Camporesi EM.

Department of Emergency Medicine, SUNY Upstate Medical University, USA.

INTRODUCTION: Emphysematous cystitis is a rare disease that occurs most often in elderly diabetic patients characterized by gas formation in the bladder wall due to infection. The infecting organism is usually an aerobic bacterium, most commonly *E. coli* although anaerobic species have also been reported. We report the use of hyperbaric oxygen in a patient with emphysematous cystitis and air in the femoral vein in which the treatment rapidly resolved the symptoms and radiological abnormalities.
METHODS: A 65-year-old female presented to the Emergency Department with altered mental status, weakness, dark urine, dysuria and fever. She was febrile and lethargic. Abdominal exam showed suprapubic tenderness. Urinalysis was positive for white blood cells and bacteria. A CT scan of the abdomen demonstrated extensive air in the bladder wall with an air bubble in the femoral vein. Presumptive diagnosis was urinary tract infection, emphysematous cystitis, and sepsis. A question of air embolism was raised due to the intravascular gas. The patient was treated with hyperbaric oxygen (2.85 atm abs, 90 minutes) on two separate occasions in the first 12 hours. Within 24 hours, the patient's condition rapidly improved. Repeat CT scan 48 hours after admission showed near complete resolution of the emphysematous cystitis. The patient grew *Klebsiella pneumoniae* from her urine.
CONCLUSIONS: Emphysematous cystitis is a rare condition caused by either aerobic or anaerobic bacteria and may be associated with both bladder wall and intravascular gas formation. Hyperbaric oxygen therapy has not been previously reported as a treatment modality. The rapid improvement in our patient may indicate a role for hyperbaric oxygen in addition to IV hydration and antibiotics in this disease.

Publication Types: Case Reports

PMID: 15568415 [PubMed - indexed for MEDLINE]

19: Undersea Hyperb Med. 2004 Fall;31(3):275-9.
Transient osteoporosis associated with hyperhomocystinemia: a possible role for hyperbaric oxygen therapy.
Domachevsky L, Keynan Y, Militianu D, Goldenberg I, Adir Y.
Israel Naval Medical Institute, Israel Defense Forces Medical Corps, PO Box 8040, 31 080 Haifa, Israel.
Transient osteoporosis of the hip is considered by some to be an early stage of avascular necrosis. Hyperbaric oxygen (HBO₂) therapy, which may be of benefit in the treatment of avascular necrosis, might therefore be used in the treatment of transient osteoporosis of the hip. We present a case of transient osteoporosis associated with elevated levels of

homocysteine in a 33-year-old white male, who was treated by HBO₂. Treatment was administered at 2.5 ATA for 90 minutes once daily, five days per week. Regular follow-up examinations in the course of the HBO₂ therapy revealed improvement in the patient's complaints and the findings of the physical examination. Repeated magnetic resonance imaging (MRI) performed after 40 and 90 sessions showed decreased edema and complete resolution of the edema, respectively. Evaluation 6 months after the completion of treatment revealed complete resolution of symptoms, with a normal physical examination.

Publication Types: Case Reports

PMID: 15568414 [PubMed - indexed for MEDLINE]

20: Klin Padiatr. 2004 Nov-Dec;216(6):370-8.

Bone marrow oedema and aseptic osteonecrosis in children and adolescents with acute lymphoblastic leukaemia or non-Hodgkin-lymphoma treated with hyperbaric-oxygen-therapy (HBO): an approach to cure? -- BME/AON and hyperbaric oxygen therapy as a treatment modality.

Bernbeck B, Christaras A, Krauth K, Lentrodt S, Strelow H, Schaper J, Janssen G, Modder U, Gobel U.

Clinic for Paediatric Oncology, -Haematology and -Immunology, Heinrich-Heine-University, Dusseldorf.
BACKGROUND: There is a striking need for additional therapies of bone marrow oedema (BME) and aseptic osteonecrosis (AON) in paediatric oncology patients. Hyperbaric oxygenation (HBO) therapy used in the treatment of osteoradionecrosis is demonstrated effectiveness. Aim of this retrospective analysis was to investigate whether HBO-therapy might lead to subjective as well as objective effects in the treatment of BME and/or AON in paediatric oncology patients with acute lymphoblastic leukaemia (ALL) or Non-Hodgkin lymphoma (NHL).
PATIENTS AND METHODS: Between 11/1988 and 01/2001 27/291 (9.3 %) patients with ALL or NHL were diagnosed with a BME and/or AON in the Clinic for Paediatric Oncology, Haematology, and Immunology at University of Dusseldorf. 19/27 patients were submitted to HBO-therapy. Patients received average 45 HBO-treatments per patient (min. 13, max. 80 treatments). The affected regions were re-evaluated with MRI for radiological extent of lesions every 3 months. Pain in its intensity and localisation was serially recorded during HBO-therapy as key symptom in 11 of 19 patients.
RESULTS: 27 patients (15 females, 12 males; mean age at diagnosis of malignancy 8.2 +/- 4.7 (SD) years, range 7 months to 16 years) presented with 138 lesions. 133/138 lesions were localised in the lower extremities. At diagnosis of BME and/or AON, 78/133 lesions were shown in females and 55/133 lesions in male. Girls < 10 years predominantly presented BME (33 BME vs. 6 AON), girls aged > 10 years predominantly offered AON (28 AON vs. 11 BME). BME was more often exhibited in boys < 10 years (34 BME vs. 10 AON) and rarely in boys > 10 years (4 BME vs. 6 AON). 11 patients

treated with HBO-therapy were serially evaluated for pain intensity throughout their HBO-therapy courses by visual analogue scale (VAS) assessment. During the first 15 treatment courses the HBO-therapy a clear-cut reduction of pain was observed. The mean pain score before the first HBO-treatment unit was 2.4 +/- 2.7 (X +/- SD), decreased before the fifth to 1.6 +/- 1.7 and prior to the 35 (th) and 40 (th) HBO treatment to 0. Girls < 10 years treated with HBO showed an increase of BME (31 --> 46) and declining AON numbers (6 --> 2). Girls > 10 years with and without HBO-therapy showed decrease of BME lesions (7 --> 4 vs. 4 --> 0), whereas AON increased in the HBO-treated group (28 --> 29) as well as the non-treated group (0 --> 4). Males < 10 years showed an increase in BME lesion numbers despite HBO intervention (24 --> 26). The AON lesion numbers dropped in parallel (6 --> 3). Male patients not treated with HBO showed constant numbers of BME (11-->11) and a decreased numbers of AON (4 --> 2). All differences are statistically not significant. CONCLUSIONS: Children and adolescents diagnosed with ALL or NHL have a risk for accrument of BME and/or AON irrespective of the age, with an almost exclusive involvement of the lower extremities. Lesions of pedal bones and ankle joints predominantly affect children < 10 years. Lesions of knee and hip joints predominantly affect children > 10 years. In children < 10 years of age we demonstrate declining AON numbers and conversion of AON to BME thereby implicating possible beneficial effect of HBO in such patients. HBO failed to show beneficial effect on BME whether by preventing new lesions or by improving existent lesions in children > 10 years.

Publication Types: Evaluation Studies
PMID: 15565553 [PubMed - indexed for MEDLINE]

21: Anesth Analg. 2004 Dec;99(6):1878.
Hyperbaric oxygen therapy and pain management in a child with continuous infraclavicular brachial plexus block.

Minville V, Chassery C, Kern D, Fourcade O, Dadure C.

Publication Types: Case Reports Letter
PMID: 15562104 [PubMed - indexed for MEDLINE]

22: Aviat Space Environ Med. 2004 Nov;75(11):995-6.

Comment on: Aviat Space Environ Med. 2004 Nov;75(11):992-4.

Risk vs. benefit in hyperbaric medicine: a brief comment.

Fife CE.
Memorial Hermann Center for Hyperbaric Medicine and Wound Care, Department of Anesthesiology, University of Texas Health Science Center, Houston, TX 77030, USA. Caroline.E.Fife@uth.tmc.edu

Publication Types: Comment
PMID: 15559002 [PubMed - indexed for MEDLINE]

23: Aviat Space Environ Med. 2004 Nov;75(11):992-4.

Comment in: Aviat Space Environ Med. 2004 Nov;75(11):995-6.

Seizure incidence in 80,000 patient treatments with hyperbaric oxygen.

Yildiz S, Aktas S, Cimsit M, Ay H, Togrol E.
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INTRODUCTION: Hyperbaric oxygen treatment (HBOT) involves some risk of central nervous system (CNS) oxygen toxicity, which may be revealed by various signs and symptoms including seizures in patients breathing O₂ at pressures of 2 ATA or higher. The aim of this study was to determine the incidence of such seizures in the Underwater and Hyperbaric Medicine Departments of two university hospitals. METHODS: We retrospectively evaluated 80,679 patient-treatments for 9 clinical indications to determine the incidence of seizures attributable to CNS O₂ toxicity. Because different protocols were used for HBOT, the treatments were studied in four groups according to the chamber type used and the medical facility at which it was located. RESULTS: Only 2 seizures were documented, yielding an incidence of 2.4 per 100,000 patient-treatments. Both cases occurred in a multiplace chamber pressurized to 2.4 ATA with O₂ delivered by mask for three x 30 min with 5-min air breaks. DISCUSSION: The seizure incidence reported here is lower than other studies published in the literature. The delivery of O₂ by mask rather than hood may be a factor. Nevertheless, it appears that the risk of seizures due to CNS O₂ toxicity during HBOT is very low as long as appropriate exclusion criteria and treatment profiles are used.
PMID: 15559001 [PubMed - indexed for MEDLINE]

24: Otol Neurotol. 2004 Nov;25(6):916-23.

Usefulness of high doses of glucocorticoids and hyperbaric oxygen therapy in sudden sensorineural hearing loss treatment.

Narozny W, Sicko Z, Przewozny T, Stankiewicz C, Kot J, Kuczkowski J.

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OBJECTIVE: We investigated the effect of pharmacologic (steroids, vasodilators, vitamins, and Betaseric) and hyperbaric oxygen therapy on patients with sudden sensorineural hearing loss. METHODS: The pharmacologic arm of the study consisted of 52 patients with defined sudden sensorineural hearing loss treated simultaneously in the ENT Department and National Center for Hyperbaric Medicine of the Medical University of Gdansk, Poland, from 1997 to 2000 (Group A). The hyperbaric oxygen therapy consisted of exposure to 100% oxygen at a pressure of 250 kPa for a total of 60 minutes in a multiplace hyperbaric chamber. The control group included 81 patients with defined sudden sensorineural hearing

loss treated in the ENT Department, Medical University of Gdansk, from 1980 to 1996 (Group B). Both groups were comparable regarding the age of the patients, season of hearing loss occurrence, tinnitus and vestibular symptom frequency, delay before therapy, and average threshold loss before the start of treatment. The treatment results (hearing gain) were estimated using pure-tone audiometry. We retrospectively analyzed the audiograms of all patients. RESULTS: Patients from Group A (blood flow-promoting drugs, glucocorticoids in high doses, betahistine, and hyperbaric oxygen therapy) showed significantly better recovery of hearing levels compared with those from Group B (blood flow-promoting drugs and glucocorticoids in low doses) at seven frequencies (500, 1,000, 2,000, 3,000, 4,000, 6,000, and 8,000 Hz) ($p < 0.05$) and four groups of frequencies (pure-tone average, high-tone average, pure middle-tone average, and overall average) ($p < 0.05$). Percentage hearing gain in all investigated frequencies was also better in Group A versus Group B, and the differences were statistically significant ($p < 0.05$). CONCLUSION: We conclude that hyperbaric oxygen therapy with high doses of glucocorticoids improves the results of conventional sudden sensorineural hearing loss treatment and should be recommended. In addition, the best results are achieved if the treatment is started as early as possible.
PMID: 15547420 [PubMed - indexed for MEDLINE]

25: Eur Cytokine Netw. 2004 Jul-Sep;15(3):217-21. Whole blood pro-inflammatory cytokines and adhesion molecules post-lipopolysaccharides exposure in hyperbaric conditions. Fildissis G, Venetsanou K, Myrianthefs P, Karatzas S, Zidianakis V, Baltopoulos G. Athens University School of Nursing Hyperbaric Oxygen Therapy Unit and Intensive Care Unit at KAT General Hospital, Athens 14561, Greece. fildiss@hol.gr
Hyperbaric oxygen (HBO) is a therapeutic intervention with applications in a large variety of diseases, including traumatic injuries and acute or chronic infections. The presence of pro-inflammatory cytokines regulates certain factors including adhesion molecules, which play a significant role in HBO effects. We have investigated the effect of HBO on pro-inflammatory cytokine release [tumor necrosis factor-alpha (TNF-alpha), interleukin 6 and 8 (IL-6 and IL-8)], and the regulation of adhesion molecules [soluble intercellular adhesion molecule-1 (sICAM-1) and soluble vascular adhesion molecule (sVCAM)] after lipopolysaccharide (LPS) stimulation in 16 healthy individuals, originating from an urban area. A total number of 64 samples were treated, divided into four groups: Group A: not stimulated with LPS and not exposed to HBO. Group B: stimulated with LPS and not exposed to HBO. Group C: not stimulated with LPS and exposed to HBO. Group D: stimulated with LPS and exposed to HBO. The LPS stimulation dose was 100 pg/ml for

0.1 ml whole blood diluted 1:10. After incubation, samples were exposed to HBO with 100% O₂ at 2.4 atmospheres absolute (ATA) for 90 min. TNF-alpha, IL-6, IL-8 and sICAM-1, sVCAM levels were determined in culture supernatant, with ELISA. We observed an enhanced effect of LPS stimulation following exposure to HBO, which caused an increase in cytokine production (TNF-alpha, IL-6, IL-8), a reduction in sICAM, and no change to sVCAM, while their levels without stimulation remained almost invariable. The decrease in sICAM levels could be related to the increased levels of IL-8, as the production of this chemokine is involved in the regulation of adhesion molecules.
PMID: 15542446 [PubMed - indexed for MEDLINE]

26: Emerg Med Australas. 2004 Oct-Dec;16(5-6):481; discussion 481-2.
Comment on: Emerg Med Australas. 2004 Apr;16(2):151-4.
Response to review article on acute carbon monoxide poisoning and the role of hyperbaric oxygen.
Hampson NB.
Publication Types: Comment Letter
PMID: 15537420 [PubMed - indexed for MEDLINE]

27: Emerg Med Australas. 2004 Oct-Dec;16(5-6):480-1; discussion 481-2.
Comment on: Emerg Med Australas. 2004 Apr;16(2):151-4.
Carbon monoxide poisoning and hyperbaric oxygen: a fairer hearing?
Smart D.
Publication Types: Comment Letter
PMID: 15537418 [PubMed - indexed for MEDLINE]

28: Emerg Med Australas. 2004 Oct-Dec;16(5-6):394-9; discussion 481-2.
Comment on: Emerg Med Australas. 2004 Apr;16(2):101-2. Emerg Med Australas. 2004 Apr;16(2):151-4.
Carbon Monoxide Research Group, LDS Hospital, Utah in reply to Scheinkestel et al. and Emerson: the role of hyperbaric oxygen in carbon monoxide poisoning.
Weaver LK, Hopkins RO, Chan KJ, Thomas F, Churchill SK, Elliott CG, Morris A.
Department of Medicine, Pulmonary/Critical Care Divisions, LDS Hospital, Salt Lake City, Utah, USA. lweaver@ihc.com
This comprehensive response was invited by the Editor of Emergency Medicine Australasia to allow our Group from Salt Lake City, Utah to review the two articles 'Where to now with carbon monoxide poisoning?' by Scheinkestel et al. and the accompanying Commentary: 'The dilemma of managing carbon monoxide poisoning' by Emerson published in the April issue of Emergency Medicine Australasia.
Publication Types: Comment
PMID: 15537400 [PubMed - indexed for MEDLINE]

29: J Clin Oncol. 2004 Dec 15;22(24):4893-900. Epub 2004 Nov 1.

Comment in: J Clin Oncol. 2004 Dec 15;22(24):4867-8. J Clin Oncol. 2005 Jul 1;23(19):4465; author reply 4466-8.

Hyperbaric oxygen therapy for radionecrosis of the jaw: a randomized, placebo-controlled, double-blind trial from the ORN96 study group.

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PURPOSE: To determine the efficacy and safety of hyperbaric oxygen therapy (HBO) for overt mandibular osteoradionecrosis. **PATIENTS AND METHODS:** This prospective, multicenter, randomized, double-blind, placebo-controlled trial was conducted at 12 university hospitals. Ambulatory adults with overt osteoradionecrosis of the mandible were assigned to receive 30 HBO exposures preoperatively at 2.4 absolute atmosphere for 90 minutes or a placebo, and 10 additional HBO dives postoperatively or a placebo. The main outcome measure was 1-year recovery rate from osteoradionecrosis. Secondary end points included time to treatment failure, time to pain relief, 1-year mortality rate, and treatment safety. **RESULTS:** At the time of the second interim analysis, based on the triangular test, the study was stopped for potentially worse outcomes in the HBO arm. A total of 68 patients were enrolled and analyzed. At 1 year, six (19%) of 31 patients had recovered in the HBO arm and 12 (32%) of 37 in the placebo arm (relative risk = 0.60; 95% CI, 0.25 to 1.41; P = .23). Time to treatment failure (hazard ratio = 1.33; 95% CI, 0.68 to 2.60; P = .41) and time to pain relief (hazard ratio = 1.00; 95% CI, 0.52 to 1.89; P = .99) were similar between the two treatment arms. **CONCLUSION:** Patients with overt mandibular osteoradionecrosis did not benefit from hyperbaric oxygenation.

Publication Types: Clinical Trial Multicenter Study Randomized Controlled Trial

PMID: 15520052 [PubMed - indexed for MEDLINE]

30: J Clin Oncol. 2004 Dec 15;22(24):4867-8. Epub 2004 Nov 1.

Comment on: J Clin Oncol. 2004 Dec 15;22(24):4893-900.

Mandibular osteoradionecrosis.

Mendenhall WM.

Publication Types: Comment Editorial

PMID: 15520050 [PubMed - indexed for MEDLINE]

31: J Am Coll Surg. 2004 Nov;199(5):836-7.

Comment on: J Am Coll Surg. 2004 Feb;198(2):243-63.

Burns and metabolism.

Weaver LK.

Publication Types: Comment Letter

PMID: 15501129 [PubMed - indexed for MEDLINE]

32: RN. 2004 Sep;67(9):30-5; quiz 36.

HBO therapy: beyond the bends.

Bailey DL, Jackson L, White D.

Norwalk Hospital, Norwalk, CT, USA.

Publication Types: Review Review, Tutorial

PMID: 15497344 [PubMed - indexed for MEDLINE]

33: Cochrane Database Syst Rev. 2004 Oct 18;(4):CD004609.

Hyperbaric oxygen therapy for the adjunctive treatment of traumatic brain injury.

Bennett MH, Trytko B, Jonker B.

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BACKGROUND: Traumatic brain injury is common and presents a health problem with significant effect on quality of life. Hyperbaric oxygen therapy (HBOT) has been suggested to improve oxygen supply to the injured brain and, therefore, to reduce the volume of brain that will ultimately perish. It is postulated that the addition of HBOT to the standard intensive care regimen may result in a reduction in patient death and disability as a result of these additional brain-preserving effects. **OBJECTIVES:** To assess the benefits and harms of adjunctive HBOT for treating traumatic brain injury. **SEARCH STRATEGY:** We searched CENTRAL (The Cochrane Library Issue 4, 2003), MEDLINE (1966 - 2003), EMBASE (1974 - 2003), CINAHL (1982 - 2003), DORCTHIM (1996 - 2003), and reference lists of articles. Relevant journals were handsearched and researchers in the field were contacted. **SELECTION CRITERIA:** Randomised studies comparing the effect on traumatic brain injury of therapeutic regimens which include HBOT with those that exclude HBOT (with or without sham therapy). **DATA COLLECTION AND ANALYSIS:** Three reviewers independently evaluated the quality of the relevant trials using the validated Oxford-Scale (Jadad 1996) and extracted the data from the included trials. **MAIN RESULTS:** Four trials contributed to this review (382 patients, 199 receiving HBOT and 183 control). There was a trend towards, but no significant increase in, the chance of a favourable outcome when defined as full recovery, Glasgow outcome score 1 or 2, or return to normal activities of daily living (relative risk [RR] for good outcome with HBOT 1.94, 95% confidence interval [CI] 0.92 to 4.08, P=0.08). Pooled data from the three trials with 327 patients that reported mortality, showed a significant reduction in the risk of dying when HBOT was added to the treatment regimen (RR 0.69, 95% CI 0.54 to 0.88, P=0.003). Heterogeneity between studies was low (I(2) =0%), and sensitivity analysis for the allocation of dropouts did not affect that result. This analysis suggests we would have to treat seven patients to avoid one extra death (number needed to treat [NNT] 7, 95% CI 4 to 22). One trial suggested intracranial pressure was favourably lower

in those patients receiving HBOT in whom myringotomies had been performed (WMD with myringotomy -8.2 mmHg, 95% CI -14.7 mmHg to -1.7 mmHg, $P=0.01$), while in two trials there was a reported incidence of 13% for significant pulmonary impairment in the group receiving HBOT versus 0% in the non-HBOT group ($P=0.007$). REVIEWERS' CONCLUSIONS: In people with traumatic brain injury, the addition of HBOT significantly reduced the risk of death but not of favourable clinical outcome. The routine application of HBOT to these patients cannot be justified from this review. In view of the modest number of patients, methodological shortcomings and poor reporting, this result should be interpreted cautiously, and an appropriately powered trial of high methodological rigour is justified to define those patients (if any) who can be expected to derive most benefit from HBOT.

Publication Types: Meta-Analysis Review
PMID: 15495120 [PubMed - indexed for MEDLINE]

34: N Engl J Med. 2004 Oct 14;351(16):1694-5; author reply 1694-5.

Comment on: N Engl J Med. 2004 Jul 1;351(1):48-55.

Neuropathic diabetic foot ulcers.

Aksenov IV.

Publication Types: Comment Letter
PMID: 15490498 [PubMed - indexed for MEDLINE]

35: Undersea Hyperb Med. 2004 Summer;31(2):251-60.

The effect of hyperbaric oxygen on human oral cancer cells.

Sun TB, Chen RL, Hsu YH.

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Discoveries of the beneficial cellular and biochemical effects have strengthened the rationale for the administration of hyperbaric oxygen therapy (HBO2) as an adjunctive therapy for the treatment of osteoradionecrosis (ORN). Malignancies, however, are considered a contraindication for HBO2 because of the possible tumor-promoting effects. The aim of this study was to examine the effects of HBO2 therapy on tumor weight, and to measure the progression of apoptosis and tumor cell proliferating activity in a cultured human oral cancer cell line. Twenty 5-week-old male NODscid mice underwent daily HBO2 of 2.5 atm abs, 90 minutes for 20 treatments. The control group, $n = 20$, did not undergo HBO2 and tumor weight, apoptosis index, and proliferating activity parameters were compared between the two groups. The results showed no significant differences ($p < 0.05$) in the whole-body weights, tumor weights, apoptotic index or proliferating activity index between the two groups. By using the apoptosis and proliferating activity assays which were better indicators of tumor cell growth than tumor weight alone, our results suggest that the clinical application of HBO2 does not

promote the growth or proliferation of human oral cancer cells.

PMID: 15485088 [PubMed - indexed for MEDLINE]

36: Undersea Hyperb Med. 2004 Summer;31(2):245-50.

Report of the use of hyperbaric oxygen therapy (HBO2) in an unusual case of secondary infertility.

Leverment J, Turner R, Bowman M, Cooke CJ.

Department of Diving and Hyperbaric Medicine, Prince of Wales Hospital, Randwick, NSW, Australia 2031.

We report the use of hyperbaric oxygen therapy (HBO2) in the treatment of an unusual case of secondary infertility. The patient had failed to conceive after a 1-year period of in-vitro fertilization, during which oral sildenafil had also been administered. However she became pregnant after an IVF cycle and the use of adjunctive HBO2 and sildenafil, which was administered intravaginally on this occasion. There is currently very little evidence to support the use of HBO2 in this context. The possible mechanisms of action of HBO2 in this case are discussed.

PMID: 15485087 [PubMed - indexed for MEDLINE]

37: Undersea Hyperb Med. 2004 Summer;31(2):227-32.

Superoxide dismutase responds to hyperoxia in rat hippocampus.

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The brain's anti-oxidant response to highly elevated oxygen (O_2) partial pressures is poorly understood. In this study we hypothesized that hyperbaric O_2 (HBO2) would stimulate superoxide dismutase (SOD) transcription in the oxidative stress-sensitive rat hippocampus and measured the time course and extent of the changes in hippocampal mRNA for all three SOD isoforms and total SOD enzyme activity. Comparisons were made between exposures to 2 hours of 1 atmosphere pressure normobaric oxygen (NBO); 2 hours of 3 atmospheres HBO2; and room air. Hyperoxia ($HBO_2 > NBO$) was associated with statistically significant increases in transcript levels of the antioxidant enzymes SOD2 (MnSOD) and SOD3 (EC-SOD) at 6 and 18 hours but not SOD1 (Cu, Zn SOD) respectively. Hyperoxia, however, did not affect total hippocampal SOD activity measured at 6 and 24 hours, indicating that the mRNA responses were necessary to maintain the anti-oxidant enzyme activity after oxidative stress.

PMID: 15485085 [PubMed - indexed for MEDLINE]

38: Undersea Hyperb Med. 2004 Summer;31(2):211-6.

Hyperbaric oxygen treatment time for cyclophosphamide induced cystitis in rats.

Oztas E, Korkmaz A, Oter S, Topal T.

Department of Medical Histology and Embryology, Gulhane Military Medical Academy, 06018 Etilik, Ankara, Turkey.

The aim of this study was to evaluate the prophylactic potential of hyperbaric oxygenation treatment and the timing of hyperbaric oxygen (HBO2) therapy for cyclophosphamide-(CYP) induced cystitis in rats. Forty male Sprague-Dawley rats were divided into 5 groups. Four groups received a single dose of CYP (100 mg/kg.) intraperitoneally (i.p.) at the same time (group 1 served as the control). Group 2 received CYP only; group 3 received HBO2 treatment (2.8 atmospheres absolute, 90 minutes, twice daily) before and the day after CYP. Group 4 received HBO2 before and on the day of CYP administration. Group 5 received HBO2 on the day of and the day after CYP. CYP injection resulted in severe cystitis. Prophylactic HBO2 treatment did not prevent the severe cystitis. After CYP injection, however, HBO2 treatment attenuated CYP-induced hemorrhagic cystitis in rats. Hyperbaric oxygen has a beneficial effect on repairing and healing bladder damage, though it does not function to prevent CYP-induced hemorrhagic cystitis.

PMID: 15485083 [PubMed - indexed for MEDLINE]

39: Undersea Hyperb Med. 2004 Summer;31(2):199-202.

Incidence of oxygen toxicity during the treatment of dysbarism.

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Oxygen (O₂) toxicity may result from exposure to partial pressures of O₂ above 0.6ATA. Potential toxic exposure for divers occurs during the treatment of dysbarism. In the recompression chamber, PO₂ may range from 0.9ATA to 3.3ATA depending upon the treatment table employed. This retrospective study examines the nature and incidence of O₂ toxicity in 998 patients who underwent recompression treatment at our facility from 1983 through 2001. Only patients evaluated for diving related injury were considered for this study. Of 1189 charts reviewed, 998 patients received recompression and were entered into this study. The total number of treatment exposures was determined as was the total number of O₂ toxicity events characterized as either pulmonary or CNS, and patients were divided into male/female analysis. Overall incidence as well as the incidence for both toxicity types was determined, and their occurrence in both male and female patients was ascertained. 2166 recompressions were undertaken, 449 female and 1717 male. The peak PO₂ for these treatments ranged from 2.6ATA to 2.9ATA. 155 O₂ toxicity events occurred in 152 patients, 49 females and 103 males. Three patients, 2 females and 1 male, had mixed events. Incidence of an O₂ toxic event = 7.0 per 100 recompressions. Incidence of pulmonary toxicity overall = 5.0 per 100 recompressions, while CNS events = 2.0 per 100 recompressions with

overall seizure rate = 0.6 per 100 recompressions. In females, pulmonary toxicity rate = 6.9 per 100 recompressions, CNS toxicity rate = 4.4 per 100 recompressions with seizures occurring at 1.3 per 100 recompressions. In males, pulmonary toxicity rate = 4.6 per 100 recompressions, CNS toxicity rate = 1.4 per 100 recompressions, and seizures at 0.4 per 100 recompressions.

PMID: 15485081 [PubMed - indexed for MEDLINE]

40: Undersea Hyperb Med. 2004 Summer;31(2):195-8.

Comment on: Undersea Hyperb Med. 2003 Summer;30(2):147-53. Undersea Hyperb Med. 2004 Summer;31(2):187; author reply 191. Undersea Hyperb Med. 2004 Summer;31(2):189-90. Extension of oxygen tolerance by interrupted exposure.

Clark JM.

Institute for Environmental Medicine, University of Pennsylvania Medical Center, USA.

Publication Types: Comment

PMID: 15485080 [PubMed - indexed for MEDLINE]

41: Undersea Hyperb Med. 2004 Summer;31(2):193-4.

How the Davis 2.36 ATA wound healing enhancement treatment table was established.

Sheffield PJ.

International ATMO, Inc, 414 Navarro, Ste 502, San Antonio, Texas 78205, USA.

Publication Types: Historical Article

PMID: 15485079 [PubMed - indexed for MEDLINE]

42: Undersea Hyperb Med. 2004 Summer;31(2):189-90.

Comment in: Undersea Hyperb Med. 2004 Summer;31(2):195-8.

Comment on: Undersea Hyperb Med. 2003 Summer;30(2):147-53.

Central nervous system oxygen toxicity during routine hyperbaric oxygen therapy.

Yildiz S, Ay H, Qyrdedi T.

Publication Types: Case Reports Comment Letter

PMID: 15485078 [PubMed - indexed for MEDLINE]

43: Undersea Hyperb Med. 2004 Summer;31(2):187; author reply 191.

Comment in: Undersea Hyperb Med. 2004 Summer;31(2):195-8.

Comment on: Undersea Hyperb Med. 2003 Summer;30(2):147-53.

Two differences in hyperbaric exposure.

Zwart BP.

Publication Types: Comment Letter

PMID: 15485077 [PubMed - indexed for MEDLINE]

44: Undersea Hyperb Med. 2004 Summer;31(2):185.

Comment on: Undersea Hyperb Med. 2003 Summer;30(2):147-53.

A mini-forum on air breaks and O₂ toxicity in clinical HBO₂ therapy.

Piantadosi CA.

Duke University Medical Center, USA.

Publication Types: Comment

PMID: 15485076 [PubMed - indexed for MEDLINE]

45: Wilderness Environ Med. 2004 Fall;15(3):198-201.

Use of the Gamow Bag by EMT-basic park rangers for treatment of high-altitude pulmonary edema and high-altitude cerebral edema.

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As part of an emergency medical system protocol, national park service rangers certified at the level of an emergency medical technician-basic (EMT-B) are taught to recognize and treat high-altitude pulmonary edema and high-altitude cerebral edema. In Sequoia and Kings Canyon National Parks, this is done with the assistance of physician on-line medical control as a backup. High-altitude pulmonary edema and high-altitude cerebral edema are both potentially fatal altitude illnesses that can be particularly problematic in the backcountry, where evacuation may be delayed. We report a case of high-altitude pulmonary edema and high-altitude cerebral edema occurring at moderate altitude that was successfully treated by park rangers with the Gamow Bag.

Publication Types: Case Reports

PMID: 15473460 [PubMed - indexed for MEDLINE]

46: Arch Phys Med Rehabil. 2004 Oct;85(10):1732.

Comment on: Arch Phys Med Rehabil. 2004 Jul;85(7):1198-204.

Hyperbaric oxygen for traumatic brain injury.

Whyte J.

Publication Types: Comment Letter

PMID: 15468040 [PubMed - indexed for MEDLINE]

47: Int J Radiat Oncol Biol Phys. 2004 Nov 1;60(3):871-8.

The efficacy of hyperbaric oxygen therapy in the treatment of radiation-induced late side effects.

Bui QC, Lieber M, Withers HR, Corson K, van Rijnsoever M, Elsaleh H.

Department of Radiation Oncology, David Geffen School of Medicine at UCLA, 200 Medical Plaza B265, Los Angeles, CA 90095-6951, USA.

PURPOSE: We investigated the efficacy of hyperbaric oxygen therapy (HBOT) in the management of patients with radiation-induced late side effects, the majority of whom had failed previous interventions. **METHODS AND MATERIALS:** Of 105 eligible subjects, 30 had either died or were not contactable, leaving 75 who qualified for inclusion in this retrospective study. Patients answered a questionnaire documenting symptom severity before and after treatment (using Radiation Therapy Oncology Group criteria), duration of improvement, relapse incidence, and

HBOT-related complications. **RESULTS:** The rate of participation was 60% (45/75). Improvement of principal presenting symptoms after HBOT was noted in 75% of head-and-neck, 100% of pelvic, and 57% of "other" subjects (median duration of response of 62, 72, and 68 weeks, respectively). Bone and bladder symptoms were most likely to benefit from HBOT (response rate, 81% and 83%, respectively). Fifty percent of subjects with soft tissue necrosis/mucous membrane side effects improved with HBOT. The low response rate of salivary (11%), neurologic (17%), laryngeal (17%), and upper gastrointestinal symptoms (22%) indicates that these were more resistant to HBOT. Relapse incidence was low (22%), and minor HBOT-related complications occurred in 31% of patients. **CONCLUSION:** Hyperbaric oxygen therapy is a safe and effective treatment modality offering durable relief in the management of radiation-induced osteoradionecrosis either alone or as an adjunctive treatment. Radiation soft tissue necrosis, cystitis, and proctitis also seemed to benefit from HBOT, but the present study did not have sufficient numbers to reliably predict long-term response.

PMID: 15465205 [PubMed - indexed for MEDLINE]

48: Am Heart J. 2004 Oct;148(4):E14.

Adjunctive effect of hyperbaric oxygen treatment after thrombolysis on left ventricular function in patients with acute myocardial infarction.

Dekleva M, Neskovic A, Vlahovic A, Putnikovic B, Beleslin B, Ostojic M.

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BACKGROUND: The role of hyperbaric oxygen in patients with acute myocardial infarction is controversial, ranging from not beneficial to having a favorable effect. This randomized study was conducted to further assess the benefit of hyperbaric oxygen treatment after thrombolysis on left ventricular function and remodeling in patients with acute myocardial infarction. **METHODS:** Seventy-four consecutive patients with first acute myocardial infarction were randomly assigned to treatment with hyperbaric oxygen treatment combined with streptokinase (HBO+) or streptokinase alone (HBO-). **RESULTS:** There was a significant decrease of end-systolic volume index from the first day to the third week in HBO+ patients compared with HBO- patients (from 30.40 to 28.18 vs from 30.89 to 36.68 mL/m², P <.05) accompanied with no changes of end-diastolic volume index in HBO+ compared with increased values in HBO- (from 55.68 to 55.10 vs from 55.87 to 63.82 mL/m², P <.05). Ejection fraction significantly improved in the HBO+ group and decreased in the HBO- group of patients after 3 weeks of acute myocardial infarction (from 46.27% to 50.81% vs from 45.54% to 44.05 %, P <.05). **CONCLUSIONS:** Adjunctive hyperbaric oxygen therapy after thrombolysis in acute myocardial

infarction has a favorable effect on left ventricular systolic function and the remodeling process.

Publication Types: Clinical Trial Randomized Controlled Trial
PMID: 15459609 [PubMed - indexed for MEDLINE]

49: *Neurochem Int.* 2004 Dec;45(8):1125-32.

Hyperbaric and normobaric reoxygenation of hypoxic rat brain slices--impact on purine nucleotides and cell viability.

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Hyperbaric oxygen treatment has been suggested as able to reduce hypoxia induced neuronal damage. The aim of the study was to compare the impact of different reoxygenation strategies on early metabolical (purine nucleotide content determined by HPLC) and morphological changes (index of cell injury after celestine blue/acid fuchsin staining) of hypoxically damaged rat neocortical brain slices. For this purpose slices (300 microm and 900 microm) were subjected to either 5 or 30 min of hypoxia by gassing the incubation medium with nitrogen. During the following reoxygenation period treatment groups were administered either 100% oxygen (O) or room air (A) at normobaric (1 atm absolute, NB-O; NB-A) or hyperbaric (2.5 atm absolute, HB-O; HB-A) conditions. After 5 min of hypoxia, both HB-O and NB-O led to a complete nucleotide status restoration (ATP/ADP; GTP/GDP) in 300 microm slices. However, reoxygenation after 30 min of hypoxia was less effective, irrespective of the oxygen pressure. Furthermore, administering hyperbaric room air resulted in no significant posthypoxic nucleotide recovery. In 900 microm slices, both control incubation as well as 30 min of hypoxia resulted in significantly lower trinucleotide and higher dinucleotide levels compared to 300 microm slices. While there was no significant difference between HB-O and NB-O on the nucleotide status, morphological evaluation revealed a better recovery of the index of cell injury (profoundly injured/intact cell-ratio) in the HB-O group. Conclusively, the posthypoxic recovery of metabolical characteristics was dependent on the duration of hypoxia and slice thickness, but not on the reoxygenation pressure. A clear restorative effect on purine nucleotides was found only in early-administered HB-O as well as NB-O in contrast to room air treated slices. However, these pressure independent metabolic changes were morphologically accompanied by a significantly improved index of cell injury, indicating a possible neuroprotective role of HB-O in early posthypoxic reoxygenation.

PMID: 15380622 [PubMed - indexed for MEDLINE]

50: *Anadolu Kardiyol Derg.* 2004 Sep;4(3):259.

Comment on: *Anadolu Kardiyol Derg.* 2004 Sep;4(3):256-8.

Cardiac decompression sickness.

Cetinguc M.

Publication Types: Comment Editorial
PMID: 15355831 [PubMed - indexed for MEDLINE]

51: *J Neurosurg.* 2004 Sep;101(3):499-504.

Effects of hyperbaric oxygen therapy on cerebral oxygenation and mitochondrial function following moderate lateral fluid-percussion injury in rats.

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OBJECT: In the current study, the authors examined the effects of hyperbaric O₂ (HBO) following fluid-percussion brain injury and its implications on brain tissue oxygenation (PO₂) and O₂ consumption (VO₂) and mitochondrial function (redox potential). METHODS: Cerebral tissue PO₂ was measured following induction of a lateral fluid-percussion brain injury in rats. Hyperbaric O₂ treatment (100% O₂ at 1.5 ata) significantly increased brain tissue PO₂ in both injured and sham-injured animals. For VO₂ and redox potential experiments, animals were treated using 30% O₂ or HBO therapy for 1 or 4 hours (that is, 4 hours 30% O₂ or 1 hour HBO and 3 hours 100% O₂). Microrespirometer measurements of VO₂ demonstrated significant increases following HBO treatment in both injured and sham-injured animals when compared with animals that underwent 30% O₂ treatment. Mitochondrial redox potential, as measured by Alamar blue fluorescence, demonstrated injury-induced reductions at 1 hour postinjury. These reductions were partially reversed at 4 hours postinjury in animals treated with 30% O₂ and completely reversed at 4 hours postinjury in animals on HBO therapy when compared with animals treated for only 1 hour. CONCLUSIONS: Analysis of data in the current study demonstrates that HBO significantly increases brain tissue PO₂ after injury. Nonetheless, treatment with HBO was insufficient to overcome injury-induced reductions in mitochondrial redox potential at 1 hour postinjury but was able to restore redox potential by 4 hours postinjury. Furthermore, HBO induced an increase in VO₂ in both injured and sham-injured animals. Taken together, these data demonstrate that mitochondrial function is depressed by injury and that the recovery of aerobic metabolic function may be enhanced by treatment with HBO.

PMID: 15352608 [PubMed - indexed for MEDLINE]

52: *Eur J Pediatr Surg.* 2004 Aug;14(4):240-4.

Hyperbaric oxygenation therapy for simple adhesive postoperative intestinal obstruction in children: comparison of the use of a short tube versus a long tube.

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We hypothesized that hyperbaric oxygenation (HBO) combined with a long tube (LT) [HBO + LT] would be more effective than HBO combined with a short tube (ST) [HBO + ST] for simple adhesive postoperative intestinal obstruction (APIO) in children, assuming that there is synergism between HBO and LT. The objective of this study was to determine retrospectively the effect of HBO + LT compared to HBO + ST for simple APIO in children. Seventy-three patients were diagnosed with simple APIO, and 51 of these patients were treated with HBO + LT during 104 HBO sessions, while 22 were treated with HBO + ST during 34 HBO sessions. HBO was performed at a pressure of 2 atmospheres for 60 minutes once daily. The recovery rates after HBO therapy were 87.5 % for the HBO + LT group and 82.4 % for the HBO + ST group ($p = 0.4496$). HBO was performed 5 ± 3 (range 1 to 15) times for the HBO + LT group and 4 ± 2 (range 1 to 8) times for the HBO + ST group ($p = 0.9847$) for ultimate recovery from simple APIO. The recovery rate after HBO therapy of up to 6 sessions was 78.2 % for HBO + LT and 92.1 % for HBO + ST ($p = 0.0360$) among the cases that recovered ultimately. The effect of HBO + LT did not significantly differ from that of HBO + ST, but the insertion of an LT is more intricate and the cost is higher than that of an ST. Therefore, we conclude that the use of an LT is not necessarily required for HBO therapy for simple APIO.

PMID: 15343463 [PubMed - indexed for MEDLINE]

53: J Appl Physiol. 2005 Jan;98(1):144-50. Epub 2004 Aug 20.

Effects of nitrogen and helium on CNS oxygen toxicity in the rat.

Arieli R, Ertracht O, Oster I, Vitenstein A, Adir Y. Israel Naval Medical Institute, Israel Defense Forces Medical Corps, POB 8040, Haifa 31080, Israel. rarieli@netvision.net.il

The contribution of inert gases to the risk of central nervous system (CNS) oxygen toxicity is a matter of controversy. Therefore, diving regulations apply strict rules regarding permissible oxygen pressures (P_{O_2}). We studied the effects of nitrogen and helium (0, 15, 25, 40, 50, and 60%) and different levels of P_{O_2} (507, 557, 608, and 658 kPa) on the latency to the first electrical discharge (FED) in the EEG in rats, with repeated measurements in each animal. Latency as a function of the nitrogen pressure was not homogeneous for each rat. The prolongation of latency observed in some rats at certain nitrogen pressures, mostly in the range 100 to 500 kPa, was superimposed on the general trend for a reduction in latency as nitrogen pressure increased. This pattern was an individual trait. In contrast with nitrogen, no prolongation of latency to CNS oxygen toxicity was observed with helium, where an increase in helium pressure caused a reduction in latency. This bimodal

response and the variation in the response between rats, together with a possible effect of ambient temperature on metabolic rate, may explain the conflicting findings reported in the literature. The difference between the two inert gases may be related to the difference in the narcotic effect of nitrogen. Proof through further research of a correlation between individual sensitivity to nitrogen narcosis and protection by N_2 against CNS oxygen toxicity in rat may lead to a personal O_2 limit in mixed-gas diving based on the diver sensitivity to N_2 narcosis. PMID: 15322063 [PubMed - indexed for MEDLINE]

54: Nurs Times. 2004 Jul 6-12;100(27):50-1.

Nurses who thrive under pressure. Interview by David Crouch.

Gleeson A.

Publication Types: Interview

PMID: 15317265 [PubMed - indexed for MEDLINE]

55: J Orthop Res. 2004 Sep;22(5):1126-34.

Attenuation of apoptosis and enhancement of proteoglycan synthesis in rabbit cartilage defects by hyperbaric oxygen treatment are related to the suppression of nitric oxide production.

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Department of Orthopaedic Surgery and Hyperbaric Oxygen Therapy Center, Chang Gung Memorial Hospital, No. 222, Mai-Chin Road, Keelung, Taiwan. Proinflammatory cytokine, nitric oxide (NO) and localized hypoxia-induced apoptosis and proteoglycan (PG) degradation are thought to be correlated to the degree of cartilage injury. This study evaluated hyperbaric oxygen (HBO)-induced changes in joint cavity oxygen tension, antigenickateran sulfate (KS) content, inducible nitric oxide synthase (iNOS) expression, PG synthesis, and cell apoptosis in full-thickness defects of rabbit cartilage. The HBO group was exposed to 100% oxygen at 2.5 atm for 2 h daily, 5 days per week. Meanwhile, the control group was kept in housing cages with normal air. The joint cavity oxygen tension was determined with an oxygen sensor. Blood serum KS was quantified by competitive indirect enzyme-linked immunosorbent assay (ELISA). After sacrifice, specimen sections were sent for histological and histochemical examination with a standardized scoring system. In situ analysis of iNOS expression and apoptosis detection were performed using immunostaining and TUNEL staining, respectively and quantified by a computerized image analysis system. This study demonstrated that HBO treatment increased joint cavity oxygen tension but decreased blood KS content. Histological and histochemical score results showed that HBO treatment significantly increased the cartilage repair. Moreover, immunostaining and TUNEL staining showed that HBO treatment suppressed the iNOS expression and apoptosis of chondrocytes, respectively. Accordingly, HBO offers a potential treatment method for cartilage injury.

PMID: 15304289 [PubMed - indexed for MEDLINE]

56: J Pediatr Surg. 2004 Aug;39(8):1188-93.

Effects of hyperbaric oxygen therapy on caustic esophageal injury in rats.

Kiyan G, Aktas S, Ozel K, Isbilen E, Kotiloglu E, Dagli TE.

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BACKGROUND/PURPOSE: Caustic esophageal burn is a serious problem in pediatric surgery. Even though many clinical and experimental studies had been performed, the complication rate could not be reduced to a satisfying level. In this study, the authors evaluated the effects of hyperbaric oxygen (HBO) therapy in caustic esophageal burn in rats. **METHODS:** Rats were divided into 4 groups, and caustic burn at the distal esophagus was created by applying 50% NaOH for 3 minutes in all groups. The first and third groups did not receive HBO therapy. HBO therapy was applied to the second group for 2 days and to the fourth group for 28 days. To evaluate the effects of short-term HBO therapy, the first 2 groups were compared for ulceration, inflammation, and submucosal vascular thrombosis after 2 days. The third and fourth groups were compared for the long-term effects of HBO therapy. Rats in these groups were killed after 28 days and compared for the collagen content, weight, and mortality rate. **RESULTS:** In the second group, which received 2 days of HBO therapy, ulcer depth and vascular thrombosis were significantly lower than these in the first group ($P = .022$ and $P = .020$, respectively). The fourth group, which received 4 weeks of HBO therapy, had a significantly reduced mortality rate, weight loss, and collagen score and hydroxyproline level if compared with the third group ($P = .035$; $P = .016$; $P = .028$; and $P = .033$, respectively). **CONCLUSIONS:** These results indicate that HBO therapy is useful in caustic esophageal burn both in short-term and long-term use.

Publication Types: Evaluation Studies

PMID: 15300524 [PubMed - indexed for MEDLINE]

57: Tohoku J Exp Med. 2004 Aug;203(4):253-65.

The effects of hyperbaric oxygen treatment on oxidant and antioxidants levels during liver regeneration in rats.

Ozden TA, Uzun H, Bohloli M, Toklu AS, Paksoy M, Simsek G, Durak H, Issever H, Ipek T.

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The effects of hyperbaric oxygen (HBO) therapy on oxidant/antioxidant metabolism are controversial and its effects on hepatic regeneration are not known. In this study, we investigated a possible beneficial effect of HBO therapy on oxidant and antioxidants levels during liver regeneration. To conduct this study, seventy percent hepatectomy was performed on forty-eight Sprague-Dawley rats and the rats were divided into two equal groups: HBO-treated group and untreated group (non-HBO group). We

determined the levels of malondialdehyde (MDA), an oxidative stress marker, and the levels of antioxidant enzymes/reagents, including glutathione (GSH), superoxide dismutase (SOD) activity, copper (Cu) and zinc (Zn), in the remnant liver samples. We also measured mitotic index (MI) and proliferating cell nuclear antigen (PCNA) levels to assess the degree of liver regeneration. HBO treatment significantly decreased MDA levels, whereas it increased SOD activity, GSH and Zn levels. In contrast, Cu levels were lower in the HBO-treated livers than the levels in the untreated remnant livers. The effect of HBO treatment may be mediated by the suppression of certain enzymes that are responsible for lipid peroxidation. In addition, HBO treatment may induce the production of antioxidant enzymes/reagents by remnant liver tissues. The HBO-treated rats maintained their body weights but the untreated rats lost body weights. HBO treatment also increased MI and PCNA levels, indicating HBO treatment enhances liver regeneration. These results indicate that HBO treatment has beneficial effects on liver regeneration by decreasing MDA and by increasing antioxidant activities. We therefore suggest that HBO therapy may be useful after liver resection.

PMID: 15297730 [PubMed - indexed for MEDLINE]

58: Eur J Cardiothorac Surg. 2004 Aug;26(2):301-5.

Aqueous oxygen: the solution to relief hypoxic pulmonary hypertension.

Corno AF, Boone Y, Mallabiabarrena I, Augstburger M, von Segesser LK.

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OBJECTIVE: To evaluate the effects of hyperbaric oxygen solution on hypoxic pulmonary hypertension.

METHODS: Eleven calves, 2-month-old, 71 ± 6 kg, underwent general anaesthesia, mechanical ventilation and median sternotomy. Catheters for continuous pressure and blood gas measurements were inserted in carotid and femoral arteries, left atrium, right atrium and pulmonary artery (PA), and a flow-probe placed around the PA. After baseline measurements 30 min hypoxic ventilation reduced the mean arterial PO₂ from 285 ± 115 to 46 ± 11 mmHg ($P < 0.0001$). At this point, without changes in hypoxic ventilation (mean arterial PO₂ maintained at 50 ± 5 mmHg), 3 ml/min of hyperbaric aqueous oxygen (AO, oxygen diluted in saline solution) was infused directly into the PA for 30 min, with continuous reading of the monitored parameters. **RESULTS:** Hypoxic ventilation raised significantly ($P < 0.005$) the values of systolic (36 ± 7 vs 22 ± 6 mmHg), diastolic (16 ± 3 vs 9 ± 4 mmHg) and mean (24 ± 4 vs 14 ± 4 mmHg) PA pressure, PA/systemic pressure ratio for systolic (0.47 ± 0.09 vs 0.24 ± 0.06) and mean (0.49 ± 0.13 vs 0.23 ± 0.08) pressures and Pulmonary Vascular Resistance (PVR) (6.89 ± 0.87 vs 2.67 ± 0.38 U), while the Pulmonary Blood Flow (PBF) decreased (2.7 ± 0.4 vs 3.7 ± 0.4

l/min). AO infusion reduced significantly ($P < 0.005$) the values obtained with hypoxic ventilation with systolic (26 ± 6 vs 36 ± 7 mmHg), diastolic (11 ± 4 vs 16 ± 3 mmHg) and mean (16 ± 4 vs 24 ± 4 mmHg) PA pressure, PA/systemic pressure ratio for systolic (0.27 ± 0.07 vs 0.47 ± 0.09) and mean (0.27 ± 0.08 vs 0.49 ± 0.13) pressures and PVR (3.42 ± 0.31 vs 6.89 ± 0.87 U), while the PBF increased (3.6 ± 0.4 vs 2.7 ± 0.4 l/min). CONCLUSIONS: Acute infusion of hyperbaric AO solution into the PA completely reverses the negative effects of acute hypoxia on pulmonary circulation. PMID: 15296887 [PubMed - indexed for MEDLINE]

59: Exp Neurol. 2004 Sep;189(1):150-61.

Transient exposure of rat pups to hyperoxia at normobaric and hyperbaric pressures does not cause retinopathy of prematurity.

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We have shown that hyperoxia reduces brain damage in a rat model of hypoxia-ischemia. The purpose of this study was to examine the possibility of hyperoxia in inducing vision-threatening retinopathy. Two different experiments were conducted in this study. PART 1: seven-day-old rat pups were subjected to unilateral carotid artery ligation followed by 2 h of hypoxia (8% O₂ at 37 degrees C). Pups were treated with 100% oxygen at 1 ATA, 1.5 ATA, and 3.0 ATA for a duration of 1 h. PART 2: Newborn rat pups were exposed to 100% oxygen at 1, 1.5, or 3.0 ATA for 1 h, the same treatment protocol used for brain protection after hypoxia-ischemia. Retinopathy was evaluated by the degree of neovascularization (measuring retinal vascular density), by the structural abnormalities (histology) in the retina, and by the expression of hypoxia-hyperoxia sensitive proteins including hypoxia-inducible factor-1alpha (HIF-1alpha) and vascular endothelial growth factor (VEGF) at 24 h, 1, 2, and 10 weeks after hyperoxia exposure. Hyperoxic treatment at all pressures administered significantly reduced the hypoxia-ischemic-induced reduction in brain weight. Retinal vascular density measurements revealed no signs of neovascularization after hyperoxia exposure. There were also no abnormalities in the structure of the retina and no changes in the protein expression of HIF-1alpha and VEGF following hyperoxia exposure. Exposure to hyperoxia for 1 h at normobaric or hyperbaric pressures did not result in the structural changes or abnormal vascularization that is associated with retinopathy of prematurity, suggesting that hyperoxia is a safe treatment for hypoxic newborn infants. PMID: 15296845 [PubMed - indexed for MEDLINE]

60: Acta Ophthalmol Scand. 2004 Aug;82(4):485-6.

Promising visual improvement of cystoid macular oedema by hyperbaric oxygen therapy.

Jansen EC, Nielsen NV.

Publication Types: Case Reports Letter

PMID: 15291949 [PubMed - indexed for MEDLINE]

61: Acta Ophthalmol Scand. 2004 Aug;82(4):449-53. Ocular refractive changes in patients receiving hyperbaric oxygen administered by oronasal mask or hood.

Evanger K, Haugen OH, Irgens A, Aanderud L, Thorsen E.

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PURPOSE: The aim of this study was to quantify ocular refractive changes after a standard hyperbaric oxygen (HBO) treatment protocol and to characterize the time period of recovery. PATIENTS AND METHODS: Hyperbaric oxygen therapy was given for 90 min daily at a pressure of 240 kPa for 21 days. Oxygen was administered to 20 patients using an oronasal mask and to 12 patients using a hood. Follow-up examinations were carried out 2-4 days after treatment, and thereafter regularly for up to 10 weeks in both groups. Refraction was assessed automatically and by the monocular subjective refraction method. A subgroup of nine of the 20 patients to whom oxygen was administered by an oronasal mask underwent a separate eye examination, which included crystalline lens opacity measurements and LOCS III gradings. RESULTS: In the patients given oxygen by mask, there was a significant myopic shift in the mean spherical equivalent, which was largest 2-4 days after treatment. The shift was -0.55 ± 0.40 D in the right eye and -0.53 ± 0.42 D in the left eye. In the patients given oxygen by hood, the largest shift was observed after 12-16 days, and was -1.06 ± 0.52 D in the right eye and -1.10 ± 0.57 D in the left eye. The refractive changes returned to baseline 6 weeks and 10 weeks after HBO treatment, respectively. No significant changes in crystalline lens transparency were revealed. CONCLUSIONS: The myopic shift after HBO therapy recovers within 10 weeks and may be more pronounced when patients are given oxygen using a hood compared with using an oronasal mask.

PMID: 15291940 [PubMed - indexed for MEDLINE]

62: Crit Care Med. 2004 Aug;32(8):1722-9.

A potential role of hyperbaric oxygen exposure through intestinal nuclear factor-kappaB.

Sakoda M, Ueno S, Kihara K, Arikawa K, Dogomori H, Nuruki K, Takao S, Aikou T.

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OBJECTIVE: Recent studies have demonstrated the therapeutic effectiveness and pharmacologic mechanisms of hyperbaric oxygen therapy (HBOT) in the treatment of a systemic shock state. To elucidate the in vivo role of HBOT during sepsis, we evaluated the effects of HBOT on intestinal mucosal injury and bacterial translocation after lipopolysaccharide challenge. DESIGN:

Experimental study. SETTING: First Department of Surgery and Division of Emergency Care, Kagoshima University School of Medicine, Kagoshima, Japan. SUBJECTS: Male rats were treated with lipopolysaccharide by an intraperitoneal route or with lipopolysaccharide and HBOT. INTERVENTIONS: The survival rate, small intestinal tissue damage, and bacterial translocation in the HBOT-treated group were compared with those in the untreated group. Moreover, plasma tumor necrosis factor- α and nitrite/nitrate concentrations, inducible nitric oxide synthase and myeloperoxidase activities, and nuclear factor- κ B in ileal mucosa were investigated. HBOT was initiated 3 hrs after lipopolysaccharide challenge and administered as 100% oxygen, at 2.53 x 10 kPa (2.5 atm absolute), for 60 mins. MEASUREMENTS AND MAIN RESULTS: When a sublethal dose of lipopolysaccharide (24 mg/kg) was given, the survival rate was much better in the HBOT-treated group (75%) than in the untreated group (33%). HBOT given 3 hrs after lipopolysaccharide injection (10 mg/kg) also lessened the histologic tissue damage of the terminal ileum and the incidence and magnitude of bacterial translocation to mesenteric lymph nodes at 24 hrs after the lipopolysaccharide injection. Moreover, HBOT was able to reduce mucosal inducible nitric oxide synthase and myeloperoxidase activities and plasma nitrite/nitrate concentrations but not serum tumor necrosis factor- α concentrations. Immunohistochemical examination revealed that HBOT specifically modified the mucosal nuclear factor- κ B activation within 4-6 hrs after the injection. CONCLUSIONS: HBOT performed 3 hrs after lipopolysaccharide challenge alleviates intestinal barrier dysfunction and improves survival rates. Herein, we propose one possible mechanism for these beneficial effects: HBOT can modify the nuclear factor- κ B activation in the intestinal mucosa and attenuate the sequential nitric oxide overproduction and myeloperoxidase activation. Consequently, bacterial translocation could be potentially decreased. We believe that the present study should lead to an improved understanding of HBOT's potential role in sepsis.

PMID: 15286550 [PubMed - indexed for MEDLINE]

63: S Afr Med J. 2004 Jul;94(7):498-500.

Pressure chamber explosion--Southern African Underwater and Hyperbaric Medical Association Statement.

van Niekerk GD.

Publication Types: Letter

PMID: 15285439 [PubMed - indexed for MEDLINE]

64: Laryngoscope. 2004 Aug;114(8):1510; author reply 1510-1.

Comment on: Laryngoscope. 2003 Aug;113(8):1356-61.

Inner ear decompression illness.

Parell GJ, Becker GD.

Publication Types: Comment Letter

PMID: 15280736 [PubMed - indexed for MEDLINE]

65: Anesthesiology. 2004 Aug;101(2):539-42.

Air embolism during an aircraft flight in a passenger with a pulmonary cyst: a favorable outcome with hyperbaric therapy.

Closon M, Vivier E, Breynaert C, Duperret S, Branche P, Coulon A, De La Roche E, Delafosse B. Department of Anesthesia and Intensive Care, Hopital de la Croix-Rousse, Lyon, France.

Publication Types: Case Reports

PMID: 15277939 [PubMed - indexed for MEDLINE]

66: Prog Neuropsychopharmacol Biol Psychiatry. 2004 Jul;28(4):667-76.

Effects of the hyperbaric oxygen treatment on the Na⁺,K⁺-ATPase and superoxide dismutase activities in the optic nerves of global cerebral ischemia-exposed rats.

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The effects of hyperbaric oxygen (HBO) treatment on the Na⁺,K⁺-ATPase and superoxide dismutase (SOD) activities were examined in the optic nerves of the rats exposed to global cerebral ischemia. Animals were exposed to global cerebral ischemia of 20-min duration and were either sacrificed or exposed to the first HBO treatment immediately, 0.5, 1, 2, 6, 24, 48, 72 or 168 h after ischemic procedure (for Na⁺,K⁺-ATPase activities measurement) or 2, 24, 48 or 168 h after ischemia (for SOD activities measurement). HBO procedure was repeated for 7 consecutive days. It was found that global cerebral ischemia induced a statistically significant decrease in the Na⁺,K⁺-ATPase activity of the optic nerves, starting from 0.5 to 168 h of reperfusion. Maximal enzymatic inhibition was registered 24 h after the ischemic damage. The decline in the Na⁺,K⁺-ATPase activity was prevented in the animals exposed to HBO treatment within the first 6 h of reperfusion. The results of the presented experiments demonstrated also a statistically significant increase in the SOD activity after 24, 48 and 168 h of reperfusion in the optic nerves of non-HBO-treated ischemic animals as well as in the ischemic animals treated with HBO. Our results indicate that global cerebral ischemia induced a significant alterations in the Na⁺,K⁺-ATPase and SOD activities in the optic nerves during different periods of reperfusion. HBO treatment, started within the first 6 h of reperfusion, prevented ischemia-induced changes in the Na⁺,K⁺-ATPase activity, while the level of the SOD activity in the ischemic animals was not changed after HBO administration.

PMID: 15276692 [PubMed - indexed for MEDLINE]

67: Braz J Med Biol Res. 2004 Aug;37(8):1231-7. Epub 2004 Jul 20.

Effect of hyperbaric oxygenation on the regeneration of the liver after partial hepatectomy in rats.

Kurir TT, Markotic A, Katalinic V, Bozanic D, Cikes V, Zemunik T, Modun D, Rincic J, Boraska V, Bota B, Salamunic I, Radic S.

Department of Pathophysiology, Clinical Hospital Split, Split, Croatia.

The aim of the present study was to assess the influence of hyperbaric oxygenation (HBO) on rat liver regeneration before and after partial hepatectomy. Rats were sacrificed 54 h after 15% hepatectomy, liver and body weights were measured, and serum alanine transaminase (ALT) and aspartate transaminase (AST) activity and albumin levels were determined. The lipid peroxide level, as indicated by malondialdehyde production in the remnant liver was measured, and liver sections were analyzed by light microscopy. Five groups of 10 rats in each group were studied. The preHBO and pre-hyperbaric pressure (preHB) groups were treated before partial hepatectomy with 100% O₂ and 21% O₂, respectively, at 202,650 pascals, daily for 3 days (45 min/day). The control group was not treated before partial hepatectomy and recovered under normal ambient conditions after the procedure. Groups postHBO and postHB were treated after partial hepatectomy with HBO and HB, respectively, three times (45 min/day). The preHBO group presented a significant increase in the initiation of the regeneration process of the liver 54 h postoperatively. The liver/body weight ratio was 0.0618 +/- 0.0084 in the preHBO compared to 0.0517 +/- 0.0016 g/g in the control animals (P = 0.016). In addition, the preHBO group showed significant better liver function (evaluated by the lowest serum ALT and AST activities, P = 0.002 and P = 0.008, respectively) and showed a significant decrease in serum albumin levels compared to control (P < 0.001). Liver lipid peroxide concentration was lowest in the preHBO group (P < 0.001 vs control and postHBO group) and light microscopy revealed that the composition of liver lobules in the preHBO group was the closest to normal histological features. These results suggest that HBO pretreatment was beneficial for rat liver regeneration after partial hepatectomy.

PMID: 15273825 [PubMed - indexed for MEDLINE]

68: *Ann Plast Surg.* 2004 Aug;53(2):141-5.

The effect of hyperbaric oxygen therapy on composite graft survival.

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Auricular composite grafts are a useful reconstructive option, particularly for nasal reconstruction. This study evaluates the effect of hyperbaric oxygen (HBO) therapy on auricular composite graft survival in rabbits. Circular chondrocutaneous composite grafts of 0.5, 1, or 2 cm in diameter were resected from the ears of rabbits. The grafts were sutured back

into position. Half the rabbits in each group received HBO postoperatively, consisting of 90 minutes at 2.4 atm. Rabbits received 7 treatments in 5 days. Control rabbits did not receive HBO. On day 21 the percentage area of graft survival was calculated from gross and histologic examination. Two-centimeter grafts treated with HBO (n = 8) had a mean graft survival rate of 85.8 +/- 15.7% compared with a survival rate of 51.31 +/- 38.5% for the control group (n = 8; P = 0.0478). There was no such benefit in smaller grafts. HBO could prove clinically useful for larger composite grafts.

PMID: 15269583 [PubMed - indexed for MEDLINE]

69: *Cephalalgia.* 2004 Aug;24(8):639-44.

A randomized, double blind study of the prophylactic effect of hyperbaric oxygen therapy on migraine.

Eftedal OS, Lydersen S, Helde G, White L, Brubakk AO, Stovner LJ.

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In a double blind, placebo-controlled study to assess the prophylactic effect of hyperbaric oxygen therapy on migraine, 40 patients were randomly assigned to a treatment group receiving three sessions of hyperbaric oxygen, or a control group receiving three hyperbaric air treatments. The patients were instructed to keep a standardized migraine diary for eight weeks before and after the treatment. Thirty-four patients completed the study. Our primary measure of efficacy was the difference between pre- and post-treatment hours of headache per week. The results show a nonsignificant reduction in hours of headache for the hyperbaric oxygen group compared to the control group. Levels of endothelin-1 in venous blood before and after treatment did not reveal any difference between the hyperbaric oxygen and control groups. We conclude that the tested protocol does not show a significant prophylactic effect on migraine and does not influence the level of endothelin-1 in venous blood.

Publication Types: Clinical Trial Randomized Controlled Trial

PMID: 15265052 [PubMed - indexed for MEDLINE]

70: *J Urol.* 2004 Aug;172(2):679.

Hyperbaric oxygen therapy for cyclophosphamide induced refractory hemorrhagic cystitis in a child.

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Publication Types: Case Reports

PMID: 15247759 [PubMed - indexed for MEDLINE]

71: *Arch Phys Med Rehabil.* 2004 Jul;85(7):1198-204.

Comment in: *Arch Phys Med Rehabil.* 2004 Oct;85(10):1732.

Hyperbaric oxygen therapy for traumatic brain injury: a systematic review of the evidence.

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OBJECTIVE: To identify the benefits and harms of hyperbaric oxygen therapy (HBOT) to treat traumatic brain injury (TBI). **DATA SOURCES:** MEDLINE, EMBASE, the Cochrane Library, HealthSTAR, CINAHL, MANTIS, professional society databases, and reference lists. Databases were searched from inception through December 2003. **STUDY SELECTION:** We included English-language studies of patients with TBI given HBOT and evaluating functional health outcomes. **DATA EXTRACTION:** Data were abstracted by 1 reviewer and checked by a second. Study quality was rated as good, fair, or poor. **DATA SYNTHESIS:** Two fair-quality randomized controlled trials of patients with severe brain injury reported conflicting results. One found no difference in mortality (48% HBOT vs 55% control) or morbidity at 1 year. In young patients with brainstem contusion, significantly more regained consciousness at 1 month with HBOT (67%) than control (11%) ($P < .03$). The other found a significant decrease in mortality in the HBOT group at 1 year (17%) compared with controls (31%) ($P = .037$). This decrease in mortality was accompanied by an increase in proportion of patients with severe disability. Patients with intracranial pressure (ICP) greater than 20 mmHg or a Glasgow Coma Scale score of 4 to 6 had significantly lower mortality at 1 year than controls. Five observational studies did not provide better evidence of effectiveness or adverse events. Two indicated a potential for initially reducing elevated ICP in some patients. However, rebound elevations higher than pretreatment levels occurred in some patients. Adverse events, including seizures, pulmonary symptoms, and neurologic deterioration, were reported; however, no study systematically assessed adverse events, and none reported adverse events in control groups. **CONCLUSIONS:** The evidence for HBOT for TBI is insufficient to prove effectiveness or ineffectiveness, and more high-quality studies are needed. The evidence indicates that there is a small chance of a mortality benefit, which may depend on subgroup selection. The effect on functional status and the incidence and clinical significance of adverse effects are unclear.

Publication Types: Review

PMID: 15241774 [PubMed - indexed for MEDLINE]

72: Otol Neurotol. 2004 Jul;25(4):553-8.

Effects of hyperbaric therapy on function and morphology of Guinea pig cochlea with endolymphatic hydrops.

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OBJECTIVE: The objective of this study was to investigate the effect on experimental endolymphatic hydrops in guinea pigs after hyperbaric therapy. **BACKGROUND:** The histopathologic character of Meniere's disease is the presence of endolymphatic hydrops. Endolymphatic hypertension could be one of the factors resulting from endolymphatic hydrops. Some treatments of Meniere's disease are aimed toward preventing the endolymphatic hypertension. Exposure to pressure change has risen in recent years. **METHODS:** Thirty-two guinea pigs were operated on the right ears to induce endolymphatic hydrops by obliterating the endolymphatic sac through an extradural posterior cranial fossa approach. After 5 weeks' survival, 12 guinea pigs were put into a chamber with an absolute atmospheric pressure of 2.2 for 3 weeks (90 minutes once a day 5 times a week). We observed the morphologic and functional changes in guinea pig cochleae of the pressure group, 4-week hydrops group ($n = 10$), 8-week hydrops group ($n = 10$), and the normal group ($n = 10$). We measured the hearing threshold of the auditory brainstem response, the 70-dB SPL action potential (AP) latency, the ratio of 70-dB SPL summing potential magnitude to action potential magnitude ($-SP/AP$) of the electrocochleogram, and the maximum scala media area (SMA) ratio, respectively. **RESULTS:** The average 70-dB SPL- SP/AP magnitude of right ears (0.29 ± 0.09) and the average maximum SMA ratio (2.23 ± 0.20) in the pressure group were significantly less than that in the 8-week hydrops group (0.69 ± 0.15 and 4.04 ± 0.52 , respectively) with the same survival time ($p < 0.05$). The results in the pressure group were almost as similar as that in the 4-week hydrops group (0.29 ± 0.13 and 2.22 ± 0.20 , respectively) ($p > 0.05$). The average hearing threshold of ABR of right ears in the pressure group (36.67 ± 14.30 -dB SPL) was lower than that of the 8-week hydrops group (44 ± 14.30 -dB SPL), but the difference was insignificant ($p > 0.05$). The average 70-dB SPL AP latency of right ears in the pressure group was not significantly different from those of the 8-week hydrops group, the 4-week hydrops group, or the normal group ($p > 0.05$). **CONCLUSIONS:** Our findings suggest hyperbaric therapy can significantly suppress the development of endolymphatic hydrops and improve cochlear function in guinea pigs. This study provided strong evidence for the development of pressure treatment of Meniere's disease without destroying the inner ear. PMID: 15241235 [PubMed - indexed for MEDLINE]

73: Radiother Oncol. 2004 Jul;72(1):1-13.

Hyperbaric oxygen therapy in the treatment of radio-induced lesions in normal tissues: a literature review.

Pasquier D, Hoelscher T, Schmutz J, Dische S, Mathieu D, Baumann M, Lartigau E.

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Late complications are one of the major factors limiting radiotherapy treatment, and their treatment is not codified. Hyperbaric oxygen (HBO) has been

used in combination with radiotherapy for over half a century, either to maximise its effectiveness or in an attempt to treat late complications. In this latter case, retrospective trials and case reports are prevailing in literature. This prompted European Society for Therapeutic Radiotherapy and Oncology and European Committee for Hyperbaric Medicine to organise a consensus conference in October 2001, dealing with the HBO indications on radiotherapy for the treatment and prevention of late complications. This updated literature review is part of the documents the jury based its opinion on. A systematic search was done on literature from 1960 to 2004, by only taking into account the articles that appeared in peer review journals. Hyperbaric oxygen treatment involving complications to the head and neck, pelvis and nervous system, and the prevention of complications after surgery in irradiated tissues have been studied. Despite the small number of controlled trials, it may be indicated for the treatment of mandibular osteoradionecrosis in combination with surgery, haemorrhagic cystitis resistant to conventional treatments and the prevention of osteoradionecrosis after dental extraction, whose level of evidence seems to be the most significant though randomised trials are still necessary. The other treatment methods are also outlined for each location.

Publication Types: Review

PMID: 15236869 [PubMed - indexed for MEDLINE]

74: Am J Physiol Heart Circ Physiol. 2004 Nov;287(5):H2234-40. Epub 2004 Jun 24.

Hyperbaric oxygen solution infused into the anterior interventricular vein at reperfusion reduces infarct size in swine.

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This study was designed to test the hypothesis that raising myocardial O₂ via diffusion of a hyperbaric oxygen solution (AO) administered through the anterior interventricular vein (AIV) will reduce infarct size by reducing reperfusion injury associated with reduced neutrophil activation. In three pilot open-chest swine experiments, myocardial tissue Po₂ was monitored using an oxygen probe during coronary occlusion (Occl) and reperfusion (Rep). One control experiment had no AIV infusion; a second control received arterial blood drawn from the femoral artery infused into the AIV during Rep. In a third open-chest experiment, AO mixed with arterial blood was infused through the AIV at Rep. In controls, tissue Po₂ in the risk region (RR) rose early in Rep and then fell to Occl levels, whereas in AO-treated animals, myocardial Po₂ remained above baseline. The following three groups of five swine then underwent 60 min of left anterior descending coronary artery Occl and Rep: 1) arterial blood

infused at Rep as controls (Con), 2) AO infused beginning 30 min after Rep (AO 30 min), and 3) AO infused immediately at Rep (AO 0 min). There were no differences among the three groups in hemodynamics or myocardial blood flow during baseline (BL) or Occl or in RR size. However, endocardial blood flow was significantly higher in RR during Rep in AO 0 min vs. control and AO 30 min (P=0.01). Both infarct size (IS) as %heart and IS as %RR were lower in AO 0 min compared with Con and AO 30 min (P <0.01 for both), and myeloperoxidase values were lower for epicardial (P <0.001), midmyocardial (P=0.03), and endocardial (P <0.001) layers in AO 0 min. AO infused into the AIV immediately at Rep diffuses into the RR and reduces IS by reducing Rep injury associated with neutrophil activation.

PMID: 15217796 [PubMed - indexed for MEDLINE]

75: Am Heart J. 2004 Jul;148(1):e1.

Hyperbaric oxygen treatment does not affect left ventricular chamber stiffness after myocardial infarction treated with thrombolysis.

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BACKGROUND: It has been shown that transient increase in left ventricular stiffness, assessed by Doppler-derived early filling deceleration time, occurs during the first 24 to 48 hours after myocardial infarction but returns to normal within several days. It has been reported that hyperbaric oxygen treatment has a favorable effect on left ventricular systolic function in patients with acute myocardial infarction treated with thrombolysis. However, there are no data on the effects of hyperbaric oxygen on diastolic function after myocardial infarction. **METHODS:** To assess acute and short-term effects of hyperbaric oxygen on left ventricular chamber stiffness, we studied 74 consecutive patients with first acute myocardial infarction who were randomly assigned to treatment with hyperbaric oxygen combined with streptokinase or streptokinase alone. After thrombolysis, patients in the hyperbaric oxygen group received 100% oxygen at 2 atm for 60 minutes in a hyperbaric chamber. All patients underwent 2-dimensional and Doppler echocardiography 1 (after thrombolysis), 2, 3, 7, 21, and 42 days after myocardial infarction. **RESULTS:** Patient characteristics, including age, sex, risk factors, adjunctive postinfarction therapy, infarct location, and baseline left ventricular volumes and ejection fraction, were similar between groups (P >.05 for all). For both groups, deceleration time decreased nonsignificantly from day 1 to day 3 and increased on day 7 (P <.001, for both groups), increasing nonsignificantly subsequently. The E/A ratio increased in the entire study group throughout the time of study (P <.001, for both groups). The pattern of changes of deceleration time was similar in

both groups ($P > .05$ by analysis of variance), as was in subgroups determined by early reperfusion success. **CONCLUSIONS:** These data in a small clinical trial do not support a benefit of hyperbaric oxygen on left ventricular diastolic filling in patients with acute myocardial infarction treated with thrombolysis.

Publication Types: Clinical Trial Randomized Controlled Trial

PMID: 15215810 [PubMed - indexed for MEDLINE]

76: QJM. 2004 Jul;97(7):385-95.

Hyperbaric oxygen: its uses, mechanisms of action and outcomes.

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Publication Types: Review Review, Tutorial

PMID: 15208426 [PubMed - indexed for MEDLINE]

77: Int J Pediatr Otorhinolaryngol. 2004 Jul;68(7):947-53.

The negative effect of hyperbaric oxygen therapy at the acute phase of electrochemical esophageal burn induced by button battery ingestion.

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OBJECTIVE: Ingestion of button battery, if lodges in esophagus, causes mucosal destruction in esophagus and may damage surrounding tissues due to electrochemical reactions which may lead to esophagus perforation, tracheoesophageal fistula and other serious problems. We designed an experimental study to test the effect of hyperbaric oxygen therapy on battery induced electrochemical tissue damage in the esophagus of a rabbit model and possible change with duration of contact time. **METHODS:** Button batteries were inserted in esophagus of 40 rabbits which were divided into four groups. Groups 1 and 2 had 15 min of duration of contact time of battery in esophagus, while Groups 3 and 4 had 30 min. Groups 1 and 3 had hyperbaric oxygen therapy for 3 days; Groups 2 and 4 did not. At the end of 3rd day all animals were sacrificed and samples were taken from the esophagus for determination of malondialdehyde levels and for histopathological examination to compare: mucosal destruction, muscular layer involvement, perforation and tracheal involvement between groups. **RESULTS:** Malondialdehyde levels, mucosal destruction, muscular layer involvement, perforation and tracheal involvement were significantly higher in groups which had 30 min of contact time compared to groups which had 15 min. The same assessments were significantly higher in Group 1 (15 min of contact time with hyperbaric oxygen therapy) compared to Group 2 (15 min of contact time without hyperbaric oxygen therapy). However, the difference between Group 3 (30 min of

contact time with hyperbaric oxygen therapy) and Group 4 (30 min of contact time, no hyperbaric oxygen therapy) was not significant. **CONCLUSION:** Our study demonstrated that if contact time is 15 min HBO had an additive adverse effect to electrochemically burned esophagus by increasing free radicals and eventually tissue damage. However, if the contact time is 30 min its adverse effect is shielded by huge electrochemical destruction due to long contact time.

Publication Types: Evaluation Studies

PMID: 15183587 [PubMed - indexed for MEDLINE]

78: Eur Urol. 2004 Jul;46(1):108-13.

Hyperbaric oxygen for the treatment of interstitial cystitis: long-term results of a prospective pilot study. van Ophoven A, Rossbach G, Oberpenning F, Hertle L.

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OBJECTIVE: We conducted a prospective pilot study to assess the safety and efficacy of hyperbaric oxygen (HBO) for the treatment of interstitial cystitis (IC). **METHODS:** Six patients underwent 30 sessions of 100% oxygen inhalation in a hyperbaric chamber and were followed up over 15 months. The measures of efficacy were changes in pain and urgency (visual analog scales), alteration in the patient's assessment of overall change in his well-being (Patient Global Assessment Form), and changes in frequency and functional bladder capacity (48-hours voiding log). Evaluation of symptom severity regarding pain and voiding problems was done using the O'Leary-Sant index. **RESULTS:** Four patients rated the therapeutic result as either excellent or good and assessed their well-being after HBO treatment as improved. Two patients showed only short-term amelioration of some of their symptoms. At 12 months follow-up the baseline functional bladder capacity increased from 37-161 ml (range) to 160-200 ml in the responder group. The 24-hour voiding frequency decreased from 15-27 to 6-11 voids per day, a pain scale improvement from 20-97 mm at baseline to 3-30 mm at 12 months follow-up and an urgency scale improvement from 53-92 mm to 3-40 mm, respectively was observed at 12 month follow-up. The symptom and pain index score decreased from 23-35 at baseline to 3-17 at 12 months follow-up. **CONCLUSION:** HBO appears to be effective to treat IC patients. Treatment was well tolerated and resulted in a sustained decrease of pelvic pain and urgency, improvement of voiding patterns and increase of functional bladder capacity for at least 12 months. Copyright 2004 Elsevier B.V.

PMID: 15183555 [PubMed - indexed for MEDLINE]

79: Cancer Lett. 2004 Jul 8;210(1):35-40.

Hyperbaric oxygen alone or combined with 5-FU attenuates growth of DMBA-induced rat mammary tumors.

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We tested the hypothesis that hyperbaric oxygen (HBO) alone and with chemotherapy (5-FU) attenuates tumor growth of DMBA-induced tumors in rats. Six series were performed: (1) Controls (air and vehicle 0.9% NaCl i.p.), (2) 5-FU (0.2 mg/kg i.p.), (3) HBO (2 bar for 90 min and vehicle), (4) HBO and 5-FU, (5) HBO (11 days) and air (next 12 days), (6) HBO (23 days). All treatments were applied on days 1, 4, 7, 10 (Series 1-4), as well as on days 14, 17 and 23 (Series 5-6). Tumor diameter increased by 76.7 and 41.2% in untreated controls and in the 5-FU group, respectively, after 10 days. Tumor size fell by 17-24.2% in the HBO groups and by 35.5% when combined with 5-FU ($P < 0.05$ compared to HBO). HBO treatment reduced the total number of blood vessels in the tumors. After completion of HBO treatment tumor size increased, but statistically insignificant, during the next 12 days.

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80: HNO. 2004 Jul;52(7):648-50.

Comment on: HNO. 2004 Jan;52(1):63-6.

[Hyperbaric oxygenation therapy following one-sided idiopathic deafness]

[Article in German]

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Publication Types: Comment Letter

PMID: 15150651 [PubMed - indexed for MEDLINE]

81: Eur Arch Otorhinolaryngol. 2004 Aug;261(7):393-6. Epub 2003 Oct 29.

Comment in: Eur Arch Otorhinolaryngol. 2005 Feb;262(2):163-4.

Should hyperbaric oxygen be added to treatment in idiopathic sudden sensorineural hearing loss?

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At present, there is still no agreement about the therapy of idiopathic sudden sensorineural hearing loss (ISSHL). Hyperbaric oxygen (HBO) is used in the therapy of ISSHL to increase the partial oxygen pressure and the oxygen concentration in the inner ear and also to improve the blood profile and the microcirculation. In our prospective randomized study, we aimed to investigate the therapeutic effects of HBO therapy in the 1st 2 weeks of the onset of ISSHL. Fifty-one hospitalized patients with confirmed ISSHL who had received therapy were grouped randomly into two groups. Twenty-one patients (group I) received steroids, plasma expander dextrans (rheomacrodex), diazepam, pentoxiphylline and salt restriction, and 30 patients (group II) received the same basic treatment with the addition of HBO therapy. Audiological assessments of the

patients were performed before and after the treatment. The hearing gains at frequencies of 250, 500, 1,000, 2,000 and 4,000 Hz were calculated separately. The level of hearing loss at the five frequencies was assessed in three groups at the first visit: equal or below 60 dB, between 61-80 dB and equal or above 81 dB. The average of the mean hearing gains at the five frequencies of the patients according to the age groups in group II was compared. The mean hearing gains at the five frequencies were compared between the two groups, and statistically significant improvement was detected in all the frequencies except at 2,000 Hz in group II. The mean hearing gains in group II were found to be significantly high in patients with initial hearing levels up to 60 dB in comparison to patients with initial hearing levels below 60 dB. When age groups and mean hearing gains were compared, there was no statistically significant difference in group I. In group II, the mean hearing gains were 39.1 \pm 18.3 dB in patients younger than 50 years and 22.7 \pm 11.3 dB in patients older than 50 years ($P=0.044$). In conclusion, the addition of HBO therapy to conventional treatment modalities significantly improves the outcome of ISSHL, especially at the frequencies of 250, 500, 1,000 and 4,000 Hz and in hearing loss of above 61 dB. Furthermore, HBO therapy was found to be more effective in patients younger than 50 years.

Publication Types: Clinical Trial Randomized Controlled Trial

PMID: 14586625 [PubMed - indexed for MEDLINE]