

35th EUBS Annual Scientific Meeting & BHA Annual Meeting 2009

Aberdeen 25-28 August 2009

Preliminary Pre-meeting Programme

Tuesday 25 August

15.30 – 17:30	National Hyperbaric Centre	Tour of hyperbaric facilities
19:30 – 21:00	Maritime Museum, Shiprow	Civic reception hosted by the Lord Provost of Aberdeen

26-28 August 2009

Preliminary Scientific Programme

Venue – Kings College Conference Centre

Wednesday 26 August

08:00	Registration
09:00	Opening Ceremony
Session 1	Health Technology Assessment and Clinical Trials
09:10	Use of hyperbaric oxygen therapy: a rapid assessment in Belgium Mattias Neyt, Belgian Health Care Knowledge Centre.
09:30	The clinical and cost effectiveness of hyperbaric oxygen therapy: The Quality Improvement Scotland HTA review. Karen Macpherson, NHS Quality Improvement Scotland.
09:50	Hyperbaric oxygen therapy indication diabetic foot syndrome – HTA report of federal joint committee for German social health care system. W. Welslau, U. van Laak.
10:05	Health technology assessment – a hyperbaric oxygen provider’s point of view. P. Germonpré, Centre for Hyperbaric Oxygen Therapy, Military Hospital Brussels.
10:20	HOPON: Hyperbaric oxygen to prevent osteoradionecrosis. Richard Shaw
10:35	Coffee

- Session 2 Carbon Monoxide Poisoning**
- 11:15 Carbon monoxide poisoning.
Jordi Desola, CRIS-UTH, Barcelona
- 11:45 Silent killer in Poland – Carbon monoxide poisoned patients treated by hyperbaric therapy in Poland 01/01/2003 – 31/05/2009.
Jacek Piechocki, Michal Patyk, Janusz Sokolowski.
- 12:00 Carbon monoxide intoxication. The treatment in Denmark and a case story.
Erik C. Jansen, Henrik Rottensten, Michal Bering Sifakis.
- 12:15 Bilateral sensorineural hearing loss after acute carbon monoxide poisoning: a case report.
L. Fischer, M. Lechner, N. Patel, M. Ignatescu
- 12:30 **Lunch**
- Session 3 Basic Research in Hyperbaric Medicine - I**
- 13:45 Evaluation of the effects of hyperbaric oxygen on radiated fibroblast cultures.
G. Ozturk, S. Solakoglu, Y. Dizdar.
- 14:00 Hyperbaric oxygen reduces endothelial adhesion molecule expression and neutrophil recruitment under flow in an in vitro chronic wound model.
A. Kendall, J.L. Whatmore, P.G. Winyard, G. Smerdon, P. Eggleton.
- 14:15 Hepatocytes glutamylsynthetase response to hyperbaric oxygenation depends on cell state at the moment of oxygenation.
P.N. Savilov, J.V. Shepeleva.
- 14:30 Effect of hyperbaric oxygen therapy on tense repair of the peripheral nerves.
B. Oroglu, T. Turker, S. Atkas, M. Alp, S. Karamursel.
- 14:45 Preconditioning to HBO may provide protection against CNS oxygen toxicity in the rat: beating the “devil’s triangle”.
Doron Kotler, Ayala Hochman, Mirit Eynan, Yehuda Arieli.
- 15:00 **Tea**
- Session 4 Clinical Hyperbaric Medicine**
- 15:30 Noninvasive evaluation of systemic hemodynamic and microcirculatory response to HBO in mechanically ventilated intensive-care patients.
S. Ledoux-Houcke, B. Ratzenhofer-Komenda, J. Salleron, R. Favory, J.L. Juille, D. Mathieu.
- 15:45 Hyperbaric Oxygen therapy used to treat chronic radiation proctitis. Maide Cimsit, Abdullah Arslan, Yavuz Dizdar, Selva Mert.
- 16:00 The normobaric oxygen paradox (NOP): a safe alternative to post-operative transfusions after hip surgery?
P. Lafere, T. Schubert, E. Munting, P. Germonpré, C. Balestra.
- 16:15 Draft EN on hyperbaric chambers: specific requirements for fire extinguishing systems.
Christian Fabricius.
- 16:30 **Session ends**
- 16:45-17:00 Buses leave for Distillery Tour, Lochter Rural Activities and spit-roast meal at Lochter.

Thursday 27 August

08:00 Registration

The Frederick Love Keays Symposium

Session 5 History and bubbles

09:00 Frederick Love Keays: the genesis of modern hyperbaric occupational medicine.
John A S Ross.

09:15 The Haldane effect.
M.A. Lang, A.O.Brubakk.

09:30 100 years ago in Hamburg: the genesis of modern hyperbaric occupational medicine.
K.-P. Faesecke

09:45 Preventing the “Bends” in Deep Hyperbaric Tunneling.
K.-P. Faesecke

10:00 Imaging Bubbles.
Andreas Møllerløgken, Norwegian University of Science and Technology.

10:30 **Coffee**

Session 6 Basic Research in Hyperbaric Medicine II

11:00 Secondary effects of bubbles.
Richard Moon, Duke University

11:30 Protective effects of clopidogrel on decompression illness sickness incidence and severity in a rat model; a pharmacological intervention against bubble induced platelet aggregation.
Jean-Michel Pontier, Nicolas Vallée, Olivier Simon.

11:45 Simulated diving after heat stress poentiated the induction of heat shock protein 70 and elevates glutathione in human endothelial cells. R Djurhuus, V Nossun, N Lundsett, W Hovin, AM Svardal, M Bjordal Havnes, L Fismen, A Hjelde and AO Brubakk.

12:00 Simulation of post dive decompression bubbles at high altitude.
S.M. Egi, T Ozyigit.

12:15 Pressure modulation of Ca²⁺ channels activity may elucidate HPNS mechanisms.
Ben Aviner, M. Mor, Y. Grossman.

12:30 **Lunch**

Session 7**Diving medicine I**

- 13:30 Central nervous system damage and repair: spinal cord decompression illness.
Dalton Dietrich The Miami Project to Cure Paralysis, University of Miami.
- 14:00 Spinal injury rehabilitation
Alan Maclean, The Queen Elizabeth National Spinal Injuries Unit
- 14:15 Association rules of dci patient clusters and reliability of clustering analysis.
Baris Aksoy, Vincent Labatut, Murat Egi, Tamer Ozyigit, Petar Denoble, Costantino Balestra, Richard Vann, Alessandro Marroni.
- 14:30 The experience and near accidents among 110 Danish diving instructors, - a questionnaire survey.
Erik C. Jansen, , Johnny Frederiksen, Michael Bering Sifakis.
- 14:45 Cross sectional study comparing demographics, health and dive practices of recreational rebreather divers compared to open circuit technical divers and recreational SCUBA divers
S Johnson, V. Allgar, C. Davey.

15:00

Tea**Session 8****Diving Medicine II**

- 15:30 Long term effects of diving.
John A S Ross.
- 16:00 Effect of long-term inactivity, short term rest, body position and leg movements on presence of venous gas emboli after decompression from hyperbaric pressure.
M. Gennser, S.L. Blogg, O. Eiken1 and I.B. Mekjavic.
- 16:15 Underwater versus surface oxygen breathing on post-dive bubble formation in scuba divers.
J-E Blatteau, J-M Pontier.
- 16:30 Biphasic spleen contraction during apnea in divers suggests chemoreceptor input.
Angelica Lodin, Matt X. Richardson, Erika Schagatay.
- 16:45 Evaluation of Critical Flicker Fusion Frequency and perceived fatigue in divers after air and nitrox diving.
W. Hemelryck, N. Donda, P. Lafère, S. Theunissen, A. Taher, A. Sakr, P. Germonpré, C. Balestra.
- 17:00 **Session ends**

19:00 for 19:30

Elphinstone Hall

Dinner and Scottish Ceilidh

Friday 28 August

Session 9 Diving Equipment and Techniques

- 09:00 Diving for handicapped people in the wider Adriatic region.
Branko Ravnak; Blanka Meznar; Ivica Cukusic.
- 09:15 Soda lime packing and CNS oxygen toxicity in closed-circuit oxygen rebreathers.
R. Arieli
- 09:30 Pulmonary Simulator for Work of Breathing Assessment.
Arne Sieber, Wielaw Grzesik, Milena Stoyanova, Matthias Wagner, Remo Bedini
- 09:45 Experimental platform for studies on VPM and Bühlmann decompression algorithms
B. Kuch, R. Bedini, G. Buttazzo, A. Sieber
- 10:00 **Coffee**

Poster Sessions

Venue – James Mackay Hall

Posters may be set up at numbered positions at any time during the meeting.

Session 10 Diving Medicine and Physiology – Posters

Posters will be manned 10:30 – 12:30

- P1 How to predict an unfavourable outcome of a spinal cord decompression sickness in a scuba diver
O. Simon, B. Gamain, A. Kauert, F. Forneris, JM Lapoussière
- P2 The experience and organization of the treatment of diving accidents in Tuscany
G.De Jaco, F.Ghelardoni, P.Tanasi, L. De Fina, G.Sbrana, S.Falini, M.Brauzzi
- P3 Deep dives in different countries
K Segadal
- P4 Guideline "Diving Accident" by German Diving and Hyperbaric Medicine Society
Peter H.J. Müller
- P5 Measurement of OH⁻-radical stress due to the combination of hyperoxia and physical exercise in closed-circuit diving
W. Kähler, D. Weyer, A. Koch
- P6 Implementation of a dive computer using mobile phones: the divephone
SM Egi, AT Naskali, M Memisoglu, U Aksu, E.D. Kunt, S. Satir, S.Basatli
- P7 Exhaled nitric oxide (NO_{exp}) measurements as a noninvasive marker of pulmonary oxygen toxicity susceptibility in humans
D. M. Fothergill and J. Gertner
- P8 Exhaled nitric oxide levels at 1500 metres in an altitude simulator

- Smith G, Turner SW, Devereux GS, Ross JAS, Watt SJ
- P9 Alteration of the alveolocapillary membrane after a dive with a PO₂ of 130 kPa.
P.J.A.M. van Ooij, A. Houtkooper, R.A. van Hulst
- P10 Cross-sectional study of pulmonary function in fighter pilots compared to non-fighter pilots and divers.
S Suzuki, M Sakai, Y Takaai, M Fujita, S Watanabe, M Uenoyama
- P11 S100B as a biomarker for neurological DCS
MB. Havnes, SA. Dybos, M. Kausberg og AO. Brubakk
- P12 Spleen contraction in accessory spleens during apnea in divers
Angelica Lodin, Matt X. Richardson, Erika Schagatay
- P13 The effect of pre-dive exercise mode on post-decompression venous gas emboli.
KM Jurd, JC Thacker, GAM Loveman, FM Seddon.
- P14 Qi-Diver Project: Acupuncture and Traditional Chinese Medicine (TCM) in Freediving Performance Enhancement
V. Zanon, G. Bosco, M. Schiavon, G. Garetto, S. Checcacci-Carboni, and EM. Camporesi
- P15 Underwater Navigation and Communication: A Novel GPS/GSM Diving Computer
B. Kuch, B.Koss, G. Buttazzo, A. Sieber
- P16 Rebreather diving safety: Alternative Control Device
Bernhard Koss, Antonio L'Abbate, Paolo Dario and Arne Sieber
- P17 Electronically Controlled Closed Circuit Rebreather for Recreational Purposes
Arne Sieber and Kurt Sjöblom

Session 11 Hyperbaric Oxygen Therapy Posters

Posters will be manned 10:30 – 12:30

- P18 Identifying eligible patients for inclusion in clinical trials: The HOLLT experience.
R. A. McGinnes, C.M. Venturoni, I.L. Millar
- P19 Hyperbaric oxygen therapy for the treatment of interstitial cystitis - case report
Florian Edsperger, Richard Leberle, Urs Braumandl
- P20 Randomised double-blind controlled phase III trial of hyperbaric oxygen therapy in patients suffering long-term adverse effects of radiotherapy for pelvic cancer
R. Ahern, J. Andreyev, P Bryson, M. Glover, M Ignatescu, G Laden, S. Martin, G. Smerdon, J. Yarnold
- P22 Hyperbaric Oxygen in Pediatric Oncology
Hajek, M., Cahova, P., Sterba, J., Ruzicka, J., Zonca, P
- P23 Martorell's Hypertensive Ulcer: Evaluation with Transcutaneous Oxygen Perfusion Measurements of Treatment with Hyperbaric Oxygen and Blood Pressure Control.
Lafère P, Vanhoutte D, Caers D, Germonpré P

- P24 Comparisons Between United Kingdom and European Protocols Used in the Treatment of Osteoradionecrosis with Hyperbaric Oxygen Therapy
J Dhanda, NJ Opie, S Parmar, E Beshara, L Machon, U Durrani
- P25 Changes in microbial community associated with chronic diabetic foot ulcers over the course of hyperbaric oxygen treatment
L. Glew, A. Bishop, G. Bruce, P. English, J. Grieg, A. J. Moody, K. Tait, P. Bryson and G. Smerdon
- P26 Hyperbaric Oxygen Therapy Used to Treat Late Soft Tissue Radiation Damage: A Case Report
Selva Mert, Abdullah Arslan, Maide Cimsit
- P27 Critical Incidents in a Level 1 Hyperbaric Unit:
Dr P. Bothma and Dr A Brodbeck
- P28 Time course of autonomic nervous control alteration induced by 100% oxygen breathing in healthy men.
Gole Y., Gargne O., Coulange M., Bessereau J., Regnard J., Jammes Y., Boussuges A.
- P29 Pressure regulator for in-chamber blood pressure measurements.
Erik C. Jansen, Michael Bering Sifakis.
- P30 A device for hyperbaric ventilation of the tracheostoma patient
Ole Hyldegaard, Erik C. Jansen
- 12:00-13: 00 **Buffet lunch available**
- 13:00 EUBS Annual General Assembly and Awards
Presentation of EUBS 2010
Closing Ceremony

Preliminary Committee Programme

Tuesday 25 August

- 10:30 start Subsea 7, Westhill, Diving Medical Advisory Group
- 15:00 – 18:00 National Hyperbaric Centre, European Committee for Hyperbaric Medicine

Wednesday 26 August

- 11:00 – 14:00 Linklater Rooms EDTC medical subcommittee

Thursday 27 August

Friday 28 August

- 14:30 – 16:00 Linklater Rooms British Hyperbaric Association annual general meeting

HYPERBARIC OXYGEN THERAPY INDICATION DIABETIC FOOT SYNDROME – HTA REPORT OF FEDERAL JOINT COMMITTEE FOR GERMAN SOCIAL HEALTH CARE SYSTEM

W. Wetslau^{1,2}, U. van Laak^{1,2}

¹ German Society for Diving and Hyperbaric Medicine, www.gtuem.org

² Member of ‘Working Group HBO Therapy’, Federal Joint Committee, www.g-ba.de

AIMS The HTA report ‘Hyperbaric Oxygen Therapy - Indication Diabetic Foot Syndrome’ was initiated by German social health care system to clear whether HBO should be a paid therapy for inpatients insured by social health care system.

INTRODUCTION In 2002 German social health care system installed a new committee which has to decide about future cost coverage for diagnostic and therapeutic measures for insured inpatients. The committee is equally seated with representatives of social health care insurance companies and representatives of hospitals. Until today decisions were taken also about HBO therapy for 12 different indications. All single decisions are based on separate HTA reports. One of these reports concerns future cost coverage for HBO therapy for inpatients with diabetic foot syndrome (DFS).

METHODS Development of this HTA report contained structured literature search (15.05.2006), clear inclusion criteria and documented decisions about literature exclusion, and evaluation of included publications according to internationally accepted standards of evidence based medicine (EBM). The report also contains an overview about actual situation of DFS, patho-physiological basis of HBO in DFS, and detailed argumentation of decision.

RESULTS Literature search identified 15 information syntheses (HTA reports and systematic reports) meeting first inclusion criteria. Further evaluation showed major weaknesses in 14 of these reports. Therefore primary studies were searched. 4 randomized controlled trials (RCT), 2 other prospective controlled studies, and 3 retrospective controlled studies were identified and met inclusion criteria. Further evaluation showed that in one of these studies ‘major amputation’ was no endpoint, and that a second study had overlapping of included patients with a third study. The remaining studies were included in a quantified meta-analysis (HBO group: n=145, controls n=152). For the endpoint ‘major amputation’ all studies showed consistent results (total of major amputations: HBO group: n=17, controls: n=50. Relative risk of major amputation (RR): 0.36 for HBO treated patients (95%CI: 0.22-0.59), P<0.0001.

DISCUSSION Interpretation of results and possible conclusions were deeply discussed in the committee. Major concerns were the relatively small number of study patients in relation to prevalence of DFS, and inclusion of studies with lower EBM level in the meta-analysis. On the other hand the committee was aware, that inclusion of these studies in the meta-analysis can be justified because of homogeneity and consistence of results in all included studies, and that quality and number of studies for HBO in DFS is relatively good compared to the situation for a number of other -well accepted- treatments for DFS. Finally the committee ended up in a decision comparable to the one of Medicare/Medicaid (Shuren et al. 2002) in accepting cost coverage for HBO only for Wagner grades III and higher.

CONCLUSION The Federal Joint Committee for German Social Health Care System concluded that costs for HBO therapy for inpatients with DFS are covered by Social Health Care System for DFS wounds graded Wagner III or higher, when there is no adequate tendency of healing under standard treatment.

HOPON: HYPERBARIC OXYGEN TO PREVENT OSTEORADIONECROSIS

Richard Shaw

Mersey Head and Neck Oncology Research Unit, School of Cancer Studies, University of Liverpool

Aims:

The aim of this presentation is to discuss the development of a feasibility RCT in HBO within the head and neck radiotherapy field. The various hurdles and regulatory frameworks will be discussed as well as issues regarding patient recruitment.

Introduction

Several NHS directors of specialist commissioning have questioned to continuing funding for HBO in H&N in the absence of convincing data. HOPON was developed by the National Cancer Research Institute head and neck clinical studies group during 2007. It was funded by Cancer Research UK as a feasibility study to recruit 50 patients over 24 months prior to evaluation for a fully powered trial.

Methods & Results

The various MHRA, EUDRACT, NCRN and ethics regulations associated with such a trial are described. NHS institutional barriers to research such R&D departments, site agreements, lack of trials practitioners, lack of central guidance are discussed.

HOPON was sponsored by the University of Liverpool and University Hospital Aintree and opened in October 2008. It is co-ordinated and led from Liverpool Cancer Trials Unit. 7 centres have been opened and 8 more are in set-up within the UK as well as 3 foreign units.

Recruitment data will be presented updated as per August 09 (currently 15 patients over 3 units)

Discussion

The potential of developing HOPON into a fully-powered RCT is discussed in light of recruitment, completeness of data, utility of primary / secondary endpoints, drop out rates.

CARBON MONOXIDE POISONING. A VERY COMMON INTOXICATION, SOMETIMES HIDDEN AND FREQUENTLY FORGOTTEN. SOME REFLECTIONS AFTER THE ANALYSIS OF 3320 CASES.

Jordi Desola

Joan Sala-Sanjaume. CRIS-UTH, Barcelona.

Free liberation of Carbon Monoxide (CO) is not frequent but CO is the result of incomplete combustion of organic materials, so inhalation of CO in fires, heating devices, stoves, braziers, escape gases from explosion engines, and other domestic sources are absolutely universal. Being CO colourless, odourless, tasteless and non irritative, unadvertised inhalation is easy. The world number of cases of poisoning by CO (CMP) is unknown but it must be necessarily very high. In contrast, the only explanation for the low number of cases of CMP reported in hospitals and emergency institutions, must deal with errors and misdiagnosis. When chronic intoxications happen as a result of continuous inhalation of low concentrations of CO, the confusion is still easier. The signs and symptoms of mild cases are completely unspecific : headache (89.1%), nausea or vomiting (52.7%), or vertigo (43.6%). In most serious but not critical cases, a transitory consciousness disturbance (26.6%), typical sherry skin rash (20.1%) and moderate bradypsichia (16.1%) can be observed, sometimes within an epidemiological environment that makes easiest the diagnosis. Inhalation of high concentrations of CO can produce very serious neurological disorders or even death, depending on the duration of the exposition, concentration of CO, and existence of individual risk factors. The diagnosis of CMP must be always based on these clinical findings. Determinations of on site CO concentration in expired air (COEA), or CO saturation (SpCO) by Pulsecooximetry can be very useful for early diagnosis. Once in the emergency department, determination of blood Carboxyhaemoglobin can be useful to confirm the clinical suspicion, but none of these determinations are neither essential nor mandatory to establish the diagnosis of CMP. It is generally accepted that the most serious is the condition of the patient, the highest will be the percentage of HbCO, but in the series of CRIS-UTH (3172) cases in December 2008) this correlation was not statistically significant. After an apparent recovering with normobaric oxygen, and being the patients asymptomatic and thus discharged from hospitals, some of them experienced a sudden neurological deterioration due to white matter demyelination. This Delayed Neurological Syndrome (DNS), has been observed after non serious cases of CMP that have not received a satisfactory treatment. When the source of CMP was a fire in a closed environment, with very high temperature, and combustion of synthetic materials, Cyanide (CNH) can be also formed thus producing a combined poisoning. It is widely accepted that the main treatment of CMP is based on the administration of Oxygen at the highest concentration possible. There is no doubt of which is the therapeutical device that can afford the highest quantity of oxygen, but some polemic is frequently established to accept the only device that gives this extra high quantity of oxygen. Such kind of discussion simply concerning not to the efficacy of a drug but simply to the convenience of providing the required highest dose of this drug, has no precedent in other fields of Medicine. CRIS-UTH, the hyperbaric therapy unit of Barcelona, is applying Hyperbaric Oxygenation since 1980 in the treatment of CMP. The 99.4% of our patients obtained a satisfactory outcome in the short term; some sequellae remained in 7 serious patients (0.22%) whose treatment was delayed for more than 24 hours; four extremelly critical patients (0.12%) died, but in all cases the HBO treatment had been delayed for many hours, or even some days. We have no reference that any of our patients had developed a DNS. According to the so-called Baud Criteria, 92 of our patients (2.92%) suffered simultaneously a serious combined intoxication by CNH plus CO. The evolution of all these patients was satisfactory; no of them had received high doses of Hydroxicobalamin. HBO is always facing any kind of reluctance mainly coming from persons and centres without access to this therapy. Such aggressive attitude is rarely observed against new expensive drugs widely supported by the pharmaceutical industry. World data on HBO treatment of CMP is consistent. An equanimous approach to CMP reviewing published results with the available therapies will endorse few suspiciacs.

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SILENT KILLER IN POLAND: CARBON MONOXIDE POISONED PATIENTS TREATED BY HYPERBARIC OXYGEN THERAPY IN POLAND IN 01.01.2003-31.05.2009

Jacek Piechocki(1,2) Michał Patyk(1,3), Janusz Sokołowski(2)

(1) Warsaw Hyperbaric Center , Poland

(2) Department of Emergency Medicine , University of Medicine in Wrocław, Poland

(3) Clinic of Anesthesiology and Intensive Care, Military Medical Institute in Warsaw, Poland

There are currently six places in Poland in which patients are treated by HBOT: Gdynia, Sosnowiec, Siemianowice Śląskie, Wrocław, Łódź and Zduńska Wola. In recent years the number of carbon monoxide poisoned patients increased significantly.

In the USA 1500 people die because of CO poisoning.

Official statistics in Poland show approximately 1400 CO poisoned people yearly, out of which 50-60 people die (Statistical Journal 2004).

However, this is probably the tip of the iceberg. Not all patients are correctly diagnosed and treated.

The most common reason of CO poisoning in Poland is heating system in many apartments, and in particular very popular water heaters in bathrooms (e.g. in Warsaw Hyperbaric Center 65% cases of poisoning).

Authors present treatment of CO poisoned patients in places providing HBOT (approx. 500 patients yearly).

Based on Warsaw's experience authors present how it was possible to shorten the time of reaching by patient the Hyperbaric Chamber from the place of accident.

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CARBON MONOXIDE INTOXICATION. THE TREATMENT IN DENMARK AND A CASE STORY.

Erik C. Jansen, Henrik Rottensten, Michael Bering Sifakis.

The hyperbaric Unit, department of Anesthesia, Center of Head and Orthopedics, Rigshospitalet, Copenhagen, Denmark.

Aims: Through treatment and investigation provide information to the health personnel and the public about the risk for carbon monoxide intoxication.

Methods. In Denmark the treatment of CO-intoxication is regulated by a directive issued by the National Board of Health in 1995. The health personnel has to seek advice at one of the hyperbaric units in Denmark if the patient has been exposed to CO and one of the following: 1. The patient has more neurologic symptoms, than normal headache. 2. The patient has circulatory/heart problems, 3. The patient is or has been unconscious, 4. The patient is pregnant, 5. The patient has > 25% CO-hemoglobin.

Average 33 patients are treated annually in the Copenhagen unit. Each patient is given between 3 and 4 HBO treatments within the first 48 hours after intoxication.

The regulations provide the possibility to treat patients with acute and chronic CO-intoxications and obtain information on the mechanism of intoxication.

Results. Among the acute and chronic intoxications we found 2 instances of intoxication from a newer gas furnace. In one case 1 person was chronically intoxicated and 2 were intoxicated through one night. In the second case one adult and 3 children were intoxicated through. Status after 2½ years after the accident: the boy 4 years old at the time of accident has problems finding the correct words. The then 10 year old boy has had no intellectual alteration since the time of the accident in spite of intensive training in a brain damage unit. The then 13 year old girl seems to have only minor brain impairments. The mother, then 42 is on pension after having been a school teacher and has difficulty in reading and is easily tired.

The reason for the accident is a gas furnace with balanced intake of air and exhaust of burned gas. The funnel has a T-piece with a lid. The fixation of the lid corroded and the exhaust gas stayed inside the building and made a closed circuit producing still more CO.



The funnel construction, which has shown dangerous unless repaired.

Discussion. In order to provide the best treatment, improved public safety and general awareness is important to include the medical expertise.

We conclude, that formal regulations in the treatment of CO intoxication provides a platform for improvement of health.

Bilateral sensorineural hearing loss after acute carbon monoxide poisoning – A case report

L. Fischer¹, M. Lechner¹, N. Patel², M. Ignatescu¹

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² Department of Otorhinolaryngology, Whipps Cross University Hospital, London, United Kingdom

Background:

Carbon monoxide (CO) which is a colourless, odourless, and tasteless toxic gas produced by incomplete combustion, claims the lives of around 50 people in the UK each year and represents the leading cause of accidental deaths by poisoning in our country.¹ Sources of carbon monoxide include faulty furnaces, inadequate ventilation of heating sources and exposure to engine exhaust. The symptoms are often non-specific and patients may demonstrate varied clinical manifestations with different outcomes, even under similar exposure conditions.² The most common symptoms are headache, myalgia, nausea, dizziness, short term memory loss or neuropsychological impairment as well as confusion and loss of consciousness with subsequent death in severe cases.³ Hyperbaric oxygen therapy is regarded as the most efficient treatment in order to decrease the risk of long-term neurological sequelae.⁴ Sensorineural hearing loss caused by carbon monoxide poisoning which we will report here seems to be a very rare neurological manifestation.

Case Report:

We report the case of an 18 year old gentleman who was found unconscious (GCS 6/15) in a storeroom of a fish and chips restaurant together with his girlfriend lying next to him who was already in rigor mortis. A faulty boiler was found to be the cause for the very high concentrations of carbon monoxide measured in the room and leading to the poisoning. The patient was intubated on scene by the ambulance crew and airlifted to the local A&E department where the diagnosis of CO poisoning was confirmed by HbCO of 12.9% after 45 minutes of ventilation with 100% oxygen. A cranial CT scan and a toxicology screen were all normal. An intensive care transfer to our Hyperbaric Unit was arranged. On arrival the patient was intubated and ventilated, he had signs of centralisation (akrocyanosis, capillary refill 5 seconds) and required inotropic support (Noradrenaline 1mg/h) throughout the first two treatments 6 and 12 h after the incident. (LHM-CO table; 18.2m/2.8 ATA; 100 minutes incl. air breaks). Grommets were inserted bilateral after the first treatment. After the second treatment the hemodynamic condition had improved (reduced inotropic demand, capillary refill 2 seconds) and extubation was successfully performed 20 hours after his initial discovery. He was dependant on CPAP for 3 more days and did therefore not undergo a third hyperbaric treatment. A neurological examination after extubation revealed profound bilateral hearing loss, which was classified by the ENT specialists as sensorineural and not conductive. On the audiogram, the hearing threshold was at 80 to 120 dB on various frequencies. The hearing loss did not improve over the next days. A treatment with high-dose Prednisolone (1mg/kg for 7 days) was started rather late (14 days after the incident). Concomitantly hyperbaric oxygen treatment was started (“Problem wound table”, 2,4 ATA, 90 minutes, once daily). The patient received a total of 30 hyperbaric treatments following the two initial emergency exposures. Audiometry at the end of the treatment showed an improvement of 20 dB on single frequencies in both ears. On follow-up, a thorough neurological exam showed no evidence of further neurological sequelae.

Discussion:

Isolated persistent sensorineural hearing loss is a very rare event in carbon monoxide poisoning and there are only a few reports found in the literature describing it as a neurological sequelae.^{5, 6, 7, 8, 9, 10, 11, 12}

The pathophysiology is only partly understood and it is not clear whether the hearing loss is due to impairment of the cochlea, of the vestibulocochlear *nerve or the relating areas of the brain involved*. Recently, L. Weaver showed that in a CO intoxicated patient with previously normal auditory acuity normal activation after auditory stimulation of the right ear and no activation after auditory stimulation of the left ear occurred using auditory functional MRI. This pattern is consistent with normal auditory processing on one side but suppression of processing on the other side, presumably in relation to brain injury from carbon

monoxide poisoning.⁴ This explains one potential pathophysiological mechanism. Furthermore, in an *in vivo* experiment L.D. Fechter et al. showed that free radical blockers have a protective effect on the cochlea when animals are exposed to carbon monoxide and thus injury to the cochlea might explain the sensorineural hearing loss in our case as well.¹³

The benefit of subsequent hyperbaric oxygen treatment has not yet been sufficiently evaluated by a randomized controlled trial but there are many reports of hearing improvement in case series^{14, 15}. In the case of the described gentleman we observed improvement of his hearing during HBO treatment of 20 dB at 250 Hz in the left ear and 20 dB at 8000 Hz in the right.

Conclusion:

We are reporting a case of near fatal acute carbon monoxide poisoning that responded well to early hyperbaric treatment. As a rather unusual complication we observed profound bilateral sensorineural hearing loss that improved under further hyperbaric exposures.

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EVALUATION OF THE EFFECTS OF HYPERBARIC OXYGEN ON RADIATED FIBROBLAST CULTURES

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AIM: The aim of this study is to evaluate the effects of HBO on fibroblast vitality and cell proliferation capacity on radiated fibroblast cultures.

INTRODUCTION: Cancer is one of the diseases that seriously affect patient survival and quality of life. Radiotherapy is used commonly among cancer treatment modalities and healthy tissues might be affected from this treatment radioactivity. Treatment of radiotherapy side effects are generally very difficult. Hyperbaric oxygen therapy (HBO) is based on breathing 100 % oxygen in pressure chambers which kept above atmospheric pressures. The positive effects of hyperbaric oxygen on neovascularisation and wound healing has rised up the idea of using HBO treatment for radiotherapy side effects.

METHODS: In this study, fibroblasts cultured from skin biopsy of a healthy donor are divided into 4 groups: Control (A), HBO (B), radiotherapy (C), radiotherapy+HBO (D). Groups C and D are radiated with a dose of 5 Gy. Daily hyperbaric oxygen for 90 minutes is applied on groups B and D with a pressure of 2.4 ATA for 7 consecutive days. Cultures are evaluated for cell vitality and proliferation index parameters on day 1, 3 and 7.

RESULTS: Cell vitality and proliferation index were found to be statistically higher in group D when compared to group C on evaluation days 3 and 7. Proliferation index of hyperbaric oxygen (applied) radiated group (Group D) reached the level of unirradiated groups (Group A and Group B) on day 3.

DISCUSSION: The results of this study indicates that HBO therapy has positive impacts on the cell vitality and proliferation capacity of radiated fibroblast cultures. Fibroblast cells that are vital and have a potential to proliferate play an important role in the healing process of the hypoxic, hypocellular, hypovascular radionecrotic tissue so this study has supporting findings with the clinical studies that encourage the use of HBO therapy for radiotherapy side effects.

HYPERBARIC OXYGEN REDUCES ENDOTHELIAL ADHESION MOLECULE EXPRESSION AND NEUTROPHIL RECRUITMENT UNDER FLOW IN AN *IN VITRO* CHRONIC WOUND MODEL

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Aims: The aim of this work was to determine the effect of hyperbaric oxygen (HBO) on the recruitment of neutrophils by endothelial cells in a chronic wound model. We wished to examine the effect of HBO on endothelial cell adhesion molecule expression, and then monitor neutrophil adhesion to these cells under flow.

Introduction: HBO can enhance the healing of chronic wounds, particularly diabetic lower extremity ulcers, but the molecular mechanisms involved are not fully understood. One of the problems in chronic wounds is excessive neutrophil recruitment. Tissue hypoxia results in impaired respiratory burst activity, and the neutrophils release destructive proteases that destroy healthy tissue and growth factors. This combination leads to a failure of the wound to heal and prolonged inflammation. If HBO reduces the recruitment of neutrophils into chronic wounds, this could explain in part the reduction in inflammation observed with HBO treatment. HBO has been shown to impair neutrophil recruitment during ischaemia reperfusion injury, but not as yet in chronic wounds. The development of an *in vitro* chronic wound model here has allowed a detailed investigation of neutrophil-endothelial cell interactions under HBO.

Methods: An *in vitro* chronic wound model was established for human umbilical vein endothelial cells (HUVEC). HUVEC were treated with 0.5 µg/ml *P. aeruginosa* lipopolysaccharide (LPS), 1 ng/ml tumour necrosis factor-alpha (TNF-α) and cultured in 2 % O₂ (i.e. hypoxia) for 24 h. HUVEC from this chronic wound model were then subjected to a 90 min HBO treatment (95 % O₂ at 2.4 ATA) or one of two controls – hypoxia (2 % O₂ at 1 ATA) or a pressure control (2 % O₂ at 2.4 ATA). Following treatment, cells were returned to normobaric hypoxia for 24 h, at which point surface and total expression of the adhesion molecules ICAM and VCAM were assessed by ELISA and western blot respectively. To assess the effect of HBO on the recruitment of circulating neutrophils by an endothelial monolayer, a flow system was established whereby neutrophils, either untreated, or subjected to the chronic wound model +/- HBO or the pressure control, were flowed over a treated HUVEC monolayer at a physiological shear stress.

Results: HBO treatment reduced HUVEC surface expression (as measured by ELISA) of ICAM and VCAM by 46.6±12.8 % (mean±SD) and 21.9±29.4 % respectively (P=0.017, n=18). HBO reduced total ICAM and VCAM content (as measured by western blot) by 10.2±6 % and 27.8±8.2 % respectively (P=0.037, n=3). In the flow assay, HBO treatment reduced neutrophil recruitment by 89.9±7.8 % (P=0.037, n=3) when HUVEC alone received HBO, by 76.5±6.7 % (P=0.037, n=3) when neutrophils alone received HBO, and by 65.2±8.8 % (P=0.037, n=3) when both HUVEC and neutrophils received HBO.

Discussion: These results demonstrate a clear effect of HBO on the recruitment of neutrophils in the context of a chronic wound model - HBO reduces the number of neutrophils adhering to a HUVEC monolayer under flow. This could be partially explained by a reduction in the expression of the endothelial adhesion molecules ICAM and VCAM, which was observed following HBO. In chronic wound patients receiving HBO, this reduced expression could result in a reduction in the numbers of neutrophils recruited to the wound and a reduction in inflammation.

HEPATOCYTES GLUTAMINSYNTHEASE RESPONSE TO HYPERBARIC OXYGENATION DEPENDS ON CELL STATE AT THE MOMENT OF OXYGENATION

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INTRODUCTION. Effects of HBO in clinic sometimes are difficult to predict. A lot of factors are now estimated to influence on it. But data of studies still remain controversial.

AIMS. To investigate if the enzyme reaction to HBO application depends on initial functional state of cell.

METHODS. Experiments were performed on 50 white female rats (mass 180-220g). Glutaminsynthetase (GS) activity was measured by colorimetria in hepatocytes microsomal fraction. There were 7 experimental groups: healthy intact animals, healthy animals with HBO, animals with liver resection (LR, removing 15-20 % of the organ's mass), animals with liver resection and HBO, animals with chronic hepatitis (caused by tetrachloromethane CCl₄ 50 % solution in olive oil in dose 0,1ml per every 100 g of a body mass, subcutaneously, once a day, 65 days), animals with chronic hepatitis and LR (operated on 65th day after CCl₄ course beginning), animals with chronic hepatitis, LR and HBO. Three everyday HBO sessions were carried out in regimen 3ata for 50 minutes. For animals with LR HBO was started in 4-8 hours after the operation. Right after the third session animals were anesthetized, decapitated and liver was taken for examination.

RESULTS. In norm the GS activity was $1,14 \pm 0,09$ nanomole /mg of protein•second. After the 3-rd HBO session of healthy animals it decreased to $0,57 \pm 0,08$ ($p < 0,05$). If the HBO was applied after LR in healthy animals the GS activity was $1,46 \pm 0,15$ whereas in non-oxygenated group it was $1,56 \pm 0,18$. CCl₄ hepatitis led to the GS activity decreasing - at the moment of operation it was only 29 % from norm whereas LR increased it up to $0,91 \pm 0,11$. If for animals operated on the background of a chronic hepatitis the HBO was applied the GS activity raised up to $3,11 \pm 0,58$, i.e. enlarged in 2,7 times in comparison with norm.

DISCUSSION AND CONCLUSION. Therefore, the GS of operated liver hepatocytes in healthy rats is refractory to HBO action. Hyperbaric Oxygen considerably enhances stimulative effect of LR on the GS activity in liver hepatocytes affected by CCl₄. The results obtained allow to suppose that response of an enzyme to the action of Hyperbaric Oxygen depends on a functional state of cell at the moment of its action on organism.

TOPIC: HYPERBARIC PHYSIOLOGY

EFFECT OF HYPERBARIC OXYGEN THERAPY ON TENSE REPAIR OF THE PERIPHERAL NERVES

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Aim: The aim is to study the effects of hyperbaric oxygen therapy (HBOT) on primary repair of peripheral nerve under tension.

Introduction: There have been some advances in peripheral nerve repairs, but full functional recovery could not be reached yet. Factors such as co-existing injuries, age, delay of repair, proximity of lesion and surgical problems like tension on the repair site have negative effects on healing. Tense nerve repair which is not unusual in clinical practice is thought to cause hypoxia on repair site. Therefore, HBOT, which was shown to improve nerve regeneration, may have enhancing effect on tense repair of the nerves.

Methods: 16 Wistar albino rats were used. Sciatic nerves of the rats were transected and 3 mm piece was excised. Then, the ends were repaired primarily with microsurgical technique. After the operations the rats were randomly assigned into two groups. HBO was administered at 2.5 ATA for 1 hour to one of the groups for three weeks. Schedule was planned to be every eight hours for the first three days, every twelve hours for the second three days, then once a day. Other group did not receive HBO.

Starting from day 12, on days 15, 17, 20 and 22 walking track analysis were performed. SFI scores were calculated. At the end of the study, on day 22, the electrophysiological recordings for compound field potential were completed. 1st and 2nd latencies for both groups were determined. The animals were sacrificed after the tests and a 0.5 cm length of each nerve that involves the repair site was harvested. Those pieces were stained with Hematoxylin-Eosin and axons were counted under light microscope. The results of axon counting and latencies were compared with Mann-Whitney U test and SFI scores were compared with Pillai's Trace test.

Results: SFI scores were found to be significantly higher in HBO group. Also mean SFI scores of each evaluation day were compared and SFI scores of HBO group were found significantly higher for each evaluation day except day 12. Average number of axons in the HBO group was found to be significantly greater than the control group. However, no difference was found between the groups for both latency 1 and latency 2 values.

Conclusion: In this first study about efficiency of HBO on tense nerve repairs, it is found that HBO enhances functional recovery of tense nerve repairs.

Keywords: hyperbaric oxygen, tension, nerve repair, SFI, electrophysiology

PRECONDITIONING TO HBO MAY PROVIDE PROTECTION AGAINST CNS OXYGEN TOXICITY IN THE RAT: BEATING THE "DEVIL'S TRIANGLE".

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Abstract.

Aims and Background: We tested the hypothesis that repeated hyperbaric O₂ preconditioning (HBO-PC) may have a protective effect against CNS oxygen toxicity (CNS-OT) in the rat.

Methods: The rats in control group 1 (C1) were kept in normobaric air together with five sham rats. Rats in the experimental group and control group 2 (C2) were exposed to HBO at 202 kPa for 1 h as preconditioning once every other day for a total of three sessions. Twenty-four hours after preconditioning, the rats in both the experimental and C1 groups were exposed to 608 kPa. We measured the latency to CNS-OT, after which all of the animals were sacrificed and tissues were harvested from the hippocampus and frontal cortex for biochemical examination.

Results: Time to CNS-OT increased significantly following preconditioning. There was a 20%, statistically significant increase in the activity of glutathione-S-transferase (GST) and glutathione-peroxidase (GP) in the cortex of the preconditioned rats. Nitrotyrosine levels in the cortex did not demonstrate any significant trend. In the hippocampus of the preconditioned rats, a significant decrease was found in the activity of glutathione-reductase (GR) and G6PD, whereas there was a significant increase in the activity of GP. Nitrotyrosine levels in the hippocampus demonstrated the same trend in all five marked proteins. The highest levels were found in the C1 group, whereas the lowest levels were found in the experimental group.

Conclusions: This study demonstrates that under well defined conditions, repeated exposure to HBO may have a preconditioning effect, providing protection against CNS-OT. The protective mechanism involves alterations in the enzymatic activity of ROS scavengers induced by HBO exposure, mainly in the hippocampus. These alterations result in lower levels of distractive RNS (and probably ROS) and prolonged latency.

NONINVASIVE EVALUATION OF SYSTEMIC HEMODYNAMIC AND MICROCIRCULATORY RESPONSE TO HBO IN MECHANICALLY VENTILATED INTENSIVE-CARE PATIENTS

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Aims:

The study aimed at observing the the behaviour of microvascular perfusion and systemic hemodynamics in ventilated and sedated ICU patients before, during and after HBO therapy, at determining the correlation between micro- and macrocirculation with regard to elevated ambient pressure, and at recording the catecholamine demand during the study period.

Patients and methods:

Inclusion criteria: routine HBO treatment according to the disease, stable hemodynamic condition without modification of the treatment within 6 h before the session.

After approval by the local ethics committee, 10 mechanically ventilated and sedated ICU patients (Ramsay Sedation Scale grading 5-6) undergoing routine HBO therapy were enrolled in this prospective observational study. Patient data: 4f, 6m, mean age: 60 yr; diagnosis: cellulitis in 8 pts., acute cerebral hypoxia in 2 pts, catecholamine support in 4 pts.

The following parameters were obtained every 5-10 min at 30 min before treatment , during a stabilization phase of 15 min after transport to the chamber with normobaric oxygen breathing(NBO), during and until 90 min, and at 120, 150 and 180 min after the session: standard monitoring of heart rate (HR), arterial blood pressure, central venous pressure (CVP), peripheral oxygen saturation (SaO₂), cardiac output (thoracic bioimpedance), and, in the subclavian region, transcutaneous oxygen (tcPO₂) and carbon dioxide (tcPCO₂) tension, microvascular perfusion (flux, laser-Doppler flowmetry). Routine arterial blood gas sampling at 1 h after the session. HBO protocol: 252.5 kPa, 90 min. *Statistics*: linear mixed model , Wilcoxon rank test. P< 0.05 = significant.

Results:

Macrocirculation: HR declined significantly by 4.6% after transport to the chamber (p= 0.041), afterwards no significant difference. No change of blood pressures and cardiac output. *Microcirculation*: Significant change of tcPO₂ with inspired oxygen pressures (p< 0.0001), no change of tcPCO₂ and flux. Correlation coefficient between cardiac output and flux: 0.43 (P= 0.0007), no correlation between flux and tcPO₂.

Significant mean decline of arterial oxygen tension by 10.1 % (P= 0.0039) after the session.

No increased catecholamine demand during and after the session.

Discussion and conclusion:

The exposure to HBO was well tolerated by hemodynamically stable ventilated intensive-care patients. In our collective, there was a positive correlation between laser Doppler flowmetry flux values and bioimpedance-based cardiac output values, contarily to the flux-tcPO₂ interrelationship. The early post-session decline of arterial PO₂ might be attributed to atelectasis formation and is likely to be reversible by applying a recruitment maneuver.

This study was created within the frame of the COST B14 action on hyperbaric medicine.

HYPERBARIC OXYGEN THERAPY USED TO TREAT CHRONIC RADIATION PROCTITIS

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Aims: To evaluate the effects of hyperbaric oxygen therapy (HBOT) in chronic radiation proctitis (CRP) which is refractory to conventional therapies.

Introduction: CRP is a late complication of pelvic radiotherapy which occurs after a latent period. In general incidence is reported as less than 5% (1,2). CRP is progressive by its nature and can be devastating. Clinical presentations are pain, urgency and diarrhea, constipation, tenesmus, mucoid or bloody discharge, bleeding or combination of these. There is no standard treatment exist, and the results of conventional treatment are poor. HBOT has been used in recent years for treatment of CRP and reported to be beneficial (3,4).

Methods: Present study reports the results of 7 cases of CRP who were non-responding to conventional treatment and referred to our center for HBOT between the years of 2001-2009. Patients' diagnosis was confirmed by endoscopy before initiating the HBOT. HBOT was given once a day, 5 days in a week. 6 patients received HBOT in a monoplace chamber. Treatment protocol was 100% O₂ for 90 minutes at 2.1 ATA. One patient treated in a multiplace chamber at 2.4 ATA, as three 25 minutes periods of 100% O₂ dispersed by 5 minutes of air breaks.

Results: The mean age of the patients (4 male, 3 female) was 63.5 (range 50-75). Common complaints were pain, frequency, diarrhea, bloody discharge, tenesmus and occasional bleeding. The primary diagnosis were prostatic carcinoma in 4 cases, others were rectal carcinoma, endometrial carcinoma and malign melanoma. Pelvic radiotherapy doses were minimum 45 Gy, maximum 73.8 Gy. The mean latent period was 5.5 months (range 2-11). The mean number of total HBOTs were 40.

Endoscopic evaluation performed after completion of HBOT showed significant healing in 5 and partial improvement in two patients. All patients were free of pain. Frequency and diarrhea absent in 6, and bloody discharge stopped in 5 patients. 5 patients (%71) discharged with complete healing and two patients with partial improvement. No complications developed due to HBOT.

Discussion: Though this study is a retrospective and non-controlled one and number of patients are few, clinical and laboratory results of this study emphasize that HBOT can be effectively used for the treatment of refractory chronic radiation proctitis.

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THE NORMOBARIC OXYGEN PARADOX (NOP): A SAFE ALTERNATIVE TO POST-OPERATIVE TRANSFUSIONS AFTER HIP surgery?

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Allogenic blood transfusion is often used to maintain adequate oxygen tissue delivery after significant blood loss. But, as the list of potential complications from blood transfusions grows, it has prompted alternative blood management strategies.

Since the recent discovery of the Normobaric Oxygen Paradox (NOP) capable of increasing erythropoietin and haemoglobin, further investigation was needed into the clinical applicability of the NOP. Based on these evidences, we hypothesized that intermittent oxygen breathing could significantly reduce the requirements for allogenic blood transfusions after traumatic hip surgery.

Methods

34 patients hospitalized for hip fracture were enrolled in this study. Each patient was given an oxygen mask during 30 minutes per day and randomly assigned to receive either air (n=15) or oxygen (n=13) starting from day 1 after surgery. Venous blood samples were taken at admission and after surgery on Day 1, 3 and 7. The postoperative follow-up was achieved by a dedicated physician, blinded to the breathed gas, without changing either the standard practice or the transfusion policy of the institution.

Results

Groups are comparable as far as age, sex ratio, delay to intervention, side ratio, intervention, blood loss, and associated pathologies are concerned. Upon hospital discharge there is no difference in haemoglobin between groups ($p = 0.674$). In the air group 53.3% of the patients needed allogenic blood transfusion versus 7.7% in the oxygen group. The association between the NOP and the reduced requirements for allogenic blood transfusion is proven significant ($p=0.037$, two sided Fischer's exact test).

Conclusions

Based on these preliminary results, it seems that the NOP applied with a minimal protocol (30 min every day), may be sufficient to either increase haemoglobin concentration or reduce post-operative blood loss in the elderly after hip surgery thus preventing the need for allogenic blood transfusions. This observation needs to be confirmed on a larger scale.

DRAFT EN ON HYPERBARIC CHAMBERS: SPECIFIC REQUIREMENTS FOR FIRE EXTINGUISHING SYSTEMS

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Introduction:

Products manufactured in compliance with harmonized standards benefit from a presumption of conformity with the corresponding essential requirements. These requirements deal in particular with the protection of health and safety of users and sometimes cover other fundamental requirements, e.g. protection of property or the environment (cf. Guide to the implementation of directives based on the new approach and the global approach – European Communities – Office for official publications of the European Communities, 2000). The medical hyperbaric chambers are, in Europe, medical devices which fall under the dispositions of the directive 93/42/EEC (Council Directive 93/42 of 14 June 1993 concerning Medical Devices; 12.7.93 N°L 169/1 – 169/43). An European standard for hyperbaric chambers already exist (EN 14931: 2004 Pressure vessels for human occupancy – multiplace pressure chamber systems for hyperbaric therapy – performance, safety requirements and testing) but it does not include a standardization of the fire extinguishing systems. The European Committee for Standardization (CEN) now has informed all technical committees about the applicability of Essential Health and Safety Requirements (EHSR) of machinery (2006/42/EC) to medical devices – and this includes risks due to extreme temperatures, fire and explosion not included in EN 14931. Therefore CEN/TC 359, (former CEN/BT/TF 127), is requested to check EN 14931.

Discussion:

The first meeting of CEN/TC 359 was held 2009-03-25/27. The meeting agreed to take the German standard DIN 13256-3 (DIN 13256 – 2: Pressure vessels for human occupancy PVHO Art 5.2.19 – Deutsches Institut für Normung – Germany – Ed 1996) as a basis for preparing the European Standard. It was agreed that only fire extinguishing systems and fire alarm systems for multiplace chambers are to be included in the standard. The meeting agreed on an informative annex dealing with prevention of fire, evacuation systems and the position of sensors in the hyperbaric chambers.

Result:

The German standard DIN 13256-3 was translated and rewritten to the first version of the a CEN/TC 359 working document (N 15). The European Standard described in this document is applicable to the performance and safety requirements of fire extinguishing systems and their associated test methods for multi-place pressure chambers designed for pressures in excess of ambient atmospheric pressure and employed in medical installations for therapeutic purposes. The document includes the following part:

1. Scope (of the standard)
 2. Standard References
 3. Definitions (of the fire extinguishing system)
 4. Performance and Requirements (of the fire extinguishing system)
 5. Test Requirements for Fire Extinguishing Systems in Hyperbaric Atmosphere
- Annex A: (description of the dummy used in the tests)
Annex B (informative)
Bibliography

The draft document must be finalized by end of 2009. After preparation of the French and German language versions the draft standard shall be published by March 2010 for a 5 month enquiry.

FREDERICK LOVE KEAYS: THE GENESIS OF MODERN HYPERBARIC OCCUPATIONAL MEDICINE

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Frederick L Keays (1871-1950) was a founder of Nassau Hospital Mineola, now Winthrop University Hospital New York, and chief of its medical service department. He worked as a physician in New York from 1899 until his retirement in 1948. He is best known to us, however, as the medical director of construction work on East River tunnels from 1904 to 1910 for S Pearson & Son, and his remarkable experience of decompression illness during this time. In a period of 557 days with about one thousand men a day working in compressed air at 32-42 pounds per square inch, his medical department saw 3692 cases of compressed air illness with 20 deaths. His account of this experience was published in 1909 and remains one of the best monographs on decompression illness ever published (1). He personally saw most of the serious cases of compressed air illness and many of the milder ones and felt that the possession of a large number of complete records of cases of compressed air illness might enable him to write a paper which would prove of practical value.

Keays made a number of observations that have stayed with us down the years. Age, obesity, fatigue, concurrent disease, alcohol abuse and newness to work were all important predisposing factors for decompression illness and, to a large extent, could be screened out of the work force. He advocated screening by medical examination for all workers and preliminary test compression for new candidates.. Keays also noted that cases of decompression recompressed more than six hours after the onset of symptoms did as well with medical treatment only and that cases with “prostration” had a high mortality. The symptoms and signs of prostration are be recognised as those of hypovolaemia today and Keays was the first to make this important observation.

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THE HALDANE EFFECT

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John Scott Haldane, British physiologist and philosopher, was born in 1860 in Edinburgh, Scotland. Haldane investigated poisonous gases occurring in coal mines and wells, sunstroke, the physiological action of carbon monoxide and the use of a caged canary for early CO detection, the regulation of lung ventilation (with J.G. Priestley), and devised the haemoglobinometer, the apparatus for blood-gas analysis. He also described the effects of oxygen deficiency and exercise on breathing. During the First World War he worked on effects of poisonous gases and designed a portable oxygen administration apparatus. His work on hypoxia and the acclimatization of the human body to high altitude revolutionized concepts in respiratory physiology. Haldane published some landmark books on his philosophical ideas about the true significance of biology. Most importantly, however, Haldane investigated the problems of deep diving for the British Admiralty, developing the 'stage decompression' method, a lasting contribution to the diving world. This elaborate experimental investigation was conducted in part in a steel pressure chamber at the Lister Institute and with divers in Scottish deep-water lochs. In 1908, J.S. Haldane published those results in his seminal paper "Prevention of Compressed-Air Illness" in the *Journal of Hygiene* with A.E. Boycott and G.C.C. Damant. Stage decompression allowed divers to be safely brought to the surface and made it possible to conduct 120 fsw salvage operations on the LAURENTIC to recover over £5,000,000 of gold ingots without recordable incident. One hundred years later, the Norwegian University of Science and Technology in Trondheim convened the Haldane Symposium, December 18-19, 2008, celebrating the past, present, and future directions of environmental physiology research in decompression.

100 YEARS AGO IN HAMBURG: THE GENESIS OF MODERN HYPERBARIC OCCUPATIONAL MEDICINE

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The project: In 1908 the construction of the first underwater road tunnel in continental Europe started in Hamburg. Digging of the starting shafts on both sides of the river Elbe required caisson-work down to 2.4 bar pressure and resulted in numerous and serious decompression-related health problems, including two fatalities. This alarmed the authorities and the public, leading to heated discussions about workers' safety in the local press and the city's Bureau of Health.

Medical precautions: The doctor initially in charge had repeatedly proved his lack of competence in dealing with these problems and was replaced by a young neurologist with a solid background in respiratory physiology: Dr. Arthur Bornstein, joined by his doctor-wife, settled on the construction-site, setting up a research laboratory including a treatment chamber plus animal chamber. Starting in January of 1909, they introduced regular medical check-ups of the work-force, discussed shift duration and decompression regime with the authorities and initiated the application of tables that were to become the back-bone of the later German State Regulation on Compressed Air Work. The continuous interaction with engineers, supervising authorities and investors soon proved to be the key to success, discussing and convincing, fighting if necessary, thereby establishing a system of prophylactic measures which laid the foundation of today's occupational medical standards in compressed air work.

Studies and publications: Their research was focused on worker's health on introduction, establishing a compulsory range of examinations before taking up compressed air work and maintenance of their health by continuous improvement of decompression safety. For the first time in medical history the suitability of oxygen was tested to speed up the excretion of nitrogen during decompression and also in the treatment of "bends" which still occurred. Between 1910 and 1914 seven publications (in German) informed the scientific community about their achievements like "Prophylaxis of Decompression Sickness", "Effect of Compressed Air on Blood Formation", "Chronic Joint Alterations, induced by Decompression Sickness", "On Oxygen Poisoning", "Physiology and Pathology of Life in Compressed Air".

Results: Inspired by the Austrian predecessors Heller, Mager and von Schroetter and accepting the work of his contemporary Haldane, Bornstein created a comprehensive approach to health and safety in compressed air work. It formed the basis of the State Decree that came into force in the German Reich in 1921. Especially his early introduction of oxygen as decompression and treatment gas opened the door to handling the working depths that are challenging today's hyperbaric medical specialists in underwater tunneling world-wide.

Discussion: It should be widely accepted that already the construction of the Hamburg Elbe tunnel one hundred years ago initiated compressed air workers' health and safety standards that are considered mandatory today. Since the actual working depths are ever increasing, there is an urgent need for a joint effort of all international specialists active in this field. The "International Arthur Bornstein Workshops", held in Hamburg three times since 1998, are offering a platform for this challenge.

PREVENTING THE “BENDS” IN DEEP HYPERBARIC TUNNELING

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Aims: Since men started to work in compressed air about one and a half centuries ago, the “bends” have been a reliable companion of this endeavor. Though there are effective procedures available to cure this sickness there still remains uncertainty about late adverse health effects after having suffered from the “bends”. Therefore every construction project involving the application of work in compressed air should aim at a “zero-bends-rate”.

Introduction: For the last twenty years modern tunnel boring machines have been challenging the medical side by digging ever deeper. Currently exposures in compressed air up to and over six bar are successfully mastered somewhere in the world. But it is the range between three and four bar which is currently in the focus of applied field research in Germany. The vast majority of “bends” observed and treated is located in the lower extremities as was pointed out already in earlier publications.

Methods: In addition to well-known and proven methods of increasing decompression safety like adjusting decompression schedules we introduced pre- and post-decompression procedures to initially reduce bubble production, speed up the process of nitrogen release from the lower extremities and prevent the wide-spread post-exposure fatigue.

The system consisted of a short “dive” to working-depth in the treatment chamber before the first over-all exposure (like in new-comers or after a holiday) to reduce bubble nuclei, a method proven by other authors; air-breaks during oxygen decompression and an additional chamber exposure for 30 minutes at 0.9 bar for the complete shift on the surface, breathing pure oxygen while working a “step-mill” at intervals to increase circulation for the benefit of venous return. The time gap between leaving the man-lock and entering the chamber was not to be longer than 30 minutes. Additionally, climbing the stairs out of the starting shaft was forbidden.

This regime was compared to the standard procedure as outlined in the German State Regulation on the occasion of three tunnel projects (between 1999 and 2006), where twin tubes were constructed one after the other, providing identical settings for the studies.

Results:

Project	time frame	max.work press.	indiv lock-ins	“bends” treated
Weser South	may 99 – nov.00	4.9 bar	1.319	13
Weser North	apr. 01 – oct. 01	4.3 bar	453	1
Lübeck South	oct. 02 – apr. 03	3.6 bar	777	16
Lübeck North	july 03 – oct. 03	3.6 bar	166	2
Hamburg Airport F 2	jan. 05 – sep. 05	3,4 bar	335	7
Hamburg Airport F 1	jan. 06 – july 06	3,4 bar	215	3

Discussion: As can be seen, the incidence of “bends” was significantly lower with the second tube in each project. These procedures still deserve discussion and further scientific verification.

In all our tunnel projects since, they have been adopted as standard successfully; especially the work-force readily accepted the extra time burden realizing the beneficial effect. The “bends” have not totally vanished, but with these procedures they present much lighter and fast and easy to treat.

The recently finished projects in Malmö/Sweden and Thun/Switzerland produced not a single case of decompression-related pathology.

PROTECTIVE EFFECTS OF CLOPIDOGREL ON DECOMPRESSION SICKNESS INCIDENCE AND SEVERITY , IN A RAT MODEL; A PHARMACOLOGICAL INTERVENTION AGAINST BUBBLE-INDUCED PLATELET AGGREGATION

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Introduction

Decompression sickness (DCS) occurs when a subject is submitted to a reduction in environmental pressure. Previous studies highlighted that bubble-induced platelet aggregation (BIPA) offered an index for evaluating DCS severity in a rat model. In the present study, we compare the effects of three well known antithrombotic agents to determine what mechanisms are the most important in the prothrombotic syndrome: acetylsalicylic acid (ASA), clopidogrel (CPD) and heparin (HPR).

Methods

Male Sprague-Dawley rats (n=120) were randomly assigned to one experimental group treated before the hyperbaric exposure and decompression with ASA (3x100 mg/kg/day, PO, n=30), or with CPD (50 mg/kg/day, PO, n=60), or with HPR (500 IU/kg, SC, n=30) or untreated group (n=49). Rats were first compressed to 1000 kPa (90 msw) for 45 min then decompressed to surface in 38 min. In a control experiment, rats were treated with ASA (n=13), CPD (n=13), or HPR (n=13), and maintained at atmospheric pressure for an equivalent period of time. Onset of neurological DCS symptoms and death were recorded during a 60-min observed period after surfacing. Blood samples for platelet count (PC) were taken 30 min before hyperbaric exposure and immediately after surfacing.

Results and discussion

Clopidogrel, a specific ADP-receptor antagonist, put off DCS risk (death rate: 3/60 with CPD, 15/30 with ASA, 21/30 with HPR and 35/49 in untreated group) and DCS severity (neurological DCS incidence: 6/60 with CPD, 6/30 with ASA, 5/30 with HPR, and 12/49 in untreated group). Clopidogrel reduced platelet count fall an bubble-induced platelet aggregation (-4,5% with CPD, -17,6% with ASA, -19,9% with HPR and -29,8% with untreated group). These results clearly support the participation of ADP in platelet activation and prothrombotic event but cannot definitely prejudge the mechanisms between bubble-induced vessel wall injury and bubble-blood components interactions.

Conclusion

Clopidogrel would minimize secondary pathophysiological pathways and could offer a benefit as an adjuvant treatment for DCS.

SIMULATED DIVING AFTER HEAT STRESS POTENTIATES THE INDUCTION OF HEAT SHOCK PROTEIN 70 AND ELEVATES GLUTATHIONE IN HUMAN ENDOTHELIAL CELLS.

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OBJECTIVES: Heat stress prior to diving has been shown to protect animals from decompression sickness (DCS), and the protection was associated with production of heat shock protein (HSP) 70. Considerable evidence indicates that DCS is due to initial damage to endothelial cells and may be related to production of nitric oxide (NO) by nitrogen oxide synthase (NOS). The enzyme is closely related to another heat shock protein, HSP90, and a cofactor, tetrahydrobiopterin that is dependent on cellular red-ox status. The present investigation has focused on the relationship between HSP70, HSP90 and the major cellular red-ox determinant, glutathione (GSH), after heat stress and simulated diving.

METHODS: Human endothelial cells (HUVEC) were exposed to heat shock (1 h, 45 °C) and a subsequent simulated dive corresponding to 250 msw. After a rapid decompression (approx. 5 min.) the cells were harvested for analysis of HSP and GSH.

RESULTS AND DISCUSSION: Heat alone induced HSP70 several fold in HUVEC, while a simulated dive for 24 hours had no effect on the HSP70 level. Notably, a dive performed 24 hours after a heat shock had a potentiating effect on the HSP70 expression increasing the HSP70 concentration on average 54 times. In contrast, a simulated dive had no significant potentiating effect on the HSP90 level, which might be due to the higher baseline level of HSP90. Moreover, both 2 hrs and 24 hrs dive had similar effects on HSP70 and HSP90, suggesting that the observed effects were independent of duration of the dive. Since the HSPs were determined immediately after a short decompression time the rapid HSP-response following a 2 hrs dive might suggest that the effects were due to compression or pressure per se rather than decompression and may involve post-translational processing of HSP.

A reverse order of exposure showed that a dive prior to heat shock decreased the HSP70 response. In contrast, the effects on the HSP90-level were much less pronounced. Comparing all groups exposed to heat shock with all groups exposed to dive only, analysis of variance indicated that heat shock significantly increased the HSP90 level compared to control, but that the effect of a dive was not significant.

Neither heat shock nor a simulated dive had any significant effect on the intracellular glutathione (GSH) level while the combination of a heat shock and a subsequent dive increased the total GSH level approximately 62 %. Reversal of the exposure order demonstrated a 47 % increase in intracellular GSH levels while a subsequent heat shock seemed to decrease the GSH-level per se and to attenuate the GSH-response to a dive. The different response compared to initial experiment was most probably due to that the initial design determined the GSH immediately after decompression, while the latter design allowed for expression of altered GSH level for 48 hours after decompression. Neither a dive nor a heat shock seemed to have any effect on the glutathione red-ox status.

CONCLUSION: Simulated diving had notable effects on cellular defence mechanisms in human endothelial cells after a prior heat shock, conditions that have been shown to confer protection against endothelial damage due to DCS. The study demonstrated for the first time that a dive had a potentiating effect on the heat-induced expression of HSP70 and suggested that the effects were due to compression or the pressure exposure per se.

SIMULATION OF POST DIVE DECOMPRESSION BUBBLES AT HIGH ALTITUDE

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Aim

The aim of this study is to investigate the differences of bubble dynamics between sea level and high altitude dives and explain why more conservative decompression should be adopted when diving at altitude.

Introduction

Dive tables designed for sea level are not suitable for diving at high altitude. The difference is often explained by the change in surface to depth pressure ratio that is the old Haldanian principle. However, there is no explanation why this fixed ratio cannot be applied to deep saturation diving. This report is an attempt to explain the conservatism of altitude diving in terms of bubble equations.

Method

The simulation of post dive decompression bubbles were accomplished for the same depths at sea level and at altitude (3500 m). The simulation software was developed by the authors based on Van Liew equations for bubble dynamics. The gas exchange is simulated using a perfusion limited, 16 compartment model. The results are compared with the Doppler recordings from altitude diving expeditions at 3412 m. altitude.

Results

Simulations showed that high altitude dives caused bigger sized bubbles, they remain longer in body and the onset time can be long. The ascent rate is also very important as it increases the gap between sea level and high altitude dives. High ascent speeds may cause almost stable bubbles or explosive bubble growth.

Discussion

Late onset time of bubbles are confirmed by altitude diving expeditions in 1991, 1994 and 1997. This is basically due to the decrease in oxygen window at altitude. The oxygen window is estimated to decrease with acclimatization, however there is no data to confirm this impact. A universal decompression method should provide same results for deep saturation and diving altitude; they should all be based on the same set of differential equations but should have different initial conditions.

PRESSURE MODULATION OF Ca²⁺ CHANNELS ACTIVITY MAY ELUCIDATE HPNS MECHANISMS

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BACKGROUND: Professional deep sea divers who are exposed to extreme pressures (1.0 MPa or higher) experience High Pressure Neurological Syndrome (HPNS), which is manifested by various cognitive, motor, sensory, and autonomic nervous system deficits, sleep disorders and changes in EEG. It has been previously suggested that hyperbaric pressure (HP) increases in granule cells the 'transfer function' between synaptic inputs and somatic spike generation, despite an evident reduction of single field excitatory post synaptic potential (fEPSP) amplitude.

We hypothesized that L-type (LTCC) and T-type (TTCC) voltage gated Ca²⁺ channels which are found in the neuron cell body and dendrites, may serve as boosters for the synaptic input at pressure.

METHODS: Rabbit cRNA of L- (Ca_v1.2) or T-type (Ca_v3.2) Ca²⁺ channel subunits combination was injected to *Xenopus laevis* oocytes, followed by 4 days incubation. Two electrodes voltage clamp experiments were performed on the oocytes inside a pressure chamber, helium compressed up to 5.1 MPa (50 ATA) at 22-25°C.

Ca²⁺ was substituted for by Ba²⁺ in the physiological solutions.

RESULTS: HP significantly decreased by $48 \pm 15\%$ (n=6, p<0.05) the maximal Ba²⁺ current in the TTCC already at relatively low HP of 2.5 MPa. In contrast, HP increased by $59 \pm 8\%$ (n=4, p<0.05) the currents in the LTCC under similar conditions. These pressure-induced changes of currents amplitude remained stable for at least an hour, as long as the ambient conditions remained unchanged.

DISCUSSION: The opposite change of maximal current in the two channels demonstrates pressure selectivity. HP augmentation of LTCC maximal currents, together with their known location in cell bodies and proximal dendrites, may corroborate the increase of 'transfer function' between synaptic inputs and somatic spike generation, i.e. boosting subthreshold synaptic potentials to generate action potentials. This indicates an increased excitability in neuronal dendrites rather than in the axons.

HP significantly reduced the TTCC current; hence it is unlikely that they play any role in boosting effect. These low threshold channels are found in the heart and brain areas, where they are involved with bodily rhythmic activity. Taken together, we suggest that HP would reduce the frequency of neuronal pacemakers and thus may cause sleep cycle disorder, motor coordination deficits, slowing of EEG, and even decrement in mental capabilities, all being HPNS manifestations.

CENTRAL NERVOUS SYSTEM DAMAGE AND REPAIR: SPINAL CORD DECOMPRESSION ILLNESS

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Brain and spinal cord injury are major clinical problems that target both the civilian and military population. Although a significant amount of research has been conducted to clarify the pathomechanisms underlying ischemic and traumatic CNS injury, no proven therapeutic interventions have been successful in Phase III clinical trials. Injury mechanisms including excitotoxicity, free radical generation, apoptotic cell death and inflammation have been identified in clinically relevant models of brain and spinal cord injury. Novel therapeutic interventions targeting specific injury mechanisms have provided encouraging preclinical data. In addition, recent neuroscience discoveries have identified mechanisms by which both endogenous as well as exogenous reparative strategies can be introduced to promote synaptic plasticity and improve functional recovery. These studies are leading to exciting treatment strategies using growth promoting factors, cellular therapies and combinational approaches.

Recently, the use of mild hypothermia has been introduced into the experimental and clinical literature regarding brain and spinal cord injury. Numerous preclinical studies have reported that mild hypothermia introduced after an ischemic or traumatic insult is both neuroprotective and leads to improved functional outcome. Indeed, clinical trials targeting patients with cardiac arrest, neonatal hypoxia, and traumatic brain and spinal cord injury have shown some degree of efficacy. Mild hypothermia may be protective by targeting multiple cell signaling cascades felt to be critical in both cell survival as well as cell death. This lecture will cover some of the newest experimental and clinical information targeting injury mechanisms and strategies to both protect and repair the nervous system after injury.

SPINAL INJURY REHABILITATION

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The Queen Elizabeth National Spinal Injuries Unit provides a national service to all patients in Scotland with non-progressive spinal cord injury. The Unit is situated in a major Scottish university teaching hospital.

The Unit admits one hundred paralysed patients annually. The majority have traumatic cord injury and are admitted within forty-eight hours. Surgical fixation, where appropriate, is performed in the Unit and the patients then undergo rehabilitation. Mean length of stay is six months.

The Unit also provides lifelong out-patient care of paralysed patients including outreach clinics throughout the country.

There is a collaborative program of research with teams from major universities in Glasgow and Stirling including assistive technology, stem cell regeneration and psychological projects.

ASSOCIATION RULES OF DCI PATIENT CLUSTERS AND RELIABILITY OF CLUSTERING ANALYSIS

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Aims

We have previously suggested a classification of DCI using multivariate statistics to assess naturally associated clusters of signs and symptoms based on 1929 cases reported by hyperbaric chambers to the Divers Alert Network from 1999 to 2003. The aim of this study is to validate the reliability of the previous work by applying 3 different alternative clustering methods, by comparing the results of two-step clustering analysis with the Perceived Severity Index (PSI) and to approve the characteristics of patient clusters using association rules.

Introduction

With advances in computer technology, data mining techniques are widely used in medical field. Implementation of multivariate statistics is suitable, especially for classifying diseases like DCI. Our recent two-step clustering analysis divided DCI patients into four classes revealing the characteristics of each group. However, further research was needed to be done in order to confirm this pioneering study's results.

Methods

We have repeated the clustering analysis using three alternative clustering methods: K-Means which is an iterative distance-based clustering algorithm, COBWEB, an incremental algorithm based on category utility and EM (expected maximization) clustering. The results of two-step algorithm and PSI classification are compared using Pearson product-moment correlation. Additionally, we used a priori algorithm to find out the association rules which reveal the relations among the signs, symptoms and clusters

Results

The three alternative classification methods yielded similar results. The correlation coefficient between Cluster 1 of two-step clustering (T1) and PSI group 4 (P4) is 0.991345. Other significant correlations are 0.995091 between T2 and P3, and 0.945531 between T3 and P1. By setting the minimum confidence to 0.5, minimum support to 0.35 and number of results to 50, we have found the association rules that confirm the two-step classification structure.

Discussion

We can conclude that PSI and two-step clustering are highly correlated. Additionally, association rules confirm the correct placement of signs and symptoms in each cluster. As a result, cluster analysis is a potential technique that can be used to classify DCI patients according to the characteristic signs and symptoms of the classes. These characteristics may help the differential diagnosis and they can be used for further studies on the relationships between the DCI classes, treatment strategies and outcomes.

THE EXPERIENCE AND NEAR ACCIDENTS AMONG 110 DANISH DIVING INSTRUCTORS, - A QUESTIONNAIRE SURVEY.

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Aims: Obtain information on the diving experience among Danish diving instructors and put this experience in a near accident/accident perspective.

Introduction: Information about circumstances connected to diving accidents is essential in the work to prevent or reduce the consequences of diving accidents. No national register contain information on accidents related to diving. Only hyperbaric chamber treatments are registered..

Methods. A written questionnaire was presented at an instructors seminar in November 2008.. The questionnaires were collected at the end of the seminar. The answers were anonymous.

Results. 110 instructors participated. The median age was 42 years

The diving activity was 71,180 dives by 104 responders to this question. The average instructor had 684 dives within a great span as 2 instructors had less than 50 dives while one instructor had an experience of 4530 dives. 98 instructors performed recreational diving, 32 technical diving, 5 made professional diving and 10 did not indicate their field. 17 participants had more than 100 dives deeper than 40 meters. The most frequent diving activity was 30 dives per instructor in 2008. On average the diver had been active for 17 years.

Concerning equipment 50 used wet suit, 101 used dry suit, 18 used semidry suit and 2 used warm water suit. 92 used computer. 92 used buddy line. The bottles were average 9,8 years old, the regulator 7.9 and the suit 5.3 years. The maintenance and servicing of equipment is performed by the instructor in 88 cases, while 20 instructors leave this to other persons.

Buddy check of equipment al always done by 61, often by 42 and seldom/never by 6.

The breathing gas is air for 106 respondents, 80 use NitrOx, 17 use Trimix and 2 use HeliOx.

Concerning the safety measures, 58 always bring an Oxybox to the site, 40 indicate "often". 67 uses a diving flag every time. 4 dive always alone 46% does not always have a buddy while diving.

Concerning diving accidents and near accidents, 45% have been involved in one or several diving related accidents. Some of the accidents were very serious. However it was not possible to obtain accident statistics, as some incidents were seen by several and nu structured registration was performed.

80 respondents indicate that they are experienced or very experienced in first aid. 56 instructors have been on a first aid course within the last 2 years.

Discussion. The group of instructors is very surprisingly experienced and their career is long. Therefore such seminar is ideal for exchange of experience. As a considerable number of the participants have experienced accidents or near accidents we find it of interest that 46% of the instructors may not have a buddy at all dives, Is also interesting, that 37% of the respondents do not always make a buddy-check. . We assume that the response to this question is given in the cases, where f buddy check is possible.

We conclude, that a seminar for instructors is an ideal platform for discussions concerning diving safety. We got information about the safety culture, but not a quantitative measure of accidents and near accidents.

LONG TERM EFFECTS OF DIVING

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Underwater diving became a practical reality in the first half of the 19th century with improved air pumping technology and the development of a watertight diving dress. It soon became apparent that there were health issues relating to barotraumas and decompression illness which required the institution of preventative measures largely focused on the need for appropriate decompression tables. A century later, central nervous system damage was still recognized as a consequence of decompression illness (1) and there were reports of bone necrosis relating to diving that gave rise to a great deal of concern (2), particularly in the light of the novel technique of saturation diving that was being developed in the Offshore Oil and Gas Industry at that time. In the United Kingdom, however, the level of significant disease (0.18%) from this cause was not found to be sufficient to justify continued radiological screening in divers (3). As continental shelf diving developed in the 1970s it became abundantly clear that there was another major health threat for divers and that was industrial accident. In the 1970s there were 32 diver deaths in the United Kingdom sector of the North Sea giving an incidence which peaked at about 1% of divers working offshore at any one time. Eighteen victims were examined at Aberdeen. Only three had suffered from the classic diving related diseases of barotrauma or decompression illness. Other causes of death were hypoxia, heat stroke, hypothermia and drowning. Six cases were attributed to human error (4). Since then there have been a further six deaths, the last in 1999. This reduction in accidental mortality has been accompanied by a fall in the prevalence and severity of decompression illness, due mainly to the statutory reduction in the permissible pressure exposure in air (5), and in 2005 and 2006 there were no accidents reported that had resulted in more than three days off work in the United Kingdom offshore workforce. While these figures are strongly influenced by the reduction in the number of divers working in the North Sea, they do indicate a very substantial improvement in operational safety.

Within the last decade, the North Sea diving workforce active prior to 1990 has contributed to health surveys in Norway and in the United Kingdom (6,7,8). While there was very little unexpected health impairment in United Kingdom divers, this was not the case in the Norwegian diver workforce in which there was a high level of disability. This difference is likely to have its origins in cross-cultural or cross national psychosocial effects which are unrelated to the practice of diving. Nevertheless, there are indications that oil industry divers have a higher level of health related anxiety than other workers and this may make them vulnerable to somatoform ailments. In addition, both national populations showed evidence of noise induced hearing loss and there was concern expressed regarding lung function in Norwegian but not in United Kingdom divers.

The established long term health effects of diving are auditory and neurological damage following barotrauma or decompression sickness and a predisposition to bone necrosis of the femoral and humeral heads. Industrial accidents and their physical consequences have also been an occupational risk. Recent surveys have indicated, however, that while operational safety is improving, there are other areas which, if targeted, offer an improvement in diver working conditions. The risk of noise induced hearing loss and hand arm vibration syndrome are clearly targets and the risk for chemical exposure is also worth greater attention. Psychological issues, as in any workforce, are very influential and it will be important to foster a correct perception of the actual, as opposed to the perceived, health risks of a diving career in the future.

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CROSS SECTIONAL STUDY COMPARING DEMOGRAPHICS, HEALTH AND DIVE PRACTICES OF RECREATIONAL REBREATHING DIVERS COMPARED TO OPEN CIRCUIT TECHNICAL DIVERS AND RECREATIONAL SCUBA DIVERS.

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Aims

Recreational rebreather diving is becoming increasingly popular but appears to be associated with significantly increased mortality when compared to other forms of recreational diving. This study was designed to look at demographic characteristics, health and diving practices of rebreather divers when compared to both open circuit recreational divers and open circuit technical divers.

Methods

We received 750 responses to a questionnaire posted on the Internet. Seven hundred and twenty two responses were entered into a statistics package, of which 333 were from rebreather divers (RB) (46%), 125 were from open circuit technical divers (Tech) (17%) and 264 were from recreational scuba divers (Scuba) (37%).

Results

There was a significant difference in age between the three type of divers ($p < 0.001$): RB divers were older (42.9 years), than the Tech divers (37.6 years) and scuba divers (41.1), There was a significant difference ($p < 0.001$) in the percentage of male divers in the different groups: RB (93%), Tech (81%) and Scuba divers (80%). There was no statistically significant difference between the groups in terms of BMI, smoking and alcohol consumption, physical activity levels, employment group or marital status.

RB divers had been diving for more years than Scuba and Tech divers; 17.6, 9.8 and 11.2 years respectively ($p < 0.001$). They had carried out a higher number of lifetime dives and did a higher proportion of deeper dives. RB and Tech divers performed significantly higher numbers of dives involving planned decompression, wreck penetration, cave diving, trimix diving or solo diving ($p < 0.001$) than Scuba divers. RB divers carried out a significantly higher number of solo dives than Tech divers.

With regard to medical history, there was a significant increase in the prevalence of PFO in RB divers (4%, 1%, 1% $p < 0.05$) and a decrease in the prevalence of depression (8%, 17%, 17% $p < 0.001$). There was no difference in prevalence of diabetes, respiratory or cardiovascular disease or epilepsy. There was a significant increase in the reporting of previous dysbaric illness (DCI and AGE) between RB, Scuba and Tech divers (26%, 6%, 17% $p < 0.001$).

Conclusion

RB divers are older than Tech divers but no less fit than Tech and Scuba divers. They are more experienced and partake in inherently riskier forms of diving. It appears unlikely that the increased mortality is attributable to the health of rebreather divers but their more extreme forms of diving may be an important factor.

EFFECT OF LONG-TERM INACTIVITY, SHORT TERM REST, BODY POSITION AND LEG MOVEMENTS ON PRESENCE OF VENOUS GAS EMBOLI AFTER DECOMPRESSION FROM HYPERBARIC PRESSURE

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INTRODUCTION: Decompression illness (DCI) has not been reported during extravehicular space activities, despite the fact that similar altitude decompressions in laboratories on Earth are associated with high rates of venous gas emboli (VGE) and DCI. Inactivity in a horizontal position reduces formation of VGE during altitude exposures (1). To confirm this finding subjects in a bed rest study were exposed to hyperbaric chamber dives.

METHODS: Ten healthy male volunteers taking part in a five week bed rest were exposed to three hyperbaric chamber dives; 100 min at 280 kPa with decompression stops for 5 min at 160 kPa, and 15 min at 130 kPa. Control dive prior to bed rest and dive during bed rest were carried out with the subjects resting supine during the whole procedure. In the post bed rest dive subjects were sitting in the chamber and performing squats at regular intervals post decompression. VGE were detected using precordial ultrasound Doppler. All procedures were approved by the local ethics committee.

RESULTS: The dives were carried out without any complications. Two subjects were removed from the statistics; one because he showed no VGE in any condition, and the other because of excessive weight loss during the test period. Of the remaining 8 subjects one had VGE during the Control dive, six had VGE post-decompression during bed rest dive, and five had VGE during the post bed rest dive. Kruskal-Wallis test was significant at $p < 0.05$.

CONCLUSIONS: Compared to the sitting and freely moving subjects supine rest for an hour prior to compression and during the hyperbaric exposure tended to reduce the amount of VGE post-decompression. However, the low bubbling was not maintained during prolonged bed rest. In respect of bubble formation tendency long term bed rest does not appear to mimic the effect of space flight.

(1): Karlsson L et al. Abstract of the UHMS, Kapalua Maui, Hawaii, 2007

UNDERWATER VERSUS SURFACE OXYGEN BREATHING ON POST-DIVE BUBBLE FORMATION IN SCUBA DIVERS.

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Aims

The value of substituting In-Water Recompression with Oxygen (IWRO) for surface Normobaric Oxygen Breathing (NOB), in emergency treatment of decompression sickness has never been studied.

The purpose of this work was to investigate whether IWRO to 6 msw depth is more effective than NOB in reducing venous circulating bubbles following a provocative dive.

Methods

Nineteen military divers performed air diving in open water to 30 msw for 30 min followed by a 9 min stop at 3 msw (MN90 table). Each diver performed 3 dives: one control dive, and two dives followed by 30 min of IWRO or NOB started 10 min after surfacing. Decompression bubbles were examined by a precordial pulsed Doppler.

Results

Bubble count was significantly lower for post-dive NOB than for control dives. IWRO dramatically suppressed circulating bubble formation with a bubble count significantly lower than for NOB or controls.

Discussion-Conclusion

IWRO to 6 msw is more effective in removing decompression bubbles than NOB. This treatment could be worthwhile in situations of “interrupted” or “omitted” decompression, where a diver returns to the water in order to complete decompression prior to the onset of symptoms.

BIPHASIC SPLEEN CONTRACTION DURING APNEA IN DIVERS SUGGESTS CHEMORECEPTOR INPUT

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Introduction: Spleen contraction develops as a response to apnea in humans, but there are conflicting results concerning its initiation. While hypoxia has been suggested to be an important stimulus (Richardson et al 2008) it has also been suggested that spleen contraction develops early during the apnea without any chemoreceptor input (Palada et al 2007). The latter study involved apneas of only 15 s duration, and the present aim was to reveal how spleen contraction develops during apneas long enough to lead to asphyxia.

Methods: Seven apnea divers (6 females and 1 male), with a mean (SD) age of 30(3) years, an apnea training history of 3(1) years, currently training apnea 5(2) h per week volunteered for the study. After a period of 20 min of horizontal rest, apneas of 2 min 30 s duration were performed after a deep inspiration without prior hyperventilation. Every 15 s, from 2 min before until 4 min after apneic episodes, spleen length (L), width (W) and thickness (T) were measured using ultrasonic Doppler (Mindray DP 6600, Atlanta, USA) for calculations of spleen volume using the formula $L\pi(WT-T^2)/3$.

Results: Mean (SD) spleen volume, 207(93) ml, was transiently reduced just after the onset of apnea to 180(79) ml ($P<0.05$), with a further reduction developing progressively across the apnea (Fig 1). The minimum volume, 153 (60) ml ($P<0.001$) appeared just after apnea termination, corresponding with a nadir SaO₂ of 91(2) %.

Discussion: Our results show that during the initial phase of apnea there is a transient spleen contraction, but the main contraction occurs later in the apnea in parallel with the developing asphyxia. The dependence of chemoreceptor input for the maximal development of the response agrees with Richardson et al (2008). We conclude that spleen contraction is biphasic due to different stimuli being involved in early and late phases of its development; While the early contraction is initiated by sympathetic nerve activity related to the onset of apnea, the later phase is likely stimulated by hypoxia, with some influence of hypercapnia.

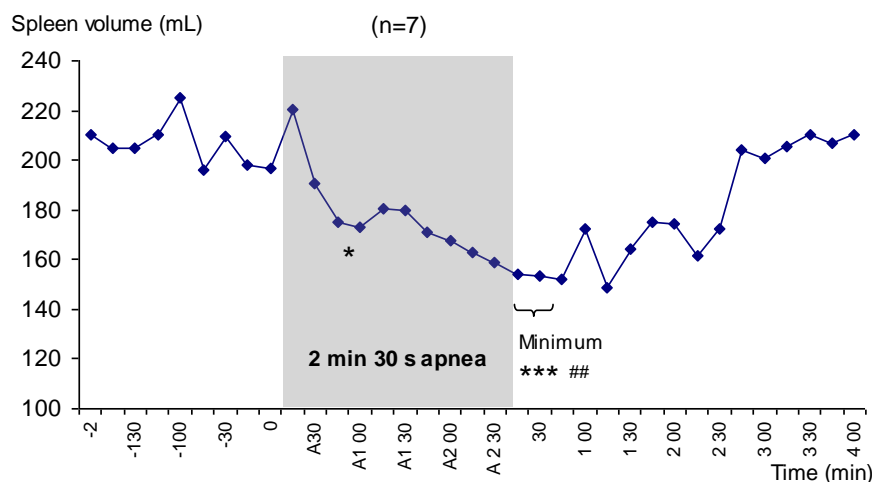


Figure. Mean (N=7) spleen volume prior to, during and after apnea. Difference from pre-apnea volume is indicated by * for $P<0.05$ and *** for $P<0.001$. ## indicates difference from early contraction volume.

References

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EVALUATION OF CRITICAL FLICKER FUSION FREQUENCY AND PERCEIVED FATIGUE IN DIVERS AFTER AIR AND NITROX DIVING.

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Introduction

It is commonly assumed that nitrox diving results in less (perceived) fatigue than air diving. The objective of this study was to verify this assertion and objectively evaluate the physiological effects in these two conditions.

Methods:

217 healthy divers (aged 10 to 66 years) took part in this study that was carried out in Egypt (Sharm-el-Sheikh). No diving restrictions were imposed. Dives were made either using air or nitrox 32% as the breathing gas. The divers were tested before and after the dive either with a "Flicker test" (n=98; air/nitrox=56/42) or a fatigue visual analogical scale (VAS) (n=119; air/nitrox = 67/52). The average depth and time was 23.93 ±6.7m for 42.5 ±8.27 minutes (air dives) and 20.5 ±4.7m for 44.2 ±7.8 minutes (nitrox dives).

Results:

When expressed as a pre-dive percentage, the post-dive answer to the Critical Flicker Fusion Frequency test was significantly increased after nitrox dives (n = 42, mean 103.5% ± 9.07, p<0.05) and highly significantly reduced when using air (n = 56, mean = 93.70% ± 9.90, p<0.001). With regards to the "VAS", there is a highly significant "perceived fatigue" of +30,5% for the air divers (n = 67, mean = 130.5% ± 86.33, p<0.001) and a non-significant difference when using a nitrox (n = 52, mean = 107.6% ± 50.62, p>0.05), when compared to pre dive answers.

Conclusions:

Nitrox seems to have beneficial effects on the post-dive perceived fatigue. These results are corroborated by a more objective measure such as the Critical Flicker Fusion Frequency which shows the same significant tendency when measuring the ability of the brain to discriminate narrow light signals.

DIVING FOR HANDICAPPED PEOPLE IN THE WIDER ADRIATIC REGION

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IAHD Adriatic

ABSTRACT

Scuba diving can be part of rehabilitation or after completed rehabilitation and proper education a leisure activity for individuals with diverse handicaps. Handicaps which permit diving are: asthma, amputation, diabetes mellitus, cerebral palsy, nerve and muscular disorders, sclerosis multiplex, spinal cord injuries etc. The project began in 2002 when three basic programs for individuals with handicap were developed. Program takes into account the specific geographical and socio-economic situation of the wider Adriatic region.

The first program educates individuals to become trainers in the following programs.

The program »Diving as a part of rehabilitation« is created for individuals at the beginning of the rehabilitation as a possible way of motivating for better cooperation in the rehabilitation process and for individuals with considerable handicaps.

The program »Diving through recreation« is carried out as recreation with a diving course for individuals, who are interested in becoming divers. After passing the exam, candidates became certified divers and can participate in organized diving holidays or dive in specialized diving centers. Most of our experience is with divers after spinal cord injury. We haven't registered any complications with involved handicapped divers in the course of implementing the project.

In 2006 Slovenia, Croatia and Bosnia and Herzegovina formed International Societies Association IAHD Adriatic which cooperates with diving and handicapped institutions in the region. Through joint actions all co-founders work with individuals who have a wide variety of handicaps.

Since lack of systematic work and research in the field of diving for handicapped people is symptomatic for all parts of the world and because the Adriatic Sea with its specific characteristics is the most visited destination for divers participating in our programs, we started systematic work concerning diving medicine and diving safety for this population.

We will improve our knowledge through experience and by gathering the data about diving. The data collection is still in progress but we need more information to make reliable conclusions.

(Source: IAHD Adriatic Arhive)

SODA LIME PACKING AND CNS OXYGEN TOXICITY IN CLOSED-CIRCUIT OXYGEN REBREATHERS.

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INTRODUCTION O₂ diving incidents investigated by our laboratory were related to an individual lack of sensitivity to CO₂ (CO₂ retention and poor detection), and to the presence of CO₂ in the inspiratory line of the rebreather. Oxygen toxicity in diving will be more severe if CO₂ is allowed to accumulate in the rebreather, with just 1 kPa of CO₂ in the inspired oxygen having been found to increase the risk of CNS oxygen toxicity in rats. Some of the incidents we investigated were related to improper filling of the soda lime in closed-circuit oxygen rebreathers.

METHODS We therefore studied the effect on CO₂ absorption of overfilling or underfilling the canister, using a continuous flow of 5% CO₂.

RESULTS With a full canister in the Oxyger 57, CO₂ began to rise at 130-160 min, reaching 1% at 240 min and 1.5% at 270 min. Similar results were obtained after a reduction of 100 g in the quantity of soda lime packed into the canister. After a reduction of 200, 300 and 400 g, the rise in CO₂ concentration occurred earlier as a function of the amount of the reduction. The level of CO₂ in the OxyNG 2 began to rise after 250 min with a full canister, reaching 1% at 340 min and 1.5% at 370 min. After a reduction of 100 g, there was a delay in the rise of CO₂, which reached 1.5% at 390 min. However, when the reduction was 200, 300 and 400 g, the rise in CO₂ concentration occurred earlier as a function of the amount of the reduction. For both rebreathers, when the quantity of soda lime was reduced by 200g or more, there was a considerable difference in timing between the two test measurements for each weight reduction, due to variations in channeling. For an excess of soda lime, moderate pressure was applied manually to achieve a full canister plus 300g in the OxyNG 2. The initial rise in CO₂ concentration started early, at 60 min with a full canister plus 300 g compared to 150 min with a full canister; 1% CO₂ was reached at 120 min, compared to 210 min with a full canister.

SUMMARY As the use of rebreathers becomes increasingly widespread in diving, close attention should be paid to proper filling of the soda lime canister.

PULMONARY SIMULATOR FOR WORK OF BREATHING ASSESSMENT

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Aim: R&D of a breathing simulator for characterization and testing of breathing equipment with freely programmable breathing patterns

Introduction: Work of breathing is an important parameter of underwater breathing apparatus, for open circuit diving systems as well as for rebreather systems. In European Normatives like EN14143 (for rebreathers) or EN250 (for open circuit regulators) maximum values are specified. Next to work of breathing, also minimum and maximum pressures for inhaling and exhaling are given. For a correct CE certification the products have to meet the requirements stated in the normatives. State of the art testing and characterization devices can be purchase at ANSTI. Today these machines are standard for regulator and rebreather testing. The systems consist of a chamber that can be pressurized and a motor driven piston for simulating breathing. Due to the motor driven piston the simulated breathing pattern have sinusoidal character, which does not really reflect the physiology of human breathing. The overall system is quite large, heavy and also expensive, thus only few test houses and manufacturers can afford such a unit. The main goal of this project was to develop a cheap and small breathing simulator, that is easy transportable and can be freely programmed to allow simulation of a variety of breathing patterns.

Methods: the core components of our breathing simulator is a rubber bellow with an outer diameter 16 cm. Minimum length is 4cm, but it can be expanded to 40 cm. A 10 W stepper motor is included to drive the bellow via 2 rubber tooth-belts. A differential pressure sensor is integrated the inlet of the bellow to allow recording of inhale and exhale pressures. A 8-Bit RISC microcontroller (ATmega 32, Atmel) operated at 8 MHz handles driving the stepper motor and samples the output of the pressure sensors. All measurement data are stored on SD card and are also transmitted via a Bluetooth serial link. A Graphical User interface was developed under Microsoft visual C++ to allow intuitive programming of the breathing pattern, the breathing minute volume and also the breathing rate. It also allows graphical representation of the measurement data.

Results: A prototype was build up and tested. It has an overall weight of 6 kg. A 20 cells NiMh battery pack (3000 mAh) allows operation of up to 4h.

Conclusion: A novel device for measurement of work of breathing and inhale and exhale pressures was designed and manufactured. This relatively inexpensive unit can be easily deployed under water or in a hyperbaric chamber. The breathing pattern can be freely programmed and is not limited to a sinusoidal form – which allows a more natural simulation of breathing and in consequence the acquisition of more realistic work of breathing data of breathing systems.

EXPERIMENTAL PLATFORM FOR STUDIES ON VPM AND BÜHLMANN DECOMPRESSION ALGORITHMS

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Aims: Mathematical platform for deep analysis of state of the art decompression algorithms

Introduction: Nowadays there exist a wide range of decompression algorithms, which basically can be divided into Haldane models (like Bühlmann) and bubble models like the Varying Permeability Model or the Reduced Gradient Bubble Model.

The present paper describes a mathematical platform for studies on decompression algorithms. This platform is open source, scalable, provides a tool set for deep analysis of present decompression algorithms and functionalities for the validation of improved, optimized or new decompression algorithms.

Methods: The experimental platform supports the Bühlmann ZHL-16 decompression algorithm and the Varying Permeability Model. Both algorithms are in general straight translations of Erik Baker's original FORTRAN code into MatLab R2008a [MathWorks]. MatLab is a numerical computing environment and programming language. MatLab as programming language has the benefit, that single functionalities can be executed isolated or in arbitrary order. Intermediate results, for example the tissue saturation at a certain point in time, can be visualized in 2D or 3D diagrams without the effort of writing code. Timings, bottlenecks and dependencies can be measured or figured out by available libraries. Both models support various levels of conservatism to provide a basis for wide studies. To increase conservatism to the ZHL-16 Erik Baker's Gradient Factors are included. The conservatism level of the VPM is supplied by manipulating the initial critical bubble radius. Furthermore the VPM algorithm uses Boyle's Law to increase the bubble radius during ascent (generally known as VPM-B) to provide additional conservatism. Both algorithms support arbitrary kinds of nitrogen / helium mixes. All constants of both algorithms (like Bühlmann's halftimes, A and B values or VPM's bubble skin compression value, bubble regeneration time, etc.) can be modified.

Results: The platform was developed under MatLab. It supports Bühlmann ZHL-16 as Haldane decompression algorithm and VPM-B as bubble model. The decompression algorithms were validated against several state of the art decompression planners like V-Planner or Freeplanner. Run time comparisons can be performed easily. The source code of the platform will be published under the GNU General Public License (GPL) at SourceForge and possibly on the EUBS database so that the whole diving community can use and develop it further.

Discussion and conclusion: The introduced platform provides a MatLab program to simulate arbitrary dive profiles. Intermediate and final results of the computed decompression schedule can be visualized in 2D and 3D diagrams to provide a basis for deep analysis, study and comparison of haldanian and bubble based models.

HOW TO PREDICT AN UNFAVOURABLE OUTCOME OF A SPINAL CORD DECOMPRESSION SICKNESS IN A SCUBA DIVER

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Introduction

In a case of a scuba diver presenting with a spinal cord decompression sickness (DCS), the physician making the initial checkup might wish being able to make a prognostic of the outcome based upon both the few clinical signs and some elements of the dive.

A gravity index elaborated by Boussuges & al. is taking these findings into account.

We have used this tool for divers presenting in our hyperbaric centre with a spinal cord DCS in order to determine the predicting factors of an unfavourable outcome.

Methods

We studied 41 consecutive cases of spinal cord DCS treated in our hyperbaric center.

They were 32 men and 9 women (mean 40 y.o) mostly experienced recreational divers.

Their mean dive profile was 39 msw maximum depth (19-67 msw) and 40 min total dive time (25-90 min).

Their dive was a successive dive in 10% of the cases.

A procedure mistake was detected in 27% of the cases like excessive ascent speed, uncompleted dive stops, *etc.*

The first clinical signs meanly appeared after 25 minutes. The mean delay between surfacing and beginning of recompression therapy was 3 hours.

Upon admission, paresthesias were the main clinical finding (80%) ; there was a motor deficit in 17% and an objective sensitive deficit in 7% of the cases. A lumbar pain was present in 31% of the cases. The vertebral level was lumbar in 59%, cervical in 34% and dorsal in 7% of the cases. In 39% the symptoms disappeared or lessend with the pre-hospital treatment, remaind unchanged in 42% or increased in 19% of the cases.

The gravity score of Boussuges is based on successive dive, the evolution of symptoms prior to recompression, and the types of symptoms: sensitive, motor or urinary. The mean value of the score of Boussuges in our series was 5, but with extremes from 0 to 20.

Medical adjuvant treatment mainly consisted of IV fluids rehydration, high dose corticotherapy and vasodilators. A great majority of divers (95%) benefited of a Comex 30 recompression table. Generally, that single session was sufficient for 48% of the cases. The others went though a series of complementary sessions (2 to 36) once or twice daily.

Results

At 24 hours, 60% of them showed a clinical improvement, 35% were stabilized and 5% aggravated. At one month 63% showed a complete recovery and 37% an incomplete recovery with more or less sequelae. In the group with a Boussuges score superior to 7 there was a complete recovery at one month in 30% and an incomplete recovery in 70% of the cases. In the group with a Boussuges score less than 7, a complete recovery was noted at one month in 84% of the cases, compared to 16% with an incomplete recovery.

Discussion

We can conclude from our study that the gravity score of Boussuges is an interesting tool to estimate the prognosis of the spinal cord DCS at the initial checkup of the diver. Although it could be enhanced with complementary findings such as the presence or not of the lumbar pain, another neurological gravity score, i.e the ASIA score and biological datas like hematocrit or platelets count.

THE EXPERIENCE AND ORGANIZATION OF THE TREATMENT OF DIVING ACCIDENTS IN TUSCANY

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The islands and coasts of Tuscany are one of the most beautiful diving site of the Mediterranean sea. So the number of dives which are performed in this area is enormous, mainly in summer season. There is an high number of diving accidents, but probably the percentage in comparison of the total number of dives is decreasing. From 1991 to 2008 we have treated in our hyperbaric chambers 651 diving accidents, an average of 53 for year, mainly in the period between May and September. 44% of the total happened in Argentario area, 32% nearby the coast of Leghorn, 22% in the islands close to Leghorn, and the remaining 2% in the south coast of Liguria. For this reason there is an high level of attention on the organization of first aid on the diving site, on the emergency evacuation of the patient. All the first aid facilities have been alerted of the procedures for the treatment to be performed before the transport to the hyperbaric chamber. So, in agreement between all the hyperbaric facilities have been developed the guidelines for the recompression treatment which are derived from the conclusions of the most recent consensus conferences

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DEEP DIVES IN DIFFERENT COUNTRIES

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Aims

The aim is to get a complete oversight over deep dives conducted in different countries.

Introduction

In connection with the discussion around possible long term health effects of diving, the question frequently comes up if possible long term effects may be related to the diving depth. In this discussion it is valuable to have solid data on the total exposures divers have been submitted to. "Deep dive" is a loosely defined term. With regard to petroleum related diving in Norway, the concept has very often been used about dives to more than 180m or 100fathoms depth.

Methods

The collection of data was initiated by systemizing information about research and test dives in Norway. These were mostly found in open reports and published scientific papers. Information on some operational dives was also found in similar ways. But information about other deep dives was much more difficult to obtain and a variety of sources have been utilized, e.g. personal conversation, logbooks, magazines, different books, proceedings and the w.w. web. Very often conflicting information was gathered and it has been necessary to double check. The collection of information from dives in other countries was started later. It very soon appeared that to collect data about all dives deeper than 180 m worldwide is probably not feasible. Therefore the aim was lowered and the depth limit set to 300 m. Also detailed information about operational dives again is difficult to obtain and is yet far from complete.

Results

Table. Number of identified dives/dive-operations sorted by country and 50m maximal depth-intervals

	≥ 300	≥ 350	≥ 400	≥ 450	≥ 500	≥ 550	≥ 600	≥ 650	≥ 700	
Brazil	1	1	1	0	0	0	0	0	0	3
Canada	1	1	0	0	0	0	0	0	0	2
China	1	0	0	0	0	0	0	0	0	1
Germany	3	3	0	6	1	1	1	0	0	15
France	12	1	2	3	5	0	2	0	1	26
Japan	1	0	1	0	0	0	0	0	0	2
Korea	0	0	0	1	0	0	0	0	0	1
Norway	11	6	0	1	1	0	0	0	0	19
CCCP/Russia	1	0	0	0	0	0	0	0	0	1
Switzerland	5	1	0	0	2	0	0	0	0	8
UK	3	1	3	3	2	0	0	1	0	13
US	36	3	1	5	1	0	0	3	0	49
All	75	17	8	19	12	1	3	4	1	140

Discussion

There are definitely more dives than this and the search will continue. Also by including number of divers per dive and duration, number of man dives divedays and other dive exposure measures can be worked out.

GUIDELINE „DIVING ACCIDENT“: GERMAN DIVING AND HYPERBARIC MEDICINE SOCIETY

Developed by Peter H.J. Müller (Chair), Wilfried Beuster, Wolfgang Hühn, Peter Knessl, Hans Joachim Roggenbach, Volker Warninghoff, Wilhelm Welslau, & Jürg Wendling
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Background: The German Diving and Hyperbaric Medicine Society (GTÜM) is responsible for this guideline. The 2008 revision has been developed in co-operation with the Austrian and Swiss affiliates of the GTÜM, who are the ÖGTH and the SUHMS. Both bear the responsibility for national adjustments.

Methods: For the initial creation of the guideline according to the methodological recommendation “Guideline for guidelines” of the German Working Group of medical-scientific Societies (www.awmf.org) in level 1 a representative group of experts was formed in 2001. In level 2 the counselling followed at Consensus Conferences according to the NIH-model in 2003, 2005 and 2008 during the scientific congress of the GTÜM. The group of experts presented the guideline and an international jury acted as directive panel during the discussions of the conferences. After finalisation the new version of the guideline was published each time in peer reviewed medical journals and made available as PDF-file on the websites of the corresponding societies.

Aims: The guideline shall provide assistance during medical decision-making procedures. In connection with quality assurance, it represents a high medical standard, even under the aspect of economical restrictions. It serves information and instructions according to up-to-date evidence to all divers, especially diving instructors and other lay persons providing first aid, physician first responders and emergency physicians as well as paramedics, and all physicians at therapeutic hyperbaric chambers.

Discussion: The format and contents of the latest revision will be presented and the treatment algorithms shall be discussed.

References: Literature & complete guideline available at <http://www.gtuem.org>

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MEASUREMENT OF OH-RADICAL STRESS DUE TO THE COMBINATION OF HYPEROXIA AND PHYSICAL EXERCISE IN CLOSED-CIRCUIT DIVING

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Aims and Introduction

Hyperoxia as well as physical exercise are known to cause oxidative stress. The OH-radical is the most aggressive reactive oxygen species (ROS) under these conditions. While there is evidence that in physical exercise the mitochondria are mainly involved in the generation of ROS and especially the OH-radical, the mechanisms of oxidative stress during hyperoxia might differ. A significant percentage of the generation of ROS and particular of the OH-radical might take place outside the mitochondria, and the Haber-Weiss- as well as the Fenton-reaction and similar pathways are discussed here.

Current knowledge about the production of ROS and particular of the aggressive OH-radical during moderate to severe physical exercise under hyperoxic conditions is limited. However, such a combination is given during closed-circuit oxygen diving and fin-swimming, and the increased mitochondrial turnover in combination with extramitochondrial ROS-generation may lead to additive effects causing relevant oxidative stress.

Methods

We used the measurement of dihydroxylated benzoates (DHB) in urine as a marker of OH-induced oxidative stress in a total number of 135 exposures of healthy subjects: before and after shame as control situation (a) (ambient air; 120min; n=9), to normobaric hyperoxia (b) (100kPa O₂; 110min; n=9), moderate hyperbaric hyperoxia at rest (c) (240kPa O₂; 90min; n=8), severe hyperbaric hyperoxia at rest (d) (280kPa O₂; 30min; n=17), and challenging open-water dives with closed-circuit rebreathers (e) (125-160kPa O₂; 45-54min; n=92).

Results

While under shame conditions (a) DHB-excretion in urine did not change ($3.4 \pm 1.6\%$), moderate to severe hyperoxia led to increases in DHB of $23.1 \pm 1.7\%$ (b; $p < 0.001$), $22.4 \pm 3.2\%$ (c; $p < 0.001$), $21.9 \pm 2.6\%$ (d; $p < 0.001$). After closed-circuit diving DHB-excretion in urine was increased by $66.3 \pm 2.7\%$ (e; $p < 0.001$).

Discussion

We conclude from these data that hyperoxia at rest leads to significant increases in the excretion of DHB in urine as a marker of OH-induced oxidative stress. However, even severe hyperbaric hyperoxia at rest causes much lower OH-stress than the combination of moderate hyperoxia plus physical exercise.

IMPLEMENTATION OF A DIVE COMPUTER USING MOBILE PHONES: THE DIVEPHONE

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Aims

The aim of this project is to design and implement a diver carried computer using a mobile phone inside a pressure and waterproof housing in combination with a series of external microcontroller based electronic modules that can measure pressure, temperature, ppO₂, environment and physiological variables and relay all data to the mobile phone wirelessly.

Introduction

Diver carried computers are basically designed to provide safe decompression profiles and their function may include measurement of tank pressure, ambient temperature, theoretical computation of acute oxygen poisoning as well. In case of rebreathers, they can also measure the ppO₂ and control gas mixture composition. All of the units in the market are based on embedded systems where the user can not reprogram the units except entering limited number of dive parameters such as the content of cylinders and the conservatism of the computation.

Methods

The Divephone (*patent pending*) system consists of an underwater instrumentation unit designed around a microcontroller (*Microchip, PIC18*) and a mobile phone (Nokia N95, Nokia S60 Series, Hp IPAQ 6815). The external module has two models: basic and technical. Basic module is wet contact activated, measures only pressure and temperature and transmits the data wirelessly to the mobile phone. Technical module has two piezo switches and also a wet connector to accommodate input/outputs from external devices such as O₂, CO₂ and humidity sensors and solenoid actuators. The external modules are buried in a pressure/water proof housing tested to 150 m. The mobile phone is carried in a housing made of transparent polycarbonate to allow infrared communication with the external module as well. Five different decompression models are programmed including: US Navy, Bühlmann, VPM, Continous Compartments and a physiological model. All models have gas mixture selection, altitude and gas switching options as well.

Results

The Divephone basic module is tested and benchmarked successfully with 7 existing dive computers in the market up to 42 m in open water conditions. The technical module is only tested in decompression chamber. The units are implemented on mobile phones with Symbian and Windows Mobile operating systems.

Discussion

The Divephone system has the advantage of rapid model implementation and deployment, ability to run multimodel decompression procedures, user specific screen layout through the application of “skins”, very large data logging capacity using external SD Cards and all the advantages of mobile phones including sending post dive SMS messages in case of preset rules for emergencies, automatic wireless transfer of dive logs to research centers via internet and/or to dive center management services. Future projects target Mac OS and the use of photo and video recording, music and film playing capacities of mobile phones to entertain divers during long decompression stops as well. The Divephone platform is further expandable with additional modules to capture a wide variety of information such as oceanographic parameters like salinity, conductivity and dissolved oxygen

EXHALED NITRIC OXIDE (NO_{exp}) MEASUREMENTS AS A NONINVASIVE MARKER OF PULMONARY O₂ TOXICITY SUSCEPTIBILITY IN HUMANS

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Introduction: Pulmonary function tests such as forced vital capacity (FVC) and diffusion capacity for CO (D_LCO) are used to detect pulmonary O₂ toxicity in humans; however, they correlate poorly with pulmonary O₂ toxicity symptoms and cannot predict susceptibility to pulmonary oxygen toxicity. Recently, measurements of expired nitric oxide (NO_{exp}) have been studied as an exhaled marker of airway inflammation in a variety of lung diseases, but the importance of NO_{exp} as a marker of the oxidative stress response of the lungs is unclear. Animal experiments have suggested that endothelium nitric oxide synthase, which is one of the constitutional enzymes that produce NO in the lungs, may be protective against pulmonary oxygen toxicity, whereas neuronal nitric oxide synthase appears to play a dominant role in the development of pulmonary oxygen toxicity.

Aims and Objectives: To measure normal day-to-day individual variability in pulmonary function and NO_{exp} and contrast these measurements with pulmonary function, NO_{exp}, and pulmonary oxygen toxicity symptoms following hyperbaric oxygen (HBO) exposures. The aim of these comparisons is to determine if NO_{exp} levels can predict or provide an early indication of pulmonary O₂ toxicity susceptibility in humans.

Methods: Pulmonary function (FVC, FIVC, FEV₁, D_LCO), and NO_{exp} baseline measures were collected from eight U.S. Navy trained divers twice a day (am and pm) for 5 days prior to conducting the HBO exposures. NO_{exp} was measured using a chemiluminescence NO analyzer (Sievers NOA 280i) at 5 different expired flow rates between 50 and 250 ml/s and used to determine alveolar NO concentration (C_ANO) and maximum airway wall flux of NO (J'_{awNO}) using a two compartment model. The HBO exposures consisted of a 6 hour and 8 hour dry resting exposure breathing 100% O₂ at 202.65 kPa in a hyperbaric chamber. The 6 and 8 hour dives were performed on consecutive days. A single 15 min air break was incorporated during the mid point of each HBO exposure. Pulmonary function and NO_{exp} was measured immediately prior to each dive, 10 minutes post dive, and then daily for 3 days after the dive or until complete recovery of pulmonary function. Only subjects who showed no decrements in pulmonary function following the 6 hr exposure conducted the 8 hr dive. A decrement in pulmonary function for an individual was considered significant if one or more of their pulmonary function tests (either FVC, FIVC, FEV₁, or D_LCO) fell more than two standard deviations below their mean baseline value for that test.

Results: Baseline individual mean and coefficients of variation for NO_{exp} at 50 ml/s (NO_{exp50}) ranged from 19 to 59 ppb (group mean 34 ppb) and 8 to 17% (group mean 13%), respectively. All eight subjects showed significant decreases in NO_{exp50} levels immediately following the 6 hr exposure (group mean change = -55%, p<0.001) that returned to normal in the majority of divers (6) by the morning after the dive. Four subjects with the lowest baseline NO_{exp50} and lowest post dive NO_{exp50} had clinical symptoms of pulmonary O₂ toxicity and showed significant decreases in pulmonary function immediately following the 6 hr exposure. In the four subjects who also conducted the 8 hr exposure, 3 subjects had pulmonary function deficits immediately post dive and all showed greater decreases in NO_{exp50} (group mean change from baseline = -63%) than following their 6 hr dive (p<0.01). The two compartment model analysis showed a significant acute decrease in J'_{awNO} (p<0.001) with no change in C_ANO (p=0.995) immediately post dive.

Discussion: The finding that individuals with the lowest post dive NO_{exp} were the first to develop pulmonary O₂ toxicity symptoms and deficits in pulmonary function from the HBO exposure and that individuals with a high baseline NO_{exp} appear to be less susceptible to pulmonary O₂ toxicity suggest that NO_{exp} may be a useful noninvasive marker of pulmonary O₂ toxicity susceptibility in humans. Further, large scale, studies are needed to confirm this finding. (Sponsored by the Office of Naval Research through the ILIR Program).

EXHALED NITRIC OXIDE LEVELS AT 1500 METRES IN AN ALTITUDE SIMULATOR

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Introduction

Exhaled nitric oxide (NO) is known to decrease under acute, moderate to severe hypoxic conditions - for example during acute exposure to high altitude (Guzel *et al.*, 2000; Hoit *et al.*, 2005; Brown *et al.*, 2006, Donnelly *et al.*, 2009). However, the observed decline in exhaled NO at altitude is still not fully understood. Furthermore, there is little information on what –if anything- happens to the different lung compartments at altitude (Hoit *et al.*, 2005).

Methods

We simulated altitude at 1500m compared to a ground level of 70m, using the University of Aberdeen's altitude (hypobaric chamber). Measurements of exhaled nitric oxide (NO) were made at both altitudes and after hypoxia reversal (using 100% supplemental oxygen) at 1500m. The NiOX® monitoring system was used in subjects (n=20) at flow rates of 100ml/s and 200ml/s. Flow-independent parameters were estimated using multiple flows in the Tsoukias Method. Oxygenation was assessed using pulse oximetry estimation of arterial oxygen saturation (SaO₂).

Results

The NiOX ® did not operate reliably at altitudes above 1500m which are beyond its design specification. Altitude resulted in hypoxia levels of an SaO₂ of 92% (p=0.001) and exhaled nitric oxide decreased (FeNO p=0.001 and VNO p=0.001 at 200ml/s, VNO p=0.002 at 100ml/s). Reversal of hypoxia demonstrated a further decrease in NO (FeNO p=0.03 100ml/s and VNO p=0.02 200ml/s). Maximal airway flux (J_{aw}NO) decreased significantly (0.036).

Summary

This study supported previous research and also showed that exhaled NO appears to decrease in the airway compartment and not in the alveoli. Reversing hypoxia does not restore levels of exhaled NO. However due to small numbers, more research is required.

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ALTERATION OF THE ALVEolocAPILLARY MEMBRANE AFTER A DIVE WITH A PO₂ OF 130 KPA.

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Objectives

It is known that breathing gasses with raised partial pressure of oxygen (PO₂) of more than 50 kPa can cause pulmonary damage. This can reduce both vital capacity and diffusing capacity for carbon monoxide (DL_{co}). Generally, changes in DL_{co} represent alterations in the alveolocapillary membrane (D_m) and/or the pulmonary vascular blood volume (V_{cap}). Applying diffusing capacity for nitric oxide (DL_{no}) a change solely of the alveolocapillary membrane can be measured. Therefore, using both DL_{co} and DL_{no} it is possible to differ between an alteration of D_m, V_{cap} or both. Aim of this study was to see if a dive with a PO₂ of 130 kPa will alter the alveolocapillary membrane.

Methods

18 healthy, non-smoking military divers (24.5 ± 2.5 yrs, 181.3 ± 6.0 cm, 85.56 ± 8.41 kg) were included in this study and were all fit to dive. They used a closed circuit rebreather (LAR VII, Draeger®) and breathed 100% oxygen. Maximal pressure at depth was 130 kPa (3 msw, PO₂ = 130 kPa) and dive-time was 60 minutes. Before (pre), immediately after (post) and four hours after the dive (post + 4) DL_{co} and DL_{no} were measured according to the ATS/ERS guidelines. DL_{co} was standardized to a hemoglobin concentration of 9.1 mmol/L. Also alveolar volume (VA), transfer coefficient for CO (K_{co}) and NO (K_{no}) were quantified. DL_{no}, K_{no}, D_m and V_{cap} were measured with Vmax MS-PFT Pro, DL_{co}, K_{co} and VA with the Vmax Encore (both Cardinal Health, Houten). Calibration was done according to the manufacturers' guidelines. Divers were not allowed to drink coffee, eat or exercise within one hour before any measurement. All participants were male.

Results

	Pre	Post	Post + 4
DL _{co} (mmol/min/kPa)	11.4 (1.6)	10.4 (1.9)	10.2 (1.5)
K _{co} (mmol/min/kPa/L)	1.57 (0.18)	1.48 (0.24)	1.42 (0.22)
DL _{no} (mmol/min/kPa)	57.32 (4.89)	55.63 (4.42)	52.65 (4.15)*
K _{no} (mmol/min/kPa/L)	8.32 (0.60)	8.20 (0.56)	7.88 (0.56)
DL _{no} /DL _{co}	4.91 (0.46)	5.13 (0.36)	5.38 (0.57)
VA (L)	7.257 (0.566)	7.081 (0.584)	7.213 (0.499)
D _m (mmol/min/kPa)	29.10 (2.48)	28.24 (2.24)	26.72 (2.10)*
V _{cap} (ml)	98.6 (11.9)	95.1 (11.9)	87.4 (10.2)*

Table 1: Mean (± SD) before (pre), immediately after (post) and four hours after (post + 4) a dive with a maximal PO₂ of 130 kPa (N=18; *=p<0.05 compared to pre)

We found a statistical significant reduction of the mean value for DL_{no}, D_m and V_{cap} four hours after the dive but not for DL_{co}, K_{co}, K_{no} and VA.

Conclusion

In our study group DL_{no} and D_m were significantly reduced. Therefore, we can conclude that the alveolocapillary membrane resistance was increased after an oxygen dive for 60 minutes with a PO₂ of 130 kPa.

CROSS-SECTIONAL STUDY OF PULMONARY FUNCTION IN FIGHTER PILOTS COMPAIRED TO NON-FIGHTER PILOTS AND DIVERS.

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AIMS: To assess the environmental factors causing changes in pulmonary function

INTRODUCTION: Possible mechanisms for changes in pulmonary function in divers include exposure to hyperoxia, decompression, increased gas density and a shift of blood into thorax from the extremities caused by immersion. Interestingly, some of similar environmental situations are observed in fighter pilots. On air combat maneuver they are suffering from hyperoxia due to high concentrate oxygen breathing, decompression to high altitude and central vascular engorgement produced via G-suit inflation and anti-G straining maneuver. To evaluate the relative contribution of environmental factors on changes in pulmonary function, we investigated pulmonary function in fighter pilots and non-fighters pilots measured at aviation medical examinations, and divers at annual medical check up.

METHODS: The subjects were healthy, male, active-duty uniformed 22 fighter pilots, 88 non-fighter pilots, and 50 divers at the age of over 40. Nine fighter pilots (41%), 23 non-fighter pilots (26%) and 29 divers (58%) were current smokers. Measurements of lung function included vital capacity, forced expiratory lung volumes, and maximal expiratory flows. The measurements were taken on a dry-sealed type spirometer (CHESTAC-33 model; Chest Ltd., Tokyo, Japan). The predicted values with which we compared the subjects' results were from the Japanese respiratory society.

RESULTS: The vital capacity as % predicted in fighter pilots (99.7 +/- 9.1 %: mean +/- SD) was significantly low compared to non-fighter pilots (104.9 +/- 10.5 %) and divers (106.2 +/- 13.9 %). Mean value of forced expiratory volume in one second as % predicted in fighter pilots was 3.7% lower than that in non-fighter pilots, although there was no significant difference between them. Forced expiratory flow rate at 75% of forced vital capacity (FEF_{75%}) in divers (1.3 +/- 0.4 L/sec) suggested low values compared to fighter pilots (1.6 +/- 1.0 L/sec) (p=0.068) and non-fighter pilots (1.5 +/- 0.6 L/sec) (p=0.057).

DISCUSSION/CONCLUSION: While the lower values of vital capacity in fighter pilots were observed, the finding that they had no decrement of FEF_{75%} indicated that the environment in air combat maneuver, such as hyperoxia due to high concentrate oxygen breathing, decompression to high altitude, and central vascular engorgement produced via G-suit inflation and anti-G straining maneuver, might not contribute small airways dysfunction that could be observed in divers.

S100B AS A BIOMARKER FOR NEUROLOGICAL DCS

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Aims: Examine whether s100B is valid as a diagnostic marker for neurological decompression sickness (DCS) and to examine a possible correlation between degree of damage (bubble grade) and serum s100B concentration.

Introduction: Possible long-term health effects (LTHE) of diving have been discussed for several years, and two international consensus conferences on LTHE of diving have concluded that diving might disturb and impair central nervous system (CNS) functions. Bubbles can be observed following almost all decompressions, but although the bubbles are considered to be the cause of DCS, bubbles per se are of no diagnostic value in individual cases. It is generally assumed that localized gas bubbles are responsible for all DCS incidents in the CNS, and it has been shown that the pathological effects of bubbles may cause a mechanical disruption of the tissue, for example the endothelium. Gas bubbles can cause changes in barrier permeability even in the absence of clinical manifestations of DCS. S100B is a glial-derived protein that is a well established biomarker reflecting CNS injury and prognosis for recovery, and high sensitivity enable us to detect mild brain injury. **Methods:** A total of 19 rats (Sprague Dawley, females) were divided into two groups, one control group and one dive group. 11 rats underwent a simulated dive in a 20 L hyperbaric chamber. The animals were compressed (200 kPa/min) to 700 kPa and decompressed (50 kPa/min) after 45 min. After decompression all rats were scanned for bubbles with ultrasound/Doppler and the amount of bubbles present in pulmonary artery was graded from 0-5 according to a previously described method Eftedal et al. The amount of s100B in serum after the dives was tested by ELISA. Bubble grade were compared to s100B protein concentration.

Results: A trend of difference in s100B concentration between the decompressed rats and the controls were found, but this is not statistical significant. However, it was a significant difference in protein concentration between the rats estimated to have a high bubble grade and the rats with no bubbles or a low bubble grade.

Discussion: This project indicates a higher level of s100b in serum of rats with high bubble grades compared to controls. The correlation between bubble grade and an increase in serum protein level of s100B indicate that this protein may be used as a biomarker for neurological damage caused by decompression. However, further research is needed to reveal the mechanisms in CNS caused by diving

SPLEEN CONTRACTION IN ACCESSORY SPLEENS DURING APNEA IN DIVERS

Angelica Lodin, Matt X. Richardson, Erika Schagatay

Introduction

Spleen contraction in human apnea divers results in elevated circulating erythrocyte levels which may prolong apneas (Schagatay et al 2001). Spleen volume during rest may vary between 200 ml and 600 ml in divers, with the highest values observed among the best divers. Accessory spleens, usually small and without known function, have been reported to occur in approximately 10% of the normal population (Halpert and Gyorkey 1959).

Methods

In a series of screening spleen measurements on 28 apnea divers, we observed 3 cases of accessory spleens; two cases of “double spleens” i.e. two spleens with shape and size within the normal range, and one case of a male diver with one normal and two small spherical accessory spleens. In order to determine the possible contractile function of these accessory spleens, two of these divers performed maximal apneas during rest while spleen volumes were measured using ultrasound. Spleen length (L), width (W) and thickness (T) were measured using ultrasonic Doppler (Mindray DP 6600, Atlanta, USA) for calculations of spleen volumes using the formula $L\pi(WT-T^2)/3$.

Results

In a female subject with one accessory spleen, resting spleen volumes were 164 ml and 53 ml respectively. Her spleens responded to 2 min 25 s of apnea by decreasing to 77 ml and 19 ml respectively, thus the total spleen volume reduction was 121 ml. In a male subject with 2 small accessory spleens with maximal diameters of 3,3 and 3,1 cm, estimated spleen volumes were 233 ml for the main spleen, and 10 and 9 ml for spleens no 2 and 3, respectively. The main spleen had contracted to 100 ml volume after 4 min 15 s of apnea, and the small spleens decreased in volume by approximately 50%. All spleens returned to pre-apneic volume within 13 min.

Discussion

The main observation is that accessory spleens may be functional and contract during apnea initiated by similar mechanisms as the main spleen, and likely contribute to elevation of circulating erythrocyte levels. When accessory spleens of a significant size are present in humans their volume reduction should be taken into account when estimating the total volume of spleen erythrocytes ejected during apnea. The possible development of spleen volume and the volume of such accessory spleens as a result of apneic training warrants further investigation.

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THE EFFECT OF PRE-DIVE EXERCISE MODE ON POST-DECOMPRESSION VENOUS GAS EMBOLI.

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Introduction

The effect of pre-dive exercise on post-decompression venous gas emboli (VGE) is contentious. The exact timing and exercise mode that afford the best protection, if it exists, remain uncertain. Differences in dive profiles and conditions, exercise mode and intensity, time between dives, and Doppler monitoring methods and durations have made it difficult to compare results between studies. The aim of this study was to examine the effect of changing the exercise mode from low impact and high intensity, used in a previous study, to higher impact and lower intensity in the present study, whilst preserving other study variables, on post-decompression VGE.

Methods

The study was approved by the Ministry of Defence Research Ethics Committee and complied with the Declaration of Helsinki. Fifteen male volunteers possessing dive medicals, mean age 36.5 (range 22 – 53), performed three identical hyperbaric chamber air dives to 18 m, with a bottom time of 100 min, decompression stops at 6 m for 5 min and at 3 m for 15 min and an ascent rate of 15 m/min. Two of the three dives were conducted with prior exercise bouts at 24 or 2 h; a dive with no prior exercise formed the control. The order in which the dives were conducted was randomly allocated. The exercise regimen consisted of jogging on the spot for one minute followed by ten star jumps, repeated for a total of 40 min. There was a period of at least 7 days between dives and no exercise was permitted for 48 h before a dive/exercise bout. Participants were fitted with Polar heart rate monitors and after a warm-up period were asked to aim at 70 % of their theoretical maximum heart rate ($220 - \text{age}$) for the exercise period. Post-dive pre-cordial Doppler monitoring began within 2 min and was carried out every 5 min for the first 30 min and every 15 min thereafter for 180 min (or longer if a peak had not been reached). Venous gas emboli were scored using the Kisman-Masurel (KM) scale.

Results

There were no cases of decompression illness in any of the participants. There was a non-significant decrease in resting median peak KM score, from 3 for the control dives to 2 for those dives with pre-dive exercise at either 2 or 24 h (Friedman test). Median resting KM scores over 180 min for each condition were ranked and compared by ANOVA, with a significant decrease demonstrated for pre-dive exercise at 2 h versus control ($P < 0.05$ *post hoc* Tukey's test). Pre-dive exercise at 2 h also showed a significant reduction in the resting Kisman Integrated Severity Score (KISS) over 180 min compared to the control ($P < 0.0434$, Wilcoxon sign-ranked test). Changing the exercise mode from that of low impact and high intensity used in a previous study conducted in our laboratory resulted in significantly lower median resting KM scores over 180 min when comparing pre-dive exercise at 2 h ($P < 0.0058$, Mann Whitney U).

Discussion

The results suggest that the exercise mode used in this study reduced post-decompression VGE over 180 min when conducted at 2 h, but not 24 h pre-dive. A previous study in our laboratory conducting high intensity exercise on a cycle ergometer, at 24 or 2 h prior to diving, demonstrated a non-significant increase in median peak KM Doppler scores, with VGE remaining significantly longer after pre-dive exercise than in the no-exercise control. The present study demonstrated that changing the exercise mode to higher impact and lower intensity showed benefit in terms of decreasing total VGE when conducted at 2 h prior to diving. This study suggests that the choice of pre-dive exercise mode at 2 h affects post-decompression VGE.

THE EFFECT OF PRE-DIVE EXERCISE MODE ON POST- DECOMPRESSION VENOUS GAS EMBOLI

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Introduction

The effect of pre-dive exercise on post-decompression venous gas emboli (VGE) is contentious. The exact timing and exercise mode that afford the best protection, if it exists, remain uncertain. Differences in dive profiles and conditions, exercise mode and intensity, time between dives, and Doppler monitoring methods and durations have made it difficult to compare results between studies. The aim of this study was to examine the effect on post-decompression VGE of changing the pre-dive exercise mode from low impact and high intensity, used in our previous study (1), to higher impact and lower intensity, whilst preserving other study variables.

Methods

The study was approved by the Ministry of Defence Research Ethics Committee and conducted in accordance with the Declaration of Helsinki. Fifteen male volunteers comprising RN divers and QinetiQ staff possessing dive medicals, mean age 36.5 (range 22 – 53), performed three identical hyperbaric chamber air dives at the QinetiQ Hyperbaric Medicine Unit, Royal Hospital Haslar, Gosport, UK. The dives were to 18 m, with a bottom time of 100 min, decompression stops at 6 m for 5 min and at 3 m for 15 min and an ascent rate of 15 m/min. Two of the three dives were conducted with prior exercise bouts at 24 or 2 h; a dive with no prior exercise formed the control. The order in which the dives were conducted was randomly allocated.

The exercise regimen consisted of jogging on the spot for one minute followed by ten star jumps, repeated for a total of 40 min. No pressure changes (flying or diving) were permitted for 7 days before commencing the trial: there was a period of at least 7 days between dives and no exercise was permitted for 48 h before a dive/exercise bout. Participants were fitted with Polar heart rate monitors and after a warm-up period were asked to aim at 70 % of their theoretical maximum heart rate (220-age) for the exercise period.

Post-dive pre-cordial Doppler monitoring was carried out using a continuous wave Techno Scientific Doppler Bubble Monitor (TSIDBM 9008). Monitoring began within 2 min and was carried out every 5 min for the first 30 min and every 15 min thereafter for 180 min (or longer if a peak had not been reached). Venous gas emboli were scored using the Kisman-Masurel (KM) scale (2) and the Kisman Integrated Severity Score (KISS) (3) calculated to give a linearised measure of VGE up to 180 min post dive.

Results

There were no cases of decompression illness in any of the participants. No significant difference was detected in the mean percentage maximum heart rate for the exercise at 2 or 24 h prior to diving, 70.7 % and 70.1 % respectively, or between the first and second exercise bouts, 71.0 % and 69.8 % respectively.

The median peak KM score decreased from 3 for the control dives to 2 for those dives with pre-dive exercise at either 2 or 24 h, but this was non-significant (Friedman test). Pre-dive exercise at 2 h resulted in a significant reduction in the mean resting KISS compared to the control (11.3 versus 17.2, $P < 0.04$, Wilcoxon sign-ranked test).

Changing the exercise mode demonstrated a difference in VGE between prior exercise conducted at 2 h, but not at 24 h or in controls. The jogging/star jump exercise used in the present study resulted in significantly lower mean KISS (11.3 versus 21.8, $P < 0.04$) and median KM scores ($P < 0.006$, Mann Whitney U) compared to the high intensity cycling exercise used in our previous study.

Discussion

The results suggest that the exercise mode used in this study reduced post-decompression VGE in the period up to 180 min when conducted at 2 h, but not 24 h pre-dive. The previous study in our laboratory conducting high intensity exercise on a cycle ergometer, at 24 or 2 h prior to diving, demonstrated a non-significant increase in median peak KM Doppler scores, with VGE remaining significantly longer with pre-dive exercise than in the no-exercise control. The present study demonstrated that changing the exercise mode to higher impact and lower intensity (jogging/star jumps) showed benefit in terms of decreasing VGE when conducted at 2 h prior to diving.

Conclusion

This study suggests that the choice of pre-dive exercise mode at 2 h affects post-decompression VGE.

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QI-DIVER PROJECT: ACUPUNCTURE AND TRADITIONAL CHINESE MEDICINE (TCM) IN FREEDIVING PERFORMANCE ENHANCEMENT

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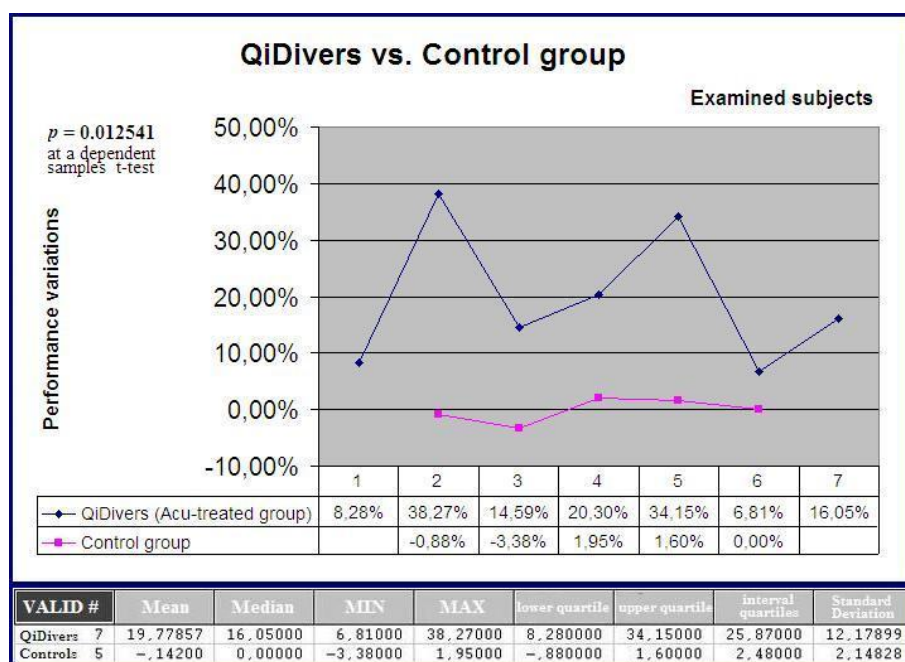
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Goals: To verify the improvement in apnea times, if any, induced by acupuncture and other traditional chinese medicine (TCM) methods among elite breath-hold divers.

Methods: All subjects volunteered and signed an informed-consent form for the study. Twenty volunteers were enrolled (15 males and 5 females), aged 23 to 56 yr (39.2 +/- 8.69, mean and SD differences), height 162 to 184 cm (173.31 +/- 6.49) and weight 52.5 to 95 Kg (72.13 +/- 10.92), apnea experience: advance-elite. The tests of static apnea were completed in Padua (Italy) from February to April '09. Acupuncture-divers (QiDivers) received: guasha and cupping at Huatuojiayi acupoints, acutaping and hot capsicum patches at the 'lower warmer' area of the TH meridian and microstimulation for extended periods (up to 7 days) at Shenmen auricular point and at the following points of the TCM nomenclature: BL-13, CV-17, GB-41, GV-13, GV-19, KI-6, LU-6, LU-7, LU-9, PC-9, TH-3, TH-5, TH-7. In our single-blind study we used an ABCD-like (Adjustable Biased Coin Design) randomization between Control and QiDiver group. Measurement comprised: Arterial Pressure, Heart Rate pre and post test, apnea times. Eight subjects could not complete the test for a variety of causes, unrelated to acupuncture. Complete data were collected in twelve subjects (8 males and 4 females), (QiDiver group n=7 and Control group n=5).

Results: In QiDivers, static apnea times increased by 19.78% +/-12.18% SD (ranging from 6.81 to 38.27 % improvement), while the control group recorded 0.14% +/- 2.15 SD, ranging from -3.38 to + 1.95%. The difference between the two groups was significant ($p < 0.013$).

Discussion: Best results were obtained in QiDivers, especially up to 7 days after the initial treatment, at a time when at least 65% of long term acupuncture needles were still in place since the beginning of the acupuncture insertion.



UNDERWATER NAVIGATION AND COMMUNICATION: A NOVEL GPS/GSM DIVING COMPUTER

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Aims: Technical and recreational diving computer extension to provide navigation assistance and an interface to communicate with the outer world.

Introduction: One important issue for divers is orientation. Till now divers use a watch, depth gauge and compass for navigation if visual orientation points fail (caused by e.g. bad visibility or no contact to the sea bottom). Recreational divers have to navigate, if they want to dive individually without dive guide. Special forces are often dependent on precise navigation to fulfil their mission – which is difficult especially in the presence of currents.

The safety of a diver is increased, if there is the opportunity to communicate with the outer world. Especially in critical situations (like unexpected strong currents, the lost of the diving boat or decompression gas) this may be of vital importance.

The present paper describes a new kind of diving computer, which provides navigation and communication assistance via GPS and GSM. The basic idea is to provide a diving computer / buoy system, which is small enough to fit into a pocket of a BC. If there is a need of navigation assistance or communication via GSM, the small buoy can be set. If the buoy reaches surface, it connects to GPS and GSM. Predefined SMS can be sent and direction and distance to previously predefined GPS-Coordinates can be shown. The buoy is connected to the diving computer via a thin cable on a reel.

Methods: Core component of the diving computer is a low power Atmel Atmega Atmega644p 8 Bit RISC microprocessor [Atmel] (4 kbyte SRAM, 2 kbyte EEPROM, operated at 8 MHz). A digital temperature and pressure sensor is integrated in the design [MS5541B, Intersema]. A 128x64 matrix display [EA DOG-M, Electronic Assembly] is used to visualize all dive computer specific parameters, plus distance and point of the compass to a preset destinations. The dive profile is continuously tracked and stored into an external memory chip [AT25DF321, Atmel]. To provide GPS and GSM functionality in one small device the combined GSM/GPS module GM862 from Telit was chosen and is integrated in the buoy system. The overall low power consumption allows powering the whole diving computer via a single AA battery. The buoy system is powered by a LiIon cell phone battery (1800mAh).

The communication between diving computer and buoy system is established via serial communication. A software schedule was implemented to handle decompression calculation and GPS/GSM communication quasi in parallel.

Results: A first prototype was build up. The diving computer is housed in an aluminum case, filled up with silicon gel. The buoy system is housed in a Drybox (specified to 50m). The Drybox has an inner size of 5cm x 10cm. Diving Computer and buoy system are connected to each other via a 40m long cable and a waterproof IP68 connector [Fisher Connectors]. The whole device is small and handy, so that it can be stored in the pocket of a regular BC if it is not used.

Discussion and conclusion: The introduced prototype combines a technical mix-gas diving computer with a GPS driven navigation system and a GSM. It allows easy navigation underwater. The buoy can be set, whenever GPS/GSM is necessary or it can be continuously dragged behind the diver (f.i. in a country where a buoy is mandatory anyway). Several test dives were successfully conducted in the Mediterranean sea. Extensive field tests will be done by the state police of Italy in July.

REBREATHING DIVING SAFETY: ALTERNATIVE CONTROL DEVICE

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Aim: adding redundancy to electronically controlled rebreathers by including additional self mixing semi closed and manual operation modes and a dedicated trimix decompression computer.

Introduction: Rebreather diving systems offer many advantages like high gas efficiency, bubble free diving and warm breathing air and become more and more popular among technical and nowadays also recreational divers. Closed circuit rebreathers (except closed circuit oxygen rebreathers that are limited to a maximum operational depth of 6m) use O₂ sensors to measure the partial pressure of O₂ inside the breathing loop. Metabolized O₂ is added to the loop either manual or with a constant mass flow orifice (Manual Closed Circuit Rebreather, MCCR) or electronically with a solenoid (Electronically Closed Circuit Rebreather, ECCR). Semi closed rebreathers (SCR) that are based on a constant mass flow do not require pO₂ measurement for a safe operation. This paper details a safety device, which allows, in the case of a failure of the main control electronic/solenoid/sensors of an ECCR a complete shut off of the gas supply to the unit and instead of that an operation as MCCR, self mixing SCR or completely manually controlled rebreather. Additionally a trimix computer was developed and integrated.

Methods: The core element of the device is a double 4 position gas switch for the distribution of O₂ and diluent. In the first position the ECCR's main electronic is supplied with gas. In the case of a failure of the ECCR, the gas switch may be turned into one of the other 3 positions, which then shuts off the main gas supply of the ECCR. In two positions O₂ and diluent are fed into calibrated sonic orifices, allowing the use of the unit as self mixing SCR – remember, a safe operation of a SCR is possible even without pO₂ monitoring. In the 4th position neither the orifices nor the ECCR are supplied with gas. Two buttons allow manual injection of O₂ or diluent into the loop. For manual O₂ injection a small safety button has to be pressed first to release the gas injection button – an important feature to avoid mixing up the buttons. The orifices can be changed by the user. A trimix decompression computer was developed and integrated into the device. Its core element is an ATmega 644P low power microprocessor. The deco algorithm is based on the Buehlmann ZH16C dataset. Gradient factors and deep stops are supported. The unit is designed to be an upgrade for the Poseidon MK6 ECCR. A special microprocessor is implemented to serve as firewall to the MK6 internal digital bus. This allows reading out all data of the MK6.

Results: In total 5 prototypes were built up and attached to MK6 rebreathers. The correct implementation of the decompression algorithms were verified in MATLAB and in a laboratory set up that allows pressurization of the system up to 20 bar. First tests were successfully carried out. A digital port allows interfacing accessories like for example a head up display.

Discussion: The developed device adds redundancy to an ECCR rebreather. Together with the recreational MK6 rebreather, the additional redundancy allows now safe diving also beyond recreational limits. In the case of a failure of the ECCR, the recommended surfacing schedule suggests ascending to 20 m on SCR position 2, then SCR on SCR position one ascend to 6m, and then flush the loop manually with O₂ to finish decompression and return safely to the surface. Even without any pO₂ sensor readings a safe ascend on the loop by using the remaining gas of the supply tanks is possible. As an alternative one SCR position can be used as MCCR O₂ constant mass flow orifice.

ELECTRONICALLY CONTROLLED CLOSED CIRCUIT REBREATHING FOR RECREATIONAL PURPOSES

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Aim: R&D of a safe electronically controlled closed circuit rebreather for recreational purposes

Introduction: Rebreather diving systems offer an optimum of gas efficiency, warm and humid breathing gas and bubble free diving, which allows “silent” bubble free diving and better integration into nature. Drawback of most of the rebreathers today on the market is their complexity. Special training is required to handle a rebreather, which does not only include diving but also the correct preparation and checking of the unit prior to a dive. Thus especially closed circuit rebreathers were till now mainly addressing professional, technical and very advanced sports divers that have a large diving experience and an advanced technical understanding. Typical problems that arose in rebreather diving and which have led to accidents and fatalities in the past are malfunction of the pO₂ sensors and the sensor electronics (which leads quickly to a pO₂ in the loop outside life sustaining limits) and CO₂ buildup in the rebreather due to malfunction of the scrubber (incorrect filling, channeling). The Poseidon MK6 rebreather was especially developed for recreational purposes. Tasks that are difficult to handle are performed automatically and special safety features were included to assure maximum safety for the diver.

Methods: A network of 4 microcontrollers is the backbone of the MK6 rebreather. One microcontroller, housed at the top of the scrubber reads out the pO₂ sensors, the ambient pressure sensor and the tank pressure sensors and drives the solenoids. Instead of using 3 sensors like in other ECCR's on the market, only 2 are used together with an innovative sensor signal validation technique that was previously presented at EUBS 2007. Here the sensors are periodically checked and validated by flushing them with a gas with a known O₂ fraction. The battery pack includes another microcontroller, memory, where all dive data including tissue loadings are stored. A large display informs about all dive-relevant data. In the case of a failure, several alarming devices are included – a Head Up Display with a bright red LED and a vibrator motor mounted on the mouthpiece, a speaker and the so called buddy LED in the battery pack and alarming symbols on the main display. Preparing an ECCR for diving includes many pre dive checks. In order to avoid from the beginning user errors when performing the pre dive checks, all these checks are completely automatically performed. They include electrical checks of all electronic components, calibration of the sensors, physical tests of the solenoids and correct function of the mouthpiece, automatic overpressure check, etc. To avoid scrubber problems, pre packed scrubber cartridges are used in the MK6. In that way scrubber filling mistakes can be excluded and the preparation of the unit for diving takes less time. An innovative mouthpiece was developed, that can be easily switched from open circuit to closed circuit. Additionally it serves as auto diluent valve in the closed circuit position.

Results: A batch of approximately 200 units were built up and shipped. CE certification was passed. The unit is lightweight and small (8 kg) and allows diving times up to more than 3 hours (according to standard tests detailed in EN14143).

Conclusion: The MK6 is the first ECCR especially developed for the recreational market. It includes automatic pre dive checks and continuous system validation to assure maximum safety of the divers and to facilitate using the device. We envisage that these are the key features for a great success of the MK6 on the recreational market.

IDENTIFYING ELIGIBLE PATIENTS FOR INCLUSION IN CLINICAL TRIALS: THE HOLLT EXPERIENCE.

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Background

HOLLT (Hyperbaric Oxygen in Lower Limb Trauma) is an international multi-centre, randomised controlled trial which aims to assess the benefit of providing hyperbaric oxygen therapy during the acute stages of management of severe lower leg injuries as an adjunct to normal trauma care. It is being conducted by an international network of trauma centres with hyperbaric treatment facilities.

The HOLLT Study at The Alfred Hospital needs to identify potential subjects from the patients with severe, lower limb trauma, specifically severe open fractures of the tibia admitted through emergency. Accurate identification of these potential subjects at admission will enhance enrolment into the HOLLT Study as well as providing information necessary to report trial results and to assess the generalisability of findings.

Aims

To evaluate the recruitment of open tibial fractures to HOLLT at The Alfred Hospital.

To describe the profile of open tibial fractures not recruited to HOLLT at The Alfred Hospital.

To assess the usefulness of the methods used to identify patients at The Alfred Hospital and compare them with alternative strategies.

Methods

Emergency admissions to the Alfred Hospital between November 2006 and March 2009 were reviewed twice weekly in order to identify lower limb injuries. Patients with a lower limb injury were reviewed to ascertain the presence of an open tibial fracture and potential eligibility for the HOLLT Study. Referrals / notifications were also received from orthopaedic and trauma doctors. Ethical approval was obtained from The Alfred Human Research Ethics Committee.

Results

406 fractured tibias were identified in emergencies presenting to The Alfred Hospital. Of these 143 were open tibial fractures with 55 potentially meeting the criteria for inclusion into the HOLLT Study. From these 55 patients 14 were recruited into the HOLLT Study. Of the 41 patients ineligible for the HOLLT Study being too ill (32%) and referral to active hyperbaric oxygen treatment (20%) accounted for half, while difficulty obtaining consent (7%) or refusal to consent (10%) and failure to identify patients (15%) accounted for a third.

Over this period an average identification of 1.9 patients per month who were potentially eligible was made. This is consistent with the numbers identified in the literature.

Conclusion

The level of identification of open tibial fractures and open tibial fractures with severe soft tissue injury is consistent with other data regarding these injuries. While not ideal the screening systems in place provide an acceptable method of identifying patients with the screening log providing a means of documenting the process. The degree of success we have had with recruitment has been enabled by the presence of a dedicated research nurse. Providing simple inclusion criteria to orthopaedic, emergency and intensive care departments allows medical staff to quickly determine a patient's eligibility for the HOLLT Study.

HYPERBARIC OXYGEN THERAPY FOR THE TREATMENT OF INTERSTITIAL CYSTITIS - CASE REPORT

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INTRODUCTION: Interstitial cystitis is a collective term for a chronic heterogeneous syndrome, which goes along with suprapubic pain, nocturia, urinary urgency and frequency and as follows with restricted quality of life. Primarily women are affected. The etiology is mostly unknown, it's assumed to be a multifactorial complaint with a complex interaction among nervous, immune and endocrine systems. Among others hypoxic condition in the submucosal layer of the urinary bladder could play a decisive role in pathogenesis. Due to its biological mechanism hyperbaric oxygen therapy is one option in treating interstitial cystitis.

CASE: A 43-year-old woman was diagnosed with interstitial cystitis in March 2008. She worked as an anaesthesiologist and therefrom she was involved in air rescue service. She was especially suffering from suprapubic pain, urinary frequency up to 12 times during the day and nocturia up to 6 times. She was constricted at such a rate that she wasn't able to do her work. After resistance to many conventional treatments (behaviour training, anticholinergics, antidepressants) a first improvement was realized with chondroitinsulfat-instillation, but only in combination with an effective pain management with metamizol and buprenorphin. After having finished this therapy the patient's clinical state was worsening rapidly. The bladder capacity was declining below 100 ml, the urinary frequency, nocturia and pain were escalating.

RESULTS: As the chart demonstrates, the outcome after 30 sessions was pleasing. As a result of a significant reduction of the urological symptoms the quality of life was rising and pain medication could be reduced. Actually working was possible again. The therapy was furthermore well tolerated, there were no complaints due to hyperbaric oxygen exposition.

parameter	before therapy	after 30 sessions
bladder capacity (ml)	70-100	180-250
nocturia	4-6	2-3
urin. frequency	12-15	6-4
pain (VAS)	8	5

DISCUSSION: Even though there are not many studies about the efficacy of hyperbaric oxygen in treating interstitial cystitis this case demonstrates that it is a reasonable option for patients resistant to conventional treatments.

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RANDOMISED DOUBLE-BLIND CONTROLLED PHASE III TRIAL OF HYPERBARIC OXYGEN THERAPY IN PATIENTS SUFFERING LONG-TERM ADVERSE EFFECTS OF RADIOTHERAPY FOR PELVIC CANCER

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Aims

To test the clinical benefits of high pressure oxygen (HBO) therapy in restoring bowel function to patients suffering chronic radiation-induced gastrointestinal complications.

Introduction:

As a consequence of increased survival rates more individuals are living with the legacy of radiotherapy treatment side effects for pelvic malignancies. The HORTIS trial offers evidence that these symptoms can be modified by HBO, but independent confirmation of long-term benefit is needed before routine adoption of this modality is justified.

Methods:

75 patients with troublesome radiation-induced gastro-intestinal symptoms unresponsive to best standard care are randomised in a 2:1 ratio to HBO or sham treatment. Patients in the HBO group are compressed to 2.4 ATA and breathe 100% oxygen at pressure following Royal Navy Therapeutic Table 66 (RNTT 66). The total time at 2.4 ATA will be 90 minutes. Each participant receives a total of 40 pressure exposures (five days per week for eight weeks). The sham-treated group is compressed to 1.3 ATA in a hyperbaric chamber and breathes 21% oxygen (air) while at pressure following the same exposure schedule.

All eligible patients (whose symptoms persist despite 3 months optimal gastroenterological intervention) attend the Gastrointestinal Late Toxicity Clinic at the Royal Marsden Hospital (London) before treatment, within 2 weeks of completion of hyperbaric therapy and 12 months later. On each occasion, patients complete a LENT SOMA assessment of radiation injury (small intestine / colon and rectal) with a nurse practitioner. They are also asked for consent to undergo flexible sigmoidoscopy, rectal photography and biopsy. The Modified Inflammatory Bowel Disease (IBDQ), EORTC Quality of Life QLQ-C30 and Defaecation Problem Subscale of QLQ-CR38 are completed at baseline and at 3, 6, 9, and 12 months after the start of HBO therapy. Data are also collected on health economic consequences.

End Points

The primary endpoint is change in modified IBDQ score. Secondary clinical endpoints include assessment of LENT SOMA scores, patient self-assessments, and photographic scores of rectal mucosa. Translational endpoints include the stimulation of neoangiogenesis and resolution of fibrosis in rectal mucosa.

Discussion:

Positive robust data supporting the use of HBO are critical to enable a large patient population living with radiation injury to gain access to effective care. The trial opened to recruitment in March 2009 and to reach our recruitment target, we need to engage gastroenterologists and oncologists as well as the hyperbaric medicine community across the UK.

HYPERBARIC OXYGEN IN PEDIATRIC ONCOLOGY

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Introduction:

Use of Hyperbaric Oxygen Treatment (HBO) for children requires special knowledges for indication and treatment. Nursing of critically ill patients demands specific equipment inside of hyperbaric chamber as well as close cooperation between pediatric and hyperbaric specialists for ensurance of adequate care of sick pediatric patients.

Methods and Results:

This is retrospective observational study. From 1994-2007 total of 33 children (19 of male sex, 14 of female sex) have been treated for complications of oncological treatment in our HBO treatment centre. Mean age of children were 8 years (1-17 years). Among 33 patients there were 22 patients with blood malignancy and 11 patients with solid tumour. Main reason for HBO treatment were: chemotherapy - induced hepatotoxicity in 17 patients (52%), extensive intracranial surgery for malignant diseases in 9 patients(27%), severe posthypoxic encephalopathy in 3 patients (9%), severe polyneuromyopathy after immunity suppressive drugs in 3 patients(9%). One child (3%) has been treated for radiation injury of urine bladder. Mean number of HBO sessions were 7,4 (1-20), treatment pressure of 1.9 to 2.5 ATA. Treatment effects have been evaluated by decrease of serum levels of alanine aminotransferase (ALT) in patiens with chemotherapy - induced hepatotoxicity. For brain tumour surgery, posthypoxic encephalopathy and critical illness polyneuromyopathy change of pre/post HBO Modified Rankin Scale and pre/post HBO electromyography were the main treatment effectivity measures. Positive effects of HBO treatment have been achieved in 85% of all patients. 1 patient died 2 months after HBO (bone marrow failure and pneumonia).

Discussion:

This is a pilot study for creating of basal imagine of HBO effects in those clinical situations. Most of patients can be evaluated as critically ill patients and the complex treatment has been supplied. HBO treatment for those patients is high risk due to infectious diseases, immune suppression therapy, need for isolation of patients inside of hyperbaric facility.

Conclusion:

According to our results HBO therapy can be very effective for pediatric patients with severe complications of oncology treatment. This is emphasized especially when biological nature of tumour requires early initiation or continuation of complex oncology treatment (chemotherapy or radiotherapy). In addition, HBO is a safe treatment method with total rate of complications of 3%.

MARTORELL'S HYPERTENSIVE ULCER: EVALUATION WITH TRANSCUTANEOUS OXYGEN PERFUSION MEASUREMENTS OF TREATMENT WITH HYPERBARIC OXYGEN AND BLOOD PRESSURE CONTROL.

Lafère P¹, Vanhoutte D¹, Caers D¹, Germonpré P¹.

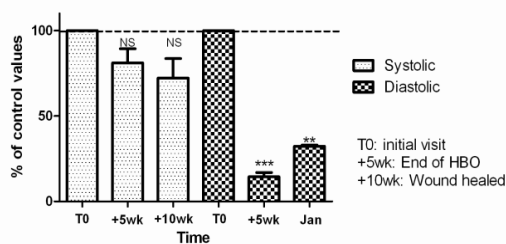
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Lower limb chronic ulcers may have various causes but are commonly due to arterial insufficiency and/or venous stasis. Sometimes, other differential diagnoses such as Martorell's Syndrome must be evoked. The authors describe a case where transcutaneous oxygen perfusion measurements (PtcO₂) have been performed, confirming the perfusion pathology in this patient and the effect of appropriate treatment.

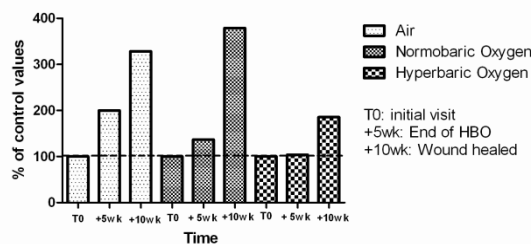
Case report

A 71-year old Caucasian woman with a ten year history of type II diabetes and hypertension was referred to our centre for treatment (HBO) of a non-healing leg ulcer. Diabetes mellitus was aggressively addressed with insulin and good control was obtained within a month (fasting blood sugar of 103 mg/dl, glycosylated hemoglobin of 5.9mg/dl). HBO resulted in a slight reduction in ulcer size from 1.7 x 1.1 cm to 1.3 x 0.7 cm; however, the ulcer remained very painful. It was decided to stop the HBO treatments and to focus on controlling the hypertension. After 5 weeks of treatment, a 24 hours blood pressure recording was performed. This showed a substantial improvement in blood pressure control. Taking the initial value as 100%, procentual variations were calculated for systolic and diastolic blood pressure, allowing an appreciation of the magnitude of change rather the absolute values (Figure 1). By 10 weeks after the initial visit, the ulcer had 100% epithelialised and the patient was pain free. Transcutaneous oxygen pressure measurements (PtcO₂) were performed (Radiometer TCM3, Copenhagen) before and at the end of the HBO treatment period, and after full healing of the wound. With the improvement of blood pressure control, especially the diastolic pressure, we observed a trend toward improvement of the PtcO₂ (Figure 2).

PROCENTUAL VARIATION OF ARTERIAL PRESSURE



PROCENTUAL VARIATION OF PtcO₂



After a full course of HBO treatment, baseline PtcO₂ was only minimally increased in normobaric air (200%, absolute value 28 mmHg) or normobaric oxygen (130%, absolute value 52 mmHg). However, after proper blood pressure control, these values are almost normal. Even the PtcO₂ measurements in hyperbaric oxygen, which were already quite acceptable at baseline (absolute values 1740 mmHg), showed a marked improvement after blood pressure control had been achieved.

Discussion

Aggressive therapy toward blood pressure control seems to be correlated with a trend to the improvement of the PtcO₂ and complete healing of the ulceration. This concurs with the idea of Nikolova which suggests that an additional criterion for diagnosis of hypertensive ulcers is "prompt healing with aggressive blood pressure therapy".

The measurement of PtcO₂ and the use of a combination of calcium channel blockers and inhibitors of angiotensin converting enzyme is compatible to the physiopathological hypothesis of arteriosclerosis associated to vasoconstriction. It also confirms that the main goal of treatment should be optimal control of the blood pressure.

COMPARISONS BETWEEN UNITED KINGDOM AND EUROPEAN PROTOCOLS USED IN THE TREATMENT OF OSTEORADIONECROSIS WITH HYPERBARIC OXYGEN THERAPY

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Aims

Previous data has shown little standardisation in the protocols used by hyperbaric chambers in the United Kingdom for the treatment of patients with osteoradionecrosis (ORN)¹. The aim of this study was to establish the number of patients with ORN being treated by United Kingdom and European hyperbaric chambers and for comparisons to be made between the protocols used by the two groups.

Method

25 United Kingdom and 69 European hyperbaric chambers were surveyed. The number of patients treated during one year and the protocols used were determined and compared.

Results

25 United Kingdom hyperbaric chambers treated 273 patients with ORN. 9 were British Hyperbaric Association (BHA) registered and 16 were Multiple Sclerosis (MS) centres. 69 European chambers treated 1304 patients with ORN. Most European chambers treated patients at higher pressures (2.5 ATA) and for longer sessions (>90mins) when compared to United Kingdom chambers that were BHA listed only (90 mins at 2.2ATA) and United Kingdom chambers including BHA listed and MS centres (<90mins at <2ATA) and the Marx protocol (90 mins at 2.4ATA). There were similarities with regards to the number of sessions. Both groups predominantly used 30 preoperative and 10 postoperative sessions.

Discussion

The optimal therapeutic use of hyperbaric oxygen therapy remains poorly understood. European chambers use higher pressures and longer sessions of hyperbaric oxygen therapy compared to both United Kingdom chambers and the Marx Protocol. UK chambers tend to use lower pressures and shorter sessions compared to the Marx Protocol. Both groups predominantly used comparable numbers of sessions to the Marx Protocol.

CHANGES IN MICROBIAL COMMUNITY ASSOCIATED WITH CHRONIC DIABETIC FOOT ULCERS OVER THE COURSE OF HYPERBARIC OXYGEN TREATMENT

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Aims: A detailed molecular analysis of microbiological communities in chronic diabetic foot ulcers over the course of hyperbaric oxygen (HBO) therapies is being performed. Correlations between the dynamics of microbiological communities during HBO therapy and subsequent wound healing will be investigated.

Introduction: In the South West of England diabetic patients with non-healing foot ulcers may be referred for HBO treatment at the Hyperbaric Medical Centre (DDRC) in Plymouth. Some patients' ulcers heal well during treatment but some do not and it is not fully understood why this is so. Analysis of the potential contributors to non-healing and evidence for direct correlations are problematic due to the numerous variables involved, e.g. smoking, underlying medical pathology, non-compliance and obesity. Evidence suggests that infection has an important role to play in the non-healing pathology and part of this could be due to synergistic or antagonistic relationships between species of bacteria and fungi within a chronic wound community. To our knowledge changes in wound microbial communities have never been reported during a course of HBO treatment.

Methods: The study has received approval from the NHS Cornwall & Plymouth Research Ethics Committee (REC Ref: 08/H0203/93). Ten diabetic patients with a non-healing foot ulcer are being recruited from the Joint Foot Clinic at Derriford Hospital in Plymouth; initial wound swabs are taken after consent to join the study has been obtained. Wounds that fail to heal with routine foot care and antibiotic treatment are referred to DDRC for HBO. Wound swabs and wound tissue samples are taken at regular intervals throughout the course of treatment. Samples are also taken from a small group of patients who do not undergo HBO treatment. The provision of appropriate randomised controls is ethically questionable, and therefore wounds not receiving HBO are generally self selected and not directly comparable to wounds undergoing HBO therapy. Three swabs taken during HBO are sent to the microbiology department at Derriford hospital for routine culture and identification. Eleven swabs are used for molecular analysis consisting of PCR amplification followed by denaturing gradient gel electrophoresis (DGGE). PCR products from routinely cultured organisms will be used to produce a control ladder for the DGGE procedure. Bands of interest will be sequenced and identified.

Results: The primary end point will be gross changes in microbial communities over the course of hyperbaric oxygen therapy.

Discussion: This wound study forms part of a PhD research programme. Results will be interpreted in light of other aspects of the research programme encompassing the effects of HBO therapy on antibiotic resistance in chronic wound bacteria. The potential role of biofilms and quorum sensing is being explored.

HYPERBARIC OXYGEN THERAPY USED TO TREAT LATE SOFT TISSUE RADIATION DAMAGE: A CASE REPORT

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Aim: The aim of this presentation is to report the positive effect of hyperbaric oxygen therapy (HBOT) in a case of late soft tissue radionecrosis (LSTR) which was resistant to conventional therapies.

Introduction: Soft tissue radionecrosis may occur as a late complication of radiation in cancer patients who undergo radiotherapy. Severe forms of LSTR such as infected non-healing ulcerations may be seen. Hypovascularity, tissue hypoxia, and fibrosis are the main causes of non-healing wounds. With its proven angiogenetic effect HBOT has been used for the treatment of late complications of radiotherapy. It has also been expected to be effective in LSTR cases which are not responding to conventional therapies (1-3).

Case: The patient was a 47 year old, Type 2 diabetic female, primarily diagnosed as invasive ductal breast cancer of the right breast. She had breast conserving surgery followed by chemotherapy and radiotherapy. An ulceration occurred in intertriginous area after one year of radiation completion. Conventional treatments and wound care were failed and she was referred to our chronic wound care clinic. The 2x2x4 cm cavitory wound was infected and surrounded by discolored and desquamated skin. Medical treatment as needed and proper daily wound care were combined with HBOT. The patient received 40 sessions of HBOT in a monoplace chamber. The treatment protocol was 100% O₂ for 90 minutes at 2.1 ATA, one session a day, 5 days a week.

Results: Wound bed distinctively improved by the 10th session of HBOT. Infection was eradicated by the 20th session of HBOT and epithelization complete in the previously desquamated peri-wound area. Following the 40th session, the wound dimensions were diminished to 2x2x1 cm, wound was clean with good granulation tissue and ready for closure. The patient was discharged for surgical suturation. No complications developed due to HBOT. She was symptom-free on two years control.

Discussion: In a radionecrotic wound the oxygen gradient between the center and the surrounding tissues of the wound is too low to start the chemotactic process for wound healing (1). Hyperbaric oxygen promotes wound healing by increasing tissue oxygen, stimulating angiogenesis, fibroblast proliferation, collagen synthesis, and epithelization (4). This case report presents the positive effect of HBOT on wound healing in a case of soft tissue radionecrosis resistant to conventional therapies.

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CRITICAL INCIDENTS IN A LEVEL 1 HYPERBARIC UNIT:

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Aims:

Our intention is to list a number of incidents that occurred in our unit, fortunately none of which led to serious problems. We aim to stimulate colleagues to do the same and start a National or International critical incident reporting database.

Introduction:

Critical incidents in the hyperbaric unit need to be monitored and lessons learned from them. Ideally the message should be passed on to other units and may help to prevent similar incidents. Anaesthetists have done that with great benefit elsewhere. No formal Critical Incident monitoring takes place in the UK (yet).

Method:

Cases were taken from the hospital database and from long serving staff's memories. I realize some cases will be missed. I will attempt to list them and discuss actions taken to prevent recurrence.

Results and Discussion:

Ventilator related issues were the most common:

1. Hypoventilation due to increased gas density and use of an inadequate ventilator (Manley).
Action: Using a volume meter.
2. Disconnection (Manley).
Action: Using a volume meter/ Disconnection alarm.
3. Ventilator (Malfunction of Siaretron).
Action: Changing ventilator operation
Shorter tubing
4. Disconnection of drains and infusion devices.
Action: Old diving chamber replaced with modern unit and good access
Neurotic checking of all attachments and lines.
5. 'The Bends' in Medical Attendant
6. Insufficient air to pressurise the chamber at the desired rate leading to ventilator failure.
Action: Changed therapy table due to automisation leading to middle ear barotrauma.
7. Middle ear barotrauma
Action: Changed therapy table
8. Tracheal tube cuff inflation errors.
9. Haemorrhagic shock post debridement of Necrotizing fasciitis
Action: Scheduling hyperbaric therapy before surgical debridement.

TIME COURSE OF AUTONOMIC NERVOUS CONTROL ALTERATION INDUCED BY 100% OXYGEN BREATHING IN HEALTHY MEN

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Abstract

Aims

For how long do hyperoxia effects persist after gas exposure?

Introduction:

High partial pressure of oxygen induces significant hemodynamic effects that may be related to autonomic nervous system activity changes.

Methods:

Ten healthy male volunteers were randomized in a double-blind fashion to breathe either medical air or 100% oxygen by face mask for 45 minutes. The role of the autonomic nervous system activity during and after gas exposure was evaluated using power spectral density of heart rate variability (HRV, cardiac control) and blood pressure variability (BPV, vasomotor control). The low frequencies (LF, 0.04-0.15 Hz) and the high frequencies (HF, 0.15-0.4 Hz) normalized (divided by total spectral density) were analyzed before (baseline), during gas exposure, and after gas exposure at 10, 30, 60 and 90 minutes. During measurements, volunteers breathed at 15 cycles.minute⁻¹. After each ECG and blood pressure measurement, ultrasonographic measurements were performed. Stroke volume (SV), cardiac output (CO) and systemic vascular resistances (SVR) were calculated.

Results:

No significant changes were observed during medical air exposure. Systolic and diastolic BP were not significantly changed both during and after oxygen exposure. During oxygen exposure, hemodynamic changes included a decrease in CO (-19.1 %) secondary to a lowering in both HR (-6.4 %) and SV (-12.2 %) and an increase in SVR (24.7 %). CO, VES and SVR changes persisted 30 minutes after the end of oxygen exposure. Power spectral density of HRV and BPV was show in the table.

	Baseline	Oxygen	R10	R30	R60	R90
LF _{HRV}	69 ± 17	64 ± 23 *	82 ± 11 ^μ	82 ± 13 ^μ	79 ± 16	77 ± 12
HF _{HRV}	31 ± 17	36 ± 23 *	18 ± 11 ^μ	18 ± 13 ^μ	21 ± 16	23 ± 12
LF _{HRV} /HF _{HRV}	2.3 ± 1.0	1.3 ± 0.8 *	4.2 ± 3.4 ^μ	4.1 ± 1.7 ^μ	3.7 ± 1.3	3.3 ± 1.4
LF _{BPV}	76 ± 13	51 ± 25 * \$	66 ± 13 £	77 ± 12	74 ± 13	73 ± 12
HF _{BPV}	24 ± 13	49 ± 25 * \$	34 ± 13 £	23 ± 12	26 ± 13	27 ± 12
LF _{BPV} /HF _{BPV}	4.9 ± 2.8	2.3 ± 1.0 £	2.4 ± 0.6 * \$	5.2 ± 1.3	5.5 ± 1.6	3.6 ± 1.1

* p < 0.05 Oxygen vs. All ; £ p < 0.05 R10 vs. All ; \$ p < 0.05 Oxygen vs. R10 ; ^μ p < 0.05 Baseline vs. R10 and R30.

Conclusion:

In healthy young adults, normobaric hyperoxia didn't affect blood pressure but induced-systemic vascular resistances increase. Sympathetic nervous activity to vasomotor control was decreased during hyperoxia and these changes persisted during the 10 first minutes of return to ambient air. Heart rate returned to baseline (inferior to 10 minutes) before stroke volume and cardiac output (30 minutes). The decrease in heart rate was attributed to an increase in parasympathetic nervous activity. Power spectral density of HRV was modified until 30 minutes after the end of hyperoxia. Ours results were in accordance with two distinct phenomena of hyperoxia, one peripheral and other one central.

Key words: Cardiovascular. Hyperoxia. Power Spectral Density Analysis.

PRESSURE REGULATOR FOR IN-CHAMBER BLOOD PRESSURE MEASUREMENTS.

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Aims: During intensive care in a hyperbaric chamber continuous measurement of the blood pressures is of great importance. At any time the back pressure for the pressure has to be larger than the highest blood pressure in order to prevent clogging of the cannula. During the hyperbaric setting the pressure in the back pressure has to adapt to the alterations in the environmental pressure.

Methods. A reduction valve is placed inside the hyperbaric chamber. The inlet pressure is 10 bar absolute pressure. This pressure is reduced to 1.2 bar absolute pressure connected to an infusion pressure bag. On the low pressure side is mounted a vent permitting some air to escape at any time. A pressure gauge is part of the infusion pressure bag and offering a possibility to assure, that the back pressure is correct. The reduction valve has a similar function as second stage in scuba gas supply.

Results. The system has worked well though app. 10 years. The system is maintaining the intended pressure during constant pressure as well as during increasing and decreasing pressure rates of 0.5 bar per minute.

Discussion. During intensive care it is of great importance to have a safe blood pressure reading at any time. In our clinic we have about 60 patients or 200 sessions annually where intensive care is part of the treatment. About 60 per cent of the cases are in a treated circulatory shock. If the back pressure has to be adjusted manually, the focus is shortly taken away from the patient. In situations where the patient is without staff inside the chamber it is of course necessary to have an automatic regulation

We conclude that an automatic pressure regulator for in-chamber blood pressure measurements is an important part of the equipment for intensive care under hyperbaric conditions.



A DEVICE FOR HYPERBARIC VENTILATION OF THE TRACHEOSTOMA PATIENT.

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Aims: Some patients with tracheostomy may need hyperbaric oxygen treatment. In some cases the use of a tracheostomy tube may be contraindicated, and the tracheostoma cannot be covered by a mask or oxygen hood.

Methods. By means of a colostomy bag and ventilation hose, we obtained a tracheostomy cover (fig 1). The hole in the colostomy bag was cut into a size equivalent to the size of the tracheostoma (fig 2). The bag and hose system was connected to a ventilator double hose and a ventilation bag on the inlet and outlet side of the system. The system was connected to an Amron gas panel. The Amron panel is of the kind used for oxygen hoods (fig3).



Figure 1



Figure 2



Figure 3

Results. The system has worked well in at least 3 cases or 90 sessions. As the skin typically is very thin, we have chosen to heat the colostomy bag for 5 seconds in a microwave oven, thereby the material gets soft and provides a better adhesion to the skin. Leakage was found to be rare as measured by means of a Haux Oxysearch \square meter during HBO sessions. The system is tight and the patients feel no breathing resistance.

Discussion. Patients with a tracheostoma without a cannula or tubing may pose a problem during HBO. Most often a mask or oxygen hood cannot cover the tracheostoma. The easiest solution is to insert a cuffed tracheostoma tube. However, a tracheostomy tube may even be contraindicated. Insertion of a tube may provide irritation or inflammation of the tracheostoma. Typically the patient has been exposed to previous therapeutic radiation. The trachea and the skin may therefore be vulnerable to adhesive tape and manipulation. The use of a double balloon system provides breathing with almost no ventilation resistance. We conclude that a modified colostomy bag system provides a useful alternative to tracheal intubation during HBO.

