

# European Journal of Underwater and Hyperbaric Medicine



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**DISCLAIMER:** All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of the EUBS.

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**ECHM**                      **European Committee for Hyperbaric Medicine**  
**SPUMS**                      **South Pacific Underwater Medicine Society**  
**UHMS**                      **Undersea and Hyperbaric Medical Society**

**MANUSCRIPTS:** see INSTRUCTIONS TO AUTHORS on inside of back cover.

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## EDITOR'S COLUMN

### Dear Readers!

The last issue of the Journal has been full with information about meetings and courses in Diving and Hyperbaric Medicine. This clearly demonstrates the importance of such a journal as the *Europ J Underw & Hyperb Med* for the continuing education of physicians and research scientists. It also serves as a forum for information on the diverse activities of our members in this relation.

As a reader of this journal you probably don't need to attend an Introductory Course in Diving Medicine. But if you know a colleague who wants to, then tell them that a course which complies with the training objectives of the European Diving Technology Committee is being planned for Thursday 7th to Sunday 10th March 2002 by David Elliott and Nick McIver in conjunction with Gerard Laden of the North East Hyperbaric Medicine unit in Hull. Easy to get to (nearest airport Humberside and plenty of ferries across the North Sea). Details from [Karen@Biomedseminars.demon.co.uk](mailto:Karen@Biomedseminars.demon.co.uk) or fax (+44) 208 786 7036.

This issue is dedicated to the Annual Scientific Meeting of the EUBS with the whole edition containing the Programme and Abstracts of the meeting in Hamburg. This way the pre-requisite to have the meetings abstracts available to all members of the Society in the EUBS Newsletter is achieved in a timely fashion.

The meeting itself and all the social events have been organised by Dr. Uli van Laak with all the verve and effectiveness a Senior Navy Officer can devote to the position of Secretary General of such a meeting. It has been a compliment for me to be asked for support and a great pleasure to assist him in this difficult task on the organising committee.

All the best for a successful conference and an enjoyable stay in Hamburg to all of you,

**Peter**

## ERRATUM

As the Editor of the *European Journal of Underwater and Hyperbaric Medicine* I regret that an error passed unnoticed in the article "Tissue Oxygen Levels During Hyperbaric Oxygen Breathing" by Dr. Valerie Flook which was published in the **June 2001** issue. Figures 4 and 5 were exchanged mistakenly while the legends of the figures are displayed accordingly to the text.

**Peter**

## WELCOME ADDRESS

**Dear Members of the EUBS!**

**Dear Participants of the EUBS 2001 Scientific Meeting!**

**Dear Friends!**

Fortunate to participate in a good number of outstanding EUBS Annual Scientific Meetings of the last two decades I have agreed unhesitatingly on taking over the responsibilities of the Secretary General of the EUBS year 2001 event in Hamburg.

The 27<sup>th</sup> Annual Scientific Meeting of EUBS is our society's 30<sup>th</sup> anniversary as well. We have dedicated the Hamburg meeting to the founders and scientists who used to be engaged in the different committees of EUBS over the three decades. At this time I would like to particular welcome those attending who belong to this founder and promoting group. Dr. Robert C. Bornmann, founding member of 1971, has come over from the States in order to serve as the Honorary Chairman of the meeting.

There is no doubt that the scientific program accompanied by the satellite workshops provides us all with an outstanding forum for discussion of new advances and concepts. However, sharing of information about positive developments and success means only one side of the medal. Unpleasant trends must be especially focussed and these lessons learnt have to be exchanged as well.

The EUBS 2001 meeting is now coincident in my time as the current President of the German Society for Diving and Hyperbaric Medicine and a severe crisis of HBOT acceptance here in Germany. The discussion about the origin of the crisis reflect some differences of opinion, however, one point is quite clear. There is a risk for other European countries that economy measures may affect them according to the same pattern.

Writing this 4 weeks before the meeting will start I can only summarise – much to my regret – that the number of registered participants is currently disappointing. This will likely turn into better due to the late booking participants but has already heavily affected our initial design. Appropriate measures had to be taken in order to prevent penalties for breach of

contracts. Hard enough for the organising people another aspect may be more worrying. It is obvious that the more HBOT orientated EUBS members have reacted with restraint for what reason ever. I am not quite sure, however, if there was a certain tendency for separate ways, meetings included, this would mean no good at all. Let's hope the best.

Beside the typical frown of a concerned Secretary General: I am delighted to welcome you all who found the way to Hamburg, a beautiful, historical and attractive city, for participation in our 30 years of EUBS anniversary Annual Scientific Meeting. We thank the different speakers and presenters for providing their time and energy and sharing their expertise with us. Special thanks is due the invited lecturers who have agreed unanimously to take over on short notice. Last but not least Congress Partner, namely Matthias Spacke, did a great job in supporting the Secretary General with his expertise and experience.

**Enjoy your EUBS 2001 in Hamburg!**

**Yours Ulrich van Laak  
Secretary General EUBS Meeting 2001**

**Dear friends!**

We are in the middle of summer and most of us are either enjoying a well earned vacation, just come back from one or on the way to one.

This is also conference time and many of the conferences we need or want to attend are held in summer. Our 2001 EUBS conference is also a summer event and I hope many of you have marked it down as one of the conferences you want AND need to attend.

This conference is, for many of us, the only opportunity we have to discuss hyperbaric and diving medicine with a good number of practitioners and experts in the field. Many of the papers presented will contain innovative ideas and although the papers themselves will be available as proceedings, the discussions and comments arising from each paper are not. Not being there means losing the possibility of hearing and even participating in the discussions that follow each presentation.

Our society has been in existence for many years, its main activity, sometimes only activity has been the annual conference, and now our journal, which must change from being only a news sheet announcing conferences, meetings and courses to a forum of discussion on the subject of Baromedicine.

The medical climate in Europe is changing, Medicine is becoming more evidence based, demanding that there is hard evidence to justify each therapeutic and preventive action. Also Governments are no longer willing to cover the costs of all forms of treatment required by their citizens, this is forced on them by the demographic time bomb with an increasingly ageing population, continually rising costs of quality medical care and the raised expectations of the citizen who demands individualized and state of the art medical care from the country's health system.

This means that treatment modalities such as HBO which, unfortunately, is being considered by many uninformed but influential persons as a non-essential mode of treatment, are finding difficulties obtaining the funding and the support necessary to survive. This year's conference host country, is a case in point with the now well known restrictions and limitations placed on state and insurance payment cover for HBO even in cases of Decompression Illness.

Such restrictions have made many of our German colleagues unable to attend our conference through lack of funds even though it is being held in their own country.

There is the need for the baromedical community to become much more proactive and aggressive in the drive to establish HBO as a universally accepted treatment modality for specific indications, we can no longer remain passive to what is going on around us. A good start would be to participate more in the activities of this Society. This year there was only one nomination for Member at Large. Surely in a Society with 400+ members there are individuals

who want to contribute actively to the Society. I hope that during this conference a number of you will approach me or other members of the Executive Committee and offer more than your moral support.

Some of you may have received through the internet information on a number of new international hyperbaric societies. I cannot understand the need to keep re-inventing the wheel. What the hyperbaric community needs is a few strong organizations not a number of weak ones dispersing the strength and efforts of the few existing active individuals.

In Europe, the EUBS should be the scientific society and association and should work in collaboration with the various national societies to form a united front. Strength comes from unity and not individualism. Our society should work closely with two other bodies that do not compete but are complementary to it. The ECHM (European Committee on Hyperbaric Medicine) carries out an important function in creating internationally acceptable standards on all the relevant sections of Baromedicine, such as indications, treatment modalities, qualifications and training. The newly formed ECB (European College of Baromedicine) aims to provide and promote training and education opportunities in Baromedicine as well as act as a central registration and certification body for qualified persons working in the field.

These three organizations are not in competition with each other, in fact they carry completely different functions and together fill all the "gaps" in Baromedicine. I promise that I and my committee will make strong efforts in the coming year to work closely with our sister organizations to prepare ourselves for the changing European medical climate and ensure the survival of Baromedicine as an approved and accepted speciality in medicine.

**Iro Cali-Corleo**  
**President EUBS**

## REMINDER

All members who have not yet paid their membership dues for this year are reminded to do so instantly. The Society needs the funds to finance her responsibilities!

You are also reminded to report any changes in your address to the Membership Secretary and to the Newsletter Editor. Otherwise we can not guarantee that the Journal and the correspondence from the Society's secretariat do reach you in time.

**Thank you very much for your co-operation.**



**EUBS Annual Scientific Meeting Hamburg 2001**  
**Pre-Congress Satellite Symposium**

**3<sup>rd</sup> HAMBURG SYMPOSIUM ON OCCUPATIONAL RISKS  
IN HYPERBARIC TUNNELING AND COMMERCIAL DIVING**

**Wednesday, September 12, 2001, 8.00 a.m. – 5.30 p.m.,  
Bernhard-Nocht-Institut fuer Tropenmedizin, Hamburg**

- 08.00 – 09.00     **Registration**
- 09.00 – 09.20     **Opening ceremony**  
*Head of the Occupational Health and Safety Authority, Hamburg*  
*Prof. Dr. Scholbeck*, Leiter Prävention, Tiefbau-Berufsgenossenschaft  
**Chairpersons:** *D. Lamont*, Health and Safety Executive;  
*Dr. B. Neubauer*, Staatlicher Gewerbearzt
- 09.25 – 09.55     **Tunnel boring in kerosene contaminated soil**  
*Jean Claude Le Péchon*; JCLP Hyperbarie, Paris
- 09.55 – 10.25     **Breathing gas contamination by volatile hydrocarbons**  
*Dr. Valerie Flook*; University Aberdeen; Unimed Scientific Ltd
- 10.25 – 10.40     **Discussion of both presentations concerning hydrocarbons**
- 10.40 – 10.55     **Coffee break**
- 10.55 – 11.25     **Welding fumes in hyperbaric environments**  
*Dr. Szelagowski*, GKSS Forschungszentrum, Geesthacht
- 11.25 – 11.45     **Discussion of problems related to welding in hyperbaric environments**
- 11.50 – 12.05     **Mercury intoxication, Vienna subway construction**  
*Prof. Dr. Zeitlhofer*, Universitätsklinik für Neurologie, Wien
- 12.05 – 12.20     **Mercury quantification in hyperbaric tunneling**  
*Dipl. Ing. D. Otti*; Österreichische Staubbekämpfungsstelle, Leoben
- 12.20 – 12.35     **Discussion of both presentations concerning mercury intoxication**
- 12.35 – 13.35     **Lunch break**  
**Chairpersons:** *Dr. W. Förster*, Tiefbau-Berufsgenossenschaft;  
N.N.
- 13.35 – 14.15     **Aspects of fire in compressed air tunnels**  
*Donald R. Lamont*; Health and Safety Executive, Bootle / Great Britain
- 14.20 – 15.00     **Mineral dust in hyperbaric tunnelling**  
*Prof. Dr. Dr. Kessel*; Institut f. Arbeitsmedizin, Med. Universität zu Lübeck
- 15.00 – 15.15     **Coffee break**
- 15.15 – 15.55     **Underwater acoustics**  
*Marie E. Knafelc*; M.D., Ph.D., Navy Experimental Diving Unit, Panama City, USA
- 16.00 – 16.40     **Commercial diving in chemical / biological contaminated waters**  
*W. Förster*; Tiefbau-Berufsgenossenschaft, Munich
- 16.45 – 17.25     **Remote control underwater vehicles**  
*W. Hornfeld*; STN ATLAS Elektronik GmbH, Bremen;  
*Prof. Dr. J. Wernstedt*; Technische Universität Ilmenau
- 17.30             **Closing ceremony**



## **EUBS Annual Scientific Meeting Hamburg 2001 Pre-Congress Satellite Symposium**

### **SELECTED TRAVEL MEDICINE PROBLEMS IN RECREATIONAL DIVING**

**Wednesday, September 12, 2001, 8.30 a.m. – 5 p.m.,  
Hotel Inter Continental Hamburg**

(Open for all Registered Participants of the EUBS 2001 Annual Scientific Meeting)

Full Day Scientific Programme:

08:30            Introductory Remarks, Risks of Diving Abroad

#### **Malaria**

08:45 – 09:30    Epidemiology – Clinical Presentation – Treatment Regimes  
09:30 – 10:00    Diagnosis – Rapid Tests  
09:45 – 10:15    Prophylaxis Update: Exposition, Chemo  
10:15 – 10:30    Lariam® Prophylaxis in Recreational Diving - Recommendations GTÜM  
  
10:30 – 14:45    Discussion  
  
10:45 – 11:00    Coffee Break

#### **Traveller's Diarrhea**

11:00 – 11:30    Clinical Presentation – Differential Diagnosis  
11:30 – 12:00    Risk Factors and Prevention  
  
12:00 – 12:15    Discussion  
  
12:15 – 13:30    Lunch

#### **Outbreak Investigation**

13:30 – 14:15    Outbreaks – Introduction of Investigation Principles  
  
14:15 – 14:30    Discussion

#### **Shistosomiasis**

14:30 – 15:00    Clinical Presentation – Treatment  
15:00 – 15:30    Biology, Epidemiology, Prevention?  
  
15:30 – 15:45    Discussion

#### **Vaccination Requirements**

15:45 – 16:15    Update of Vaccination – Mandatory Prophylaxis or Nice to Have?  
  
16:15 – 16:30    Discussion  
  
16:30            Closing Remarks

# **EUBS ANNUAL SCIENTIFIC MEETING HAMBURG 2001**

**Hotel Inter Continental Hamburg, Germany**

## **Scientific Programme THURSDAY, SEPTEMBER 13, 2001**

### **09:00 WELCOME ADDRESSES**

**Ramiro Cali-Corleo** President EUBS

**Ulrich van Laak** President German Society for Diving & Hyperbaric Medicine

### **09:15 WELCOME LECTURE EUBS 30<sup>th</sup> ANNIVERSARY**

#### **EUBS: 30 YEARS OF PROGRESS AND GROWTH**

**Robert C. Bornmann** Founding Member of EUBS 1971 and  
Honorary Chairman EUBS 2001

**Leon J. Greenbaum, Jr.** UHMS Executive Director 1986 - 2000

**(10:00 – 18:00 2<sup>nd</sup> International “Arthur-Bornstein-Workshop” on Compressed Air Work  
and Deep Diving in Tunnel Construction (for invited Workshop Participants))**

### **10:00 INVITED LECTURE**

#### **ILLUSTRATED HISTORY OF HYPERBARIC CHAMBERS**

**Gerhard Haux**

**HAUX Life Support, Karlsbad-Ittersbach, Germany**

### **10:30 – 11.00 COFFEE BREAK**

### **11:00 – 13.00 SESSION A**

#### **DIVING PHYSIOLOGY I**

Chairpersons: **Greta Bolstad, Norway**  
**Jürgen Wenzel, Germany**

11:00 A01 METHOD FOR QUANTIFYING MECHANICAL ENDOTHELIAL DAMAGE  
**Vibeke Nossun, A. O. Brubakk (Norway)**

11:15 A02 EXTRACELLULAR DEHYDRATION INDUCES FUNCTIONAL MOTOR SPINAL DEFICIT AFTER  
SEVERE DECOMPRESSION IN THE RABBIT  
**J.-L. Méliet, Philippe Léni †, J.-P. Menu, F. Laforest, B. Zouani (France)**

11:30 A03 EFFECTS OF SELECTIVE GABA RECEPTOR LIGANDS ON NARCOSIS PRODUCED BY  
NITROGEN, ARGON AND NITROUS OXIDE AT RAISED PRESSURE IN THE RAT  
**Hélène N. David, J. H. Abraini (France)**

11:45 A04 AEROBIC ENDURANCE TRAINING REDUCE BUBBLE FORMATION AND INCREASE SURVIVAL  
IN RATS EXPOSED TO HYPERBARIC PRESSURE  
**Ulrik Wisløff, A.O. Brubakk (Norway)**



- 12:00 A05 EFFECT OF SINGLE DRY AND WET DIVES TO 50 METERS IN SPORT DIVERS ON ERYTHROPOIETIN (EPO) PLASMA CONCENTRATIONS  
**Till S. Mutzbauer, A. Gruenes, B. Neubauer, I. Lorenz, M. Weiss, M. Schneider, K. Tetzlaff (Germany)**
- 12:15 A06 NO EVIDENCE OF PERSISTING EXPIRATORY FLOW LIMITATION IN DIVERS AFTER REPEATED OXYGEN REBREATHING DIVING  
**Till S. Mutzbauer, B. Neubauer, K. Tetzlaff (Germany)**
- 12:30 A07 COMBINED INFLUENCES OF BREATH-HOLD, VALSALVA MANEUVER, AND INTENSIVE EXERCISE ON HEART RATE AND BLOOD PRESSURE  
**Uwe Hoffmann, M. Smerecnik, P. Buttgerit, D. Leyk (Germany)**
- 12:45 A08 UNDERWATER ACOUSTICS AND NEUROLOGICAL DECOMPRESSION ILLNESS: NEUROPHYSIOLOGICAL EVALUATION IN A RAT MODEL.  
**Avi Shupak, D. Tal, Y. Arieli, H. Pratt (Israel)**

**13:00 – 14:00 BREAK FOR LUNCH**

**14:00 – 15:15 SESSION B**

**DIVING PHYSIOLOGY II**

Chairpersons: **Alessandro Marroni, Italy**  
**Wilhelm Welslau, Germany**

**14:00 KEYNOTE LECTURE**

BEYOND THE ABYSS: PHYSIOLOGICAL RESTRICTIONS TO VERY DEEP OPERATIONAL DIVING  
**Jürgen Wenzel**  
**DLR-Institute of Aerospace Medicine, Linder Höhe, Cologne, Germany**

- 14:30 A09 SUBMARINE ESCAPE FROM 25 ATA USING HYPEROXIC ESCAPE GAS  
**Michael Gennser, S. L. Blogg, G. A. M. Loveman, F. M. Seddon, J. C. Thacker, M. G. White (Sweden)**
- 14:45 A10 INFLUENCE OF RADICAL SCAVENGER VITAMINS ON ERYTHROPOIETIN (EPO) PLASMA CONCENTRATIONS IN TRAINED SUBJECTS AFTER OXYGEN DIVING  
**Till S. Mutzbauer, A. Gruenes, B. Neubauer, I. Lorenz, M. Weiss, M. Schneider (Germany)**
- 15:00 A11 REALISATION OF A DOPPLER ULTRASOUND DEVICE FOR UNDERWATER ASSESSMENT OF BLOOD FLOW AND DETECTION OF BUBBLE FORMATION  
**Raschied Schabana, S. Hamich, P. Radermacher, C-M. Muth, K. Paulat (Germany)**

**15:15 – 17:15 SESSION C**

**HYPERBARIC OXYGEN THERAPY - PHYSIOLOGY**

Chairpersons: **Jordi Desola, Spain**  
**Hendrik Liedtke, Germany**

- 15:15 A12 EFFECT OF HYPERBARIC OXYGEN (HBO) THERAPY ON NEUTROPHIL ACCUMULATION AND AREA OF ISCHEMIC TISSUE DAMAGE DURING PERMANENT CEREBRAL ISCHEMIA IN RATS  
**Astrid Hjelde, M. Hjelstuen, O. Haraldseth, S.R. Thom, A. O. Brubakk (Norway)**

- 15:30 A13 ALTERATIONS OF ARTERIAL BLOOD GASES AFTER HYPERBARIC OXYGEN THERAPY IN CRITICALLY ILL PATIENTS  
**Beatrice Ratzenhofer-Komenda, A. Offner, S. Gabor, H. Klemen, F. Quehenberger, P. Spornbauer, J. H. Fadaï, U. Romirer, G. Prause, F.-M. Schmolle-Jüttner (Austria)**
- 15:45 A14 EXTRACELLULAR SUPEROXIDE DISMUTASE CONTRIBUTES TO INCREASED CEREBRAL BLOOD FLOW AFTER HYPERBARIC OXYGEN  
**Ivan T. Demchenko, T. D. Oury, J. D. Crapo, C. A. Piantadosi (USA)**

#### **16:00 – 16:15 COFFEE BREAK**

- 16:15 A15 HUMAN BEHAVIOURAL THERMOREGULATION DURING HYPOXIC EXPOSURE  
**Petra Golja, I. B. Mekjavić (Slovenia)**
- 16:30 A16 HYPERBARIC OXYGENATION AND SPONTANEOUS ELECTRICAL ACTIVITY OF MONKEY BRAIN  
**Nicola Dekleva (Yugoslavia)**
- 16:45 A17 MONITORING OF BRAIN ENERGETIC METABOLISM UNDER NORMOBARIC NARCOSIS, COMPARISON BETWEEN UNIQUE AND MULTIPLE EXPOSURES: A MULTI-PROBE MICRODIALYSIS STUDY IN FREE-MOVING RAT  
**Aurélien Moulins, F. Escalettes, J. Reybaud, J.-L. Méliet, J.-J. Risso (France)**
- 17:00 A18 MECHANISMS OF HYPERBARIC OXYGEN (HBO) – INDUCED ADAPTIVE RESPONSE  
**Andreas Rothfuß, P. Radermacher, G. Speit (Germany)**

#### **17:15 – 18:30 SESSION D**

##### **CLINICAL HYPERBARIC OXYGEN THERAPY I**

Chairpersons: **Robert C. Bornmann, USA**  
**Leon J. Greenbaum, USA**

- 17:15 A19 DOCUMENTATION OF THE EFFECT OF HYPERBARIC OXYGENATION FOR CEREBRAL PALSY AND THE BRAIN INJURED CHILD – SEQUENTIAL SPECT IMAGING  
**Richard A. Neubauer (USA)**
- 17:30 A20 HYPERBARIC TREATMENT OF NECROTISING SOFT TISSUE INFECTIONS: HOW MANY SESSIONS ARE NECESSARY?  
**Joachim Hencke, A. Jakopin, C.-M. Grosse-Wietfeld, R. Wetsch (Germany)**
- 17:45 A21 HYPERBARIC OXYGEN IN THE TREATMENT OF THERMAL BURNS – EXPERIENCES WITH CHILDREN AND ELDERLY PEOPLE  
**Heiko Renner, C. May, S. Gabor, H. Klemen, B. Hellbom, U. Anegg, A. Maier, H. Pinter, F.-M. Smolle-Jüttner (Austria)**
- 18:00 A22 APPLICATION OF THE HBO THERAPY BY CROHN DISEASE WITH FISTULA FORMATION  
**Oliver W. Jacobs, J. Rebhen, M. K. Müller (Germany)**
- 18:15 A23 HYPERBARIC OXYGEN THERAPY ON 17<sup>TH</sup> OF AUGUST EARTHQUAKE IN MARMARA  
**Şenol Yildiz, S. Özkan, K. Dündar (Turkey)**

#### **19:00 – 20:00 WELCOME RECEPTION at the Hotel Inter Continental**

#### **20.00 EVENING AT LEISURE**

**Scientific Programme**  
**FRIDAY, SEPTEMBER 14, 2001**

**(09:00 – 18:00 Full Day Excursion 2<sup>nd</sup> International “Arthur-Bornstein.Workshop” on Compressed Air Work and Deep Diving in Tunnel Construction Workshop Participants)**

**09:00 – 11.00 SESSION E**

**DIVING MEDICINE II (DAN EUROPE SECTION)**

Chairpersons: **Ioannis Polichronidis, Greece**  
**Kay Tetzlaff, Germany**

**09:00 KEYNOTE LECTURE**

**DIVERS ALERT NETWORK EUROPE – LOOKING BACK OF TEN YEARS ACTIVITIES**  
**Alessandro Marroni**  
**DAN Europe Operations Headquarters, Roseto, Italy**

- 09:30 A24 INFLUENCE OF SYMPATHIC INNERVATION ON THE HEART RATE DURING SCUBA DIVING. PRELIMINARY RESULTS  
**Stefaan Deneweth, H. Vanbogaert, M. Lambrechts, A. Marroni, R. Cali Corleo, C. Balestra, P. Germonpré (Belgium)**
- 09:45 A25 ENHANCEND LYMPHATIC CAPTATION OF PROTEINS DURING NORMOBARIC OXYGEN BREATHING. A LYMPHOSCINTIGRAPHY STUDY  
**Thyl Snoeck, M. Ezquer, O. Leduc, A. Leduc, F. Willeput, C. Ballestra, A. Marroni, R. Cali Corleo, P. Germonpré (Belgium)**
- 10:00 A26 THE USE OF A „PROPORTIONAL M-VALUE REDUCTION CONCEPT“ (PMRC) CHANGING THE ASCENT PROFILE WITH THE INTRODUCTION OF EXTRA DEEP STOPS REDUCES THE PRODUCTION OF CIRCULATION VENOUS GAS EMBOLI AFTER COMPRESSED AIR DIVING. DSL SPECIAL PROJECT 01/2001  
**Alessandro Marroni, R. Cali Corleo, C. Balestra, P. Longobardi, P. Germonpré, E. Voellm, M. Pieri, R. Pepoli (Italy)**
- 10:15 A27 THE SPEED OF ASCENT DILEMMA: “INSTANT SPEED OF ASCENT” OR ”TIME TO SURFACE” – WHICH ONE REALLY MATTERS?  
INSTANT SPEED OF ASCENT VS. DELTA-P IN THE LEADING TISSUE AND POST-DIVE DOPPLER BUBBLE PRODUCTION. DSL SPECIAL PROJECT 02/2001  
**Alessandro Marroni, R. Cali Corleo, C. Balestra, P. Longobardi, P. Germonpré, E. Voellm, M. Pieri, R. Pepoli (Italy)**
- 10:30 A28 INCIDENCE OF ASYMPTOMATIC CIRCULATING VENOUS GAS EMBOLI IN UNRESTRICTED, UNEVENTFUL RECREATIONAL DIVING. SKIN COOLING APPEARS TO BE RELATED TO POST-DIVE DOPPLER DETECTABLE BUBBLE PRODUCTION. AN UNEXPECTED FINDING. DSL SPECIAL PROJECT 03/2001  
**Alessandro Marroni, R. Cali Corleo, C. Balestra, P. Longobardi, P. Germonpré, E. Voellm, M. Pieri, R. Pepoli (Italy)**
- 10:45 A29 CEREBRAL LACUNARY SPOTS IN RELATION TO PATENCY OF FORAMEN OVALE, AN MRI INVESTIGATION  
**Brigitte Farkas, E. Duboc, C. Pietrons, J. Widelec, F. Vanderschueren, C. Balestra, A. Marroni, R. Cali Corleo, P. Germonpré (Belgium)**

**11:00 – 11.30 COFFEE BREAK**

**11:30 – 13:15 SESSION F**

**APNEA DIVING, HYPOTHERMIA**

Chairpersons: **Jean-Louis Méliet, France**  
**Peter Müller, Germany**

**11:30 KEYNOTE LECTURE**

**NEW DETAILS IN APNEA DIVING PHYSIOLOGY**  
**Peter Radermacher**  
**University of Ulm, Ulm, Germany**

- 12:00 A30 INSPIRED AIR TEMPERATURE AND HUMIDITY DO NOT INFLUENCE BRAIN STEM TEMPERATURE: IMPLICATIONS FOR INHALATION REWARMING THERAPY OF HYPOTHERMIC VICTIMS  
**Igor B. Mekjavic, K. Rogelj, M. Radobuljac, O. Eiken (Slovenia)**
- 12:15 A31 HUMAN BEHAVIOURAL THERMOREGULATION DURING HYPOXIC EXPOSURE  
**Petra Golja, I. B. Mekjavic (Slovenia)**
- 12:30 A32 RESCUE LIFTING SYSTEM AND SARRRAH-PROJECT: RECOVERY DEVICE FOR THE RESCUE OF DIVERS AND MEN-OVERBOARD IN ACCIDENTAL HYPOTHERMIA  
**Wolfgang Baumeier, M. Schwindt, L. Bahlmann, P. Schmucker (Germany)**
- 12:45 A33 ARTERIAL BLOOD GAS PARTIAL PRESSURES DURING DIVING IN ELITE APNEA DIVERS  
**Enrico Calzia, A. Pittner, R. Schabana, S. Hamich, P. Radermacher, C.-M. Muth (Germany)**
- 13:00 A34 NEW METHOD FOR RESCUER ARTIFICIAL VENTILATION SUITABLE FOR ENVIRONMENTAL CONDITIONS WITH REDUCED AMBIENT PRESSURE  
**Till S. Mutzbauer, B. Neubauer, P.H.J. Mueller, K. Tetzlaff (Germany)**

**13:15 – 14:45 BREAK FOR LUNCH**

**14:45 – 16.30 SESSION G**

**DIVING MEDICINE II**

Chairpersons: **Ramiro Cali-Corleo, Malta**  
**Wilfried Beuster, Austria**

- 14:45 A35 ROLE OF PATENT FORAMEN OVALE (PFO) AND MRI LESIONS IN UNEXPLAINED DIVING ACCIDENTS (DCS II)  
**Andreas Koch, H. Kirsch, A. Rump, E. Bettinghausen, H. Rieckert (Germany)**
- 15:00 A36 NO INCREASED INCIDENCE OF PATENT FORAMEN (PFO) IN UNEXPLAINED DIVING ACCIDENTS (DCS II) WITHOUT OBVIOUS MRI-LESIONS  
**Andreas Koch, A. Rump, P. McCormack, E. Bettinghausen, H. Rieckert (Germany)**
- 15:15 A37 THE RISK OF DECOMPRESSION SICKNESS CAUSED BY HYPOBARIC ENVIRONMENT EXPOSURE AFTER DIVING (DRIVING TO ALTITUDE AND FLYING)  
**Nobuo Naraki, M. Mohri (Japan)**
- 15:30 A38 MEDICAL PROBLEMS IN RECREATIONAL DIVERS IN YUGOSLAVIA: ASSESSMENT OF FITNESS TO DIVE AND ANALYSIS OF DIVING RELATED INJURIES DURING THE PERIOD 1994-1998  
**Dragana Ivkovic, N. Dekleva, N. Dikic, M. Zarkovic, B.M. Zivojnovic, M. Djuknic, Z. Vidovic, A. Marroni (Yugoslavia)**

15:45 A39 THE INFLUENCE OF INCREASED PRESSURE AND INCREASED OXYGEN PARTIAL PRESSURE ON THE CORROSION OF DENTAL ALLOYS  
**Frank Heblich, R. Lenz, K. Ludwig (Germany)**

16:00 A40 MANAGEMENT OF A COMPLEX DIVING ACCIDENT – A CASE REPORT OF RELAPSED ARTERIAL GASEMBOLISM (VIDEO-PRESENTATION)  
**Armin Kemmer, Adel Taher (Germany)**

**16:15 – 16:45 COFFEE BREAK**

**16:45 – 18.00 SESSION H**

**HYPERBARIC OXYGEN TECHNOLOGY & VALIDATION**

Chairpersons: **Costantino Balestra, Belgium**  
**Alexis Rump, Germany**

**16:45 KEYNOTE LECTURE**

CRISIS OF HYPERBARIC OXYGEN THERAPY IN GERMANY – ORIGIN, DEVELOPMENT, CURRENT IMPACTS AND POSSIBLE FUTURE  
**Ulrich van Laak**  
**President German Society of Diving and Hyperbaric Medicine (GTÜM)**

17:30 A41 PROJECT HORTIS: INVESTIGATING RADIOTHERAPY-INDUCED LATE EFFECTS OF NORMAL TISSUE WITH HYPERBARIC OXYGENATION  
**Dick Clarke (USA)**

17:45 A42 THERAPEUTICAL HYPERBARIC FACILITIES IN EUROPE ARE MEDICAL DEVICES: CONSEQUENCES  
**Robert Houman (Belgium)**

**18:30 DEPARTURE FOR RECEPTION & “RICKMER RICKMERS”**

**19:00 RECEPTION BY THE MAYOR OF HAMBURG**

**20:00 – 24:00 SOCIAL EVENING HISTORICAL “RICKMER RICKMERS”**

## Scientific Programme

### SATURDAY, SEPTEMBER 15, 2001

#### 09:00 – 10.30 SESSION I

##### POSTER PRESENTATIONS & DISCUSSION

Chairpersons: **Jürg Wendling, Switzerland**  
**Claus-Martin Muth, Germany**

- A43 NECROTISING FASCIITIS, TRUNCAL AND NECK INVOLVEMENT, SUCCESSFULLY TREATED SURGICALLY, ANTIBIOTICS AND HYPERBARIC OXYGENATION: A CASE REPORT  
**Dusko Micevic, M. Dragan, S. Vojkan, S. Vladislav, B. Ivanko, R. Slobodan (Yugoslavia)**
- A44 HYPERBARIC OXYGEN THERAPY AS A TREATMENT MODALITY FOR CALCIPHYLAXIS  
**Andreja Marn Pernat, R. Kveder, A. F. Bren, R. Ponikvar, I. B. Mekjavic (Slovenia)**
- A45 NEUROPROTECTION BY THE USE OF HYPERBARIC OXYGENATION AFTER PERMANENT FOCAL CEREBRAL ISCHEMIA  
**Holger Schade, W.-R. Schäbitz, R. Kollmar, J. Bardutzky, S. Heiland, S. Schwab, U. Carl (Germany)**
- A46 LEIPZIG'S HBO AMBULANCY: FINANCIAL ASPECTS  
**Harald Englisch, D. Kühnert (Germany)**
- A47 STRESS-INDUCED HORMONAL AND MOOD RESPONSES IN SCUBA DIVERS: A FIELD STUDY.  
**Udo Anegg, G. Dietmaier, A. Maier, F. Tomaselli, S. Gabor, K.-W. Kallus, F.-M. Schmolle-Jüttner (Austria)**
- A48 MEASUREMENT OF THE RADIOACTIVITY OF THE ADRIATIC SEA IN BOMBED AREA OF CAPE ARZA, BOKA BAY, MONTENEGRO, YUGOSLAVIA  
**Nenad Dikic, B.M. Zivojnovic, D. Ivkovic, S. Pavlovic, R. Pavlovic, M. Savovski, P. Radivkovic, S. Crnkovic, B. Markovic, N. Dekleva (Yugoslavia)**
- A49 EARLY 20<sup>TH</sup>-CENTURY HYPERBARIC RESEARCH IN HAMBURG - THE WORKS OF ARTHUR AND ADELE BORNSTEIN  
**Karl-Peter Faesecke (Germany)**
- A50 FIRE FIGHTING COURSE IN THE HYPERBARIC ENVIRONMENT  
**Peter Germonpré, R. Houman (Belgium)**

#### 10:30 – 11.00 COFFEE BREAK

#### 11:00 – 12:15 SESSION J

##### CINICAL HYPERBARIC OXYGEN THERAPY II

Chairpersons: **Peter Radermacher, Germany**  
**Clemens Mader, Austria**

#### 11:00 KEYNOTE LECTURE

THE ROLE OF HYPERBARIC OXYGEN THERAPY IN RADIOTHERAPY

**Ulrich M. Carl**  
**Diakoniekrankehaus Rotenburg/W., Germany**

- 11:30 A51 HYPERBARIC OXYGEN THERAPY: TREATMENT RESULTS IN THERAPY RESISTANT POSTIRRADIATION COMPLICATIONS IN PROSTATE TUMORS  
**Heiko Renner, R. Mayer, S. Gabor, H. Klemen, A. Hackl, F.-M. Smolle-Jüttner (Austria)**



- 11:45 A52 LATE RADIATION SEQUELAE IN WOMEN AFTER BREAST CONSERVING CANCER THERAPY: EFFECTS OF HYPERBARIC OXYGEN THERAPY (HBO)  
**Ulrich M. Carl, J. J. Feldmeier, G. Schmitt, K. A. Hartmann (Germany)**
- 12:00 A53 EFFECTS OF HYPERBARIC OXYGEN THERAPY (HBO) AND NORMOBARIC CARBOGEN ON THE RADIATION RESPONSE OF THE RHABDOMYOSARCOMA R1H  
**Ulrich M. Carl, K. A. Hartmann, A.J. van der Kleij, P. Sminia (Germany)**

**12:15 – 14:00 BREAK FOR LUNCH**

**14:00 – 17.00 SESSION K**

**CLINICAL HYPERBARIC OXYGEN THERAPY III**

Chairpersons: **Martin Hamilton-Farrell, England**

**Ulrich M. Carl, Germany**

- 14:00 A54 HYPERBARIC OXYGEN FOR THE TREATMENT OF IATROGENIC ARTERIAL GAS EMBOLISM: CASE REPORT  
**Michaela Ignatescu, P. Longobardi, F. Sfogliafferri, F. De Pasquale, S. Maitan, C. Olivieri, M. Baccanelli (Italy)**
- 14:15 A55 A PILOT STUDY OF HYPERBARIC OXYGEN THERAPY IN PATIENTS WITH FAECAL INCONTINENCE ASSOCIATED WITH PUDENDAL NERVE NEUROPATHY. EARLY RESULTS  
**Gerard Laden, J. Cundall, A. Gardiner, P. Grout, G. S. Duthie (England)**
- 14:30 A56 HYPERBARIC OXYGEN THERAPY IMPROVES CLINICAL EFFECTS OF TRANSMYOCARDIAL LASER REVASCULARISATION AND CABG  
**Jacek Kot, Z. Sicko, L. Anisimowicz, M. Narkiewicz, W. Pawlitzak, P. Betlejewski (Poland)**
- 14:45 A57 THE ROLE OF HBO-THERAPY IN THE TREATMENT OF OSTEOMYELITIS OF THE JAW. A RETROSPECTIVE ANALYSIS OF PATIENTS TREATED AT THE DEPARTMENT OF CRANIO-MAXILLOFACIAL SURGERY IN ZÜRICH (1988-2000).  
**Marc Baltensperger, O. Keller, A. Gönen, K. Grätz, J. Schmutz (Switzerland)**

**15:00 – 15:15 COFFEE BREAK**

**15:15 INVITED LECTURE**

MEDICAL CHALLENGES DURING THE CONSTRUCTION OF THE RECENTLY COMPLETED LATEST HAMBURG ELBE-TUNNEL

**Karl-Peter Faesecke**

HyperMedConsult, Hamburg

**16:00 EUBS CLOSING CEREMONY**

**16:30 GTÜM Executive Committee Business Meeting**

**19:30 DEPARTURE FOR RECEPTION “GRÖNINGER BREWERY”**

**20:00 – 24:00 FAREWELL EVENING AT “GRÖNINGER BREWERY”**



**SSMSH** SOCIÉTÉ SUISSE DE MÉDECINE  
SUBAQUATIQUE ET HYPERBARE  
**SGUHM** SCHWEIZERISCHE GESELLSCHAFT  
FÜR UNTERWASSER- UND HYPERBARMEDIZIN  
**SSMS** SOCIETÀ SVIZZERA DI MEDICINA  
SUBAQUEA ED IPERBARICA  
**SUHMS** SWISS UNDERWATER &  
HYPERBARIC MEDICAL SOCIETY



## Das neue Manual „Tauchtauglichkeit“ von GTÜM, SGUHM und ÖGTH

**Präsentation und Workshop in deutscher Sprache  
im Anschluss an das EUBS 2001 Annual Scientific Meeting in Hamburg**

**Sonntag, 16. September 2001, 0900 bis 1600 Uhr, Hotel Inter Continental Hamburg**

### Programm

Grußwort der Präsidenten GTÜM, SGUHM und ÖGTH  
Präsentation des Projektes eines gemeinsamen Manuals  
Der Weg zu gemeinsamen Standards  
Risiken und Gefahren beim Tauchen  
Der Beitrag der medizinischen Tauglichkeitsuntersuchung zur Tauchsicherheit  
Gibt es allgemein gültige Kriterien für die Tauglichkeit?  
Tauchtauglichkeit zwischen Beruf und Hobby  
Schlaglichter aus dem Tauchtauglichkeit Manual (Diskussion mit Beteiligung des Auditoriums):

- Probleme rund um den Schwindel
- Wie viel Kondition ist genug ?
- Wie gefährlich ist ein PFO ?
- Air-trapping: Lungenkrankheit oder Tauchunfallereignis?
- Trotz Asthma in die Tiefe ?
- Parästhesien und Tauchen
- Wie geht es weiter nach Tauchunfall ?
- Ist ein gelähmter Patient tauchtauglich ?
- Wie erkenne ich die Prädisposition zur Panik ?
- Der Spontanpneumothorax als Ausschlussgrund ?
- Wie geht es weiter nach Schädel-Hirn-Trauma ?
- Diabetes als Handicap
- Wer ist zu jung ?
- Wer ist zu alt ?
- Medikamenteneinnahme als Kontraindikation

Die Richtlinien für den Untersuchungsgang  
Kosten der Tauchtauglichkeitsuntersuchung  
Das Tauglichkeitsattest  
Ausblick und Abschluss

### Referenten und Diskussionsteilnehmer

Oskar Ehm, Jürg Wendling, Uwe Hoffmann, Peter Müller, Frank Böhm, Peter Nussberger, Wolfgang Förster, Karl-Peter Faesecke, Wilfried Beuster, Jürgen Wenzel, Ulrich van Laak, u. a., sowie das Auditorium.

### Zielgruppe Teilnehmer

Ärzte, die nach den Richtlinien der drei deutschsprachigen tauchmedizinischen Fachgesellschaften Tauchtauglichkeitsatteste ausstellen, interessierte Gäste.

### Kosten

Für die Teilnehmer des EUBS 2001 Annual Scientific Meetings FREI – für Nicht-Teilnehmer des EUBS 2001 Annual Scientific Meetings sind Tageskarten zum Preis von **€ 80,00** erhältlich.

### Anmeldung

- Schriftlich über „Registration Form EUBS 2001“
- On-line über [www.eubs2001.de](http://www.eubs2001.de)

## Abstracts of the 27<sup>th</sup> EUBS Annual Scientific Meeting Sessions September 12-16, 2001 Hamburg, Germany

The following abstracts are the abstracts submitted by authors of the papers they will present in slide or poster sessions at the 27<sup>th</sup> annual meeting of the European Underwater and Baromedical Society

Each abstract is numbered in chronological order as they appear in the Programme.

**A01 METHOD FOR QUANTIFYING MECHANICAL ENDOTHELIAL DAMAGE.** Vibeke Nossum, Alf Brubakk. Department of Physiology and Biomedical Engineering, Norwegian University of Science and Technology, N-7489 Trondheim, Norway.

**Background:** Several previous studies have shown that gas bubbles can lead to mechanical damages as cracks and wounds in the endothelial layer of the blood vessel wall. As the number of gas bubbles increases, the possibility for endothelium damage also increases and this damage seems to be related to the amount of gas present. This also leads to a change in endothelial vasoactive response. Even if it is well documented that gas bubbles can damage the endothelium, there is a need to develop methods that can quantify this mechanical damage. We report here a method that combines silver nitrate staining with image processing to quantify the exact amount of mechanical damage in the endothelial layer in a blood vessel.

**Materials and methods:** Blood vessels (mainly pulmonary artery) were harvested from pigs (8-11 wk) that had undergone decompression. They had different levels of endothelial damage caused by different amount of gas bubbles and some of the endothelium was even removed by gentle rubbing of the intimal surface with a wooden stick. The arteries were divided into segments for measurements of the endothelial-dependent vasodilatation. Afterwards, the segments were cut open in strip form and stained with silver nitrate (0.25 % AgNO<sub>3</sub>) after standard methods. Areas of injury are characterised with diffuse silver staining or decreased staining intensity. Each of the segments was then examined by light microscopy and photographed at 250x. The quantification process took place in an image-processing program called Adobe PhotoShop. To ensure that the method was reliable the determination of damaged endothelium was repeated 3 times for each picture over a period of one year.

**Results:** The quantification of mechanical damage in each animal where repeated three times for every picture throughout a year and the final results gave small differences between each quantification. A comparison of tension measurements and mechanical endothelial disruption showed correlation between relaxation response and endothelial integrity. It can be seen that there is good preservation of the endothelium and that the ruptured area clearly shows.

**Conclusion:** The method demonstrates mechanical damage in the endothelial layer. The quantification makes it easy to compare biochemical and mechanical endothelial damage in the same vessel segment and gives a measure of the mechanical damage observed. Results from three quantifications of the same segment showed little variation and reliable reproducibility. This method is a contribution to further investigations to determine mechanisms behind reduced endothelial response caused by gas bubbles.

**A02 EXTRACELLULAR DEHYDRATION INDUCES FUNCTIONAL MOTOR SPINAL DEFICIT AFTER SEVERE DECOMPRESSION IN THE RABBIT.** Jean-Louis Méliet, Philippe Léni †, Jean-Pierre Menu, Franck Laforest, Boualem Zouani. I.M.N.S.S.A., B.P. 610, F-83800 Toulon Naval, France.

**Background:** Spinal cord injury (SCI) is a frequent occurrence during decompression sickness (DCS), combining varied sensitive and motor deficits. The motor dysfunction is regarded as a pejorative gravity factor. On the other hand, high haematocrit levels during DCS are correlated with persistent neurological sequelae. Although we showed in a previous paper (Léni & al., EUBS 99, Haïfa) that haemoconcentration is not required for the generation of SCI in DCS, we investigated the consequences of a pre-existing extracellular dehydration on the development of spinal neurological deficits in an animal model of DCS.

**Methods:** The effects of a rapid decompression were studied in furosemide pre-treated white NZ male conscious rabbits compared to control groups. A first group (control-decompression, CD, n=8) was submitted to compressed air at 6 ATA for 25 min and rapidly decompressed. A second one (control-furosemide, CF, n=8) was given furosemide (5 mg/kg IM) then left 30 min in the decompression chamber at atmospheric pressure. The third group (furosemide-decompression, FD, n=8) was given furosemide in the same conditions than CF then compressed and decompressed 30 min later with the same profile than CD. Clinical (inclined plan) and electrophysiological (somatosensory evoked potentials, SEPs, and motor evoked potentials, MEPs) tests were recorded 24 hours later.

**Results:** Results clearly show that animals submitted to DCS alone or haemoconcentration (assessed by plasmatic protein PP and haematocrit Ht levels, +17% and +9.8% respectively) alone did not show any significant sign of neurological disorder. On the opposite, the animals of the FD group (PP : +10%, Ht : +4.8%) revealed significant increase in MEP latency (+10.5%) without any change of MEP amplitude, nor clinical or SEP parameters.

**Discussion:** The reasons why haemoconcentration makes motor alterations more likely than sensitive remain unclear : rheological disturbances could act by producing ischemia. But SEPs, the most sensitive parameters, are not modified. MEPs latency changes are most often linked to myelin degeneration. We could consider in this case that a modification in the spinal motor pathway conduction is produced either by blood flow reduction or electrolytic changes in the interstitial fluid, or both, leading directly or indirectly to myelin functional alterations. The participation of these different mechanisms deserves to be elucidated in further investigations.

**Grants:** French Ministry of Defence (DGA/DRET 20-96) – MEDSUBHYP.

† *In memoriam...*

#### **A03 EFFECTS OF SELECTIVE GABA RECEPTOR LIGANDS ON NARCOSIS PRODUCED BY NITROGEN, ARGON AND NITROUS OXIDE AT RAISED PRESSURE IN THE RAT.** Hélène N. David and Jacques H. Abraini. Université de Caen, Caen, France

Although the traditional view has been that general anaesthetics dissolve in the lipid bilayer of the cellular membrane, occupying or expanding its volume and thereby disrupting its dynamic properties, it has been reported that inhalational anaesthetics, including the inert gases xenon and nitrous oxide, would interact directly with ion-channel neurotransmitter. However, there is no evidence that inert gases, such as argon or nitrogen, that only exhibit narcotic potency at raised pressure may act by similar mechanisms. In the present study, we compared the effects of intracerebroventricular infusion of highly selective GABA receptor ligands on the loss of righting reflex produced by nitrogen, argon, and nitrous oxide at raised pressure in the rat.

Pretreatment with the GABA<sub>A</sub> receptor antagonist SR95531 resulted in a significant increase of both the nitrogen effective pressure (EP<sub>50</sub>) and argon EP<sub>50</sub> ( $P < 0.005$ ), but not of the nitrous oxide EP<sub>50</sub>. Pretreatment with the GABA<sub>A</sub> inverse agonist flumazenil also reduced in a modest, but significant, increase of the argon EP<sub>50</sub> ( $P < 0.025$ ), but showed no effect on the nitrogen- and nitrous oxide EP<sub>50</sub>. In contrast, pretreatment with the GABA<sub>B</sub> receptor antagonist 2-hydroxysaclofen had no significant effect on neither the nitrogen, argon, nor nitrous oxide EP<sub>50</sub> value.

These results suggest that the anaesthetic action of nitrogen and argon, but not of nitrous oxide, may involved GABA neurotransmission. This agrees with data that have suggested that nitrous oxide would act by inhibiting glutamate transmission rather than potentiating GABA transmission. At the view of the present data, we suggest that argon may produce its narcotic action at pressure by interacting directly with- and modulate allosterically the GABA<sub>A</sub> receptor at the benzodiazepine site. In addition, we hypothesise that nitrogen at raised pressure may also act at the ion-channel GABA<sub>A</sub> receptor but not at the benzodiazepine site.

#### **A04 Aerobic endurance training reduce bubble formation and increase survival in rats exposed to hyperbaric pressure.** Ulrik Wisløff and Alf O. Brubakk. Department of Physiology and Biomedical Engineering, Norwegian University of Science and Technology, Trondheim, Norway. Faculty of Medicine, Medical Technology Center, 7489 Trondheim, Norway.

**Background:** The formation of bubbles is the basis for injury to divers after decompression, called decompression illness. The present study investigated the effect of endurance training on bubble formation in the rat.

**Material and Methods:** A total of 52 adult female 300-370g Sprague Dawley rats were randomly assigned to either training or sedentary control. Trained rats exercised on a treadmill 1.5-hr per day for 1 day, 2 or 6-wk (5-d per week) at exercise intervals that alternated between 8-min at 85-90% of VO<sub>2max</sub> and 2-min at 50-60%. Rats were compressed in pairs, one sedentary and one trained, at a rate of 200 kPa/minute to a pressure of 700 kPa, maintained for 45 minutes breathing air. At the end of the bottom period rats were decompressed linearly to the surface at a rate of 50 kPa/minute. Immediately after surfacing the animals were anesthetized and the right ventricle was insonated using ultrasound.

**Results:** Intensity-controlled interval training significantly increased VO<sub>2max</sub> by 12 % and 60 % after 2 and 6 weeks. At 6 weeks, left and right ventricular weights were 14 % and 17 % higher in trained rats. No effect of training was observed on skeletal muscle weight. Bubble formation was significantly reduced in trained rats both after 2 and 6 weeks, the same effect was seen after training for 1.5 hours the day prior to decompression. All rats trained for 1.5 hour and 2 weeks, and most rats trained for 6 weeks survived the protocol, whereas most sedentary rats died within 60 minutes post decompression.

**Conclusions:** This study shows that aerobic endurance training protects rats from severe decompression and death. This is apparently a result of less bubbling in the trained animals. Data showed that the increase in aerobic capacity

per se was not the main mechanism, but rather an acute effect most notable after 20 hours with much less effect after 48 hours.

#### **A05 EFFECT OF SINGLE DRY AND WET DIVES TO 50 METERS IN SPORT DIVERS ON ERYTHROPOIETIN (EPO) PLASMA CONCENTRATIONS.** Till S. Mutzbauer<sup>1</sup>, Angelika Gruenes<sup>1,4</sup>, Birger Neubauer<sup>2</sup>, Ingrid Lorenz<sup>3</sup>, Manfred Weiss<sup>4</sup>, Marion Schneider<sup>4</sup>, Kay Tetzlaff<sup>5</sup>

<sup>1</sup>Dept. Anaesth. and Crit. Care, Armed Forces Med. Ctr., Ulm, <sup>2</sup>Office of Occupational Safety, Hamburg,

<sup>3</sup>Transfusion Medicine, Univ. Duesseldorf, Duesseldorf, <sup>4</sup>Dept. Anaesth. Univ. Ulm, <sup>5</sup>Naval Medical Institute of the German Navy, Kronshagen; Germany

**Specific objective:** EPO constitutes a hypoxia induced gene product, the expression of which is negatively influenced by oxidative stress and positively influenced by water immersion. The present study was performed to investigate the influence of short intervals of dry and wet hyperbaric environmental conditions on EPO plasma concentrations were evaluated.

**Materials and Methods:** Eight trained recreational divers were randomly allocated to perform a dive to 50 meters (.6 MPa ambient pressure) in the wet and the dry compartment of a hyperbaric chamber in a crossover design on different days. The bottom time was 15, total dive time 32 minutes. Before diving, after each dive and 24 hours later venous blood was obtained from the divers. EPO plasma concentrations were determined by a highly sensitive chemiluminescence immunoassay.

**Statistics:** Wilcoxon signed-rank test.

**Description of Results:** Both wet and dry environmental conditions revealed no differences comparing EPO plasma concentrations pre dive, post dive and 24 hours post dive.

conditions	pre dive		post dive		24 hrs post dive		EPO conc. mU/ml
	Median	Range	Median	Range	Median	Range	
dry	6.9	(4.0 - 10.4)	7.0	(4.2 - 11.0)	6.2	(3.8 - 9.0)	mU/ml
wet	6.5	(3.2 - 10.2)	5.8	(3.2 - 9.2)	5.4	(4.6 - 7.6)	

**Conclusions:** There is no evidence of effect on EPO plasma concentrations by hyperoxia generated during short exposure to hyperbaric environmental conditions. Furthermore, there is no evidence of submersion as a possible co-factor influencing EPO plasma concentrations under these conditions.

#### **A06 NO EVIDENCE OF PERSISTING EXPIRATORY FLOW LIMITATION IN DIVERS AFTER REPEATED OXYGEN REBREATHING DIVING.** Till S. Mutzbauer<sup>1</sup>, Birger Neubauer<sup>2</sup>, Kay Tetzlaff<sup>3</sup>.

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**Specific objective:** In cross-sectional studies reduced expiratory flow rates at low lung volumes have been measured in oxygen divers compared to control subjects. Aim of this study was to evaluate lung function changes after dives on closed circuit oxygen rebreathers.

**Materials and Methods:** Nine healthy male special forces divers performed four dives on three consecutive days using a LAR VI (Dräger, Lübeck, Germany) closed circuit oxygen rebreather according to standardized procedures. Forced expiratory volume in one second (FEV1) and expiratory flow rates at 50% (F50) and 25% (F25) of forced vital capacity were obtained from the divers in an upright position without dive equipment before and after the dives using a mobile MicroLoop Spirometer (Micro Medical Ltd. Rochester, UK).

**Statistics:** Data of subsequent measurements were compared by two-tailed Wilcoxon matched pairs test statistics.

**Description of Results:** Comparison of the initial pre-dive (1) values and the values obtained after the last dive (4) revealed no differences. As displayed in the table no progressive decline of FEV1 and expiratory flow rates at low lung volumes could be observed after subsequent dives. A simultaneous reduction of these parameters on a level of statistical significance could only be found after the first dive. Increasing F50s were detected after dive 2.

<b>FEV1</b>	Mean	4,54*	D	4,40	N	4,45	D	4,42		4,53	D	4,48*	N	4,59*	D	4,47
[L]	Std.Dev.	0,59	I	0,64	I	0,57	I	0,55		0,62	I	0,56	I	0,63	I	0,57
<b>F50</b>	Mean	4,68*	V	4,39	G	4,47*	V	4,70		4,71	V	4,55*	G	4,89*	V	4,44
[L/s]	Std.Dev.	1,09	E	1,05	H	1,03	E	1,04		0,83	E	0,86	H	0,99	E	0,71
<b>F25</b>	Mean	1,85*	1	1,66	T	1,74	2	1,88		1,94*	3	1,79*	T	1,96	4	1,84
[L/s]	Std.Dev.	0,43		0,41		0,37		0,36		0,34		0,37		0,36		0,28

\*indicates a difference between distributions of the marked and the subsequent measurement with  $p < 0.05$

**Conclusions:** Inconsistent changes in expiratory volumes and flows were found. There is no evidence of persisting lung function impairment related to repeated closed circuit oxygen rebreather diving.

**A07 COMBINED INFLUENCES OF BREATH-HOLD, VALSALVA MANEUVER, AND INTENSIVE EXERCISE ON HEART RATE AND BLOOD PRESSURE.** Uwe Hoffmann, M.Smerecnik, P.Buttgereit, D.Leyk. German Sport University, Cologne, Germany

**Background:** Breath-hold for several seconds and intensive dynamic exercise frequently occurs in several sportive activities. Moreover, the breath-hold is very often combined with a Valsalva maneuver. As consequence of these combined stresses different competing physiological reflexes and regulation processes are activated: Breath-hold initiates O<sub>2</sub> conserving mechanisms; the Valsalva maneuver has a direct impact on arterial pressure and on blood flow to and from the heart; dynamic exercise leads to increases in both, mean arterial blood pressure (MBP) and heart rate (HR). We investigated the interactive effects of these regulation processes on heart rate and blood pressure.

**Methods:** 9 sports students participated in eight cycle ergometer sessions in upright and supine position. After a warm-up of 5 min exercise at 30 W workload was increased to 300 W for 30 s (HiWL) followed by a resting phase. HiWL was varied with regard to respiration: free, relaxed apnoe (AP), 20 mmHg (V20) and 40 mmHg (V40) Valsalva maneuver. MBP, and HR were monitored continuously. 3-way MANOVA and post-hoc comparison were applied.

**Results:** In the experiments with breath-hold HR showed a typical pattern: a decrease within the last seconds of HiWL (over-all maximum  $25.8 \pm 4.4$ s after onset of 300 W exercise), a minimum immediately after begin of recovery (up to 46 b/min lower than the preceding maximum), followed by a second relative maximum and minimum. In the breath-hold experiments a steep MBP increase up to  $153 \pm 24$ mmHg (V40 supine) occurring after maximum in HR. However, only minor differences were found between the different breath-hold regimen.

**Conclusions:** The O<sub>2</sub> conserving mechanisms during breath-hold superpose the exercise stimulus for HR and MBP. It can be speculated that vasoconstriction leads to a continual MBP increase compensated by a HR decrease. Neither central blood volume nor intrathoracic pressure seem to have a major impact on the regulation process under this conditions. Since breath-hold diving similar results are reported for heart rate responses it must be assumed that similar increases in blood pressure occur in those situations also.

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**A08 UNDERWATER ACOUSTICS AND NEUROLOGICAL DECOMPRESSION ILLNESS: NEUROPHYSIOLOGICAL EVALUATION IN A RAT MODEL.** Avi Shupak<sup>1</sup>, D. Tal<sup>1</sup>, Y. Arieli<sup>1</sup>, H. Pratt<sup>2</sup>. <sup>1</sup>Israel Naval Medical Institute and <sup>2</sup>The Evoked Potential Laboratory, Faculty of Medicine, Technion, Haifa 31080, Israel

**Background:** High-frequency sound at 30-40kHz, being in the resonance range of bubbles with a diameter in the range of 40-80 microns, might cause bubble enlargement by rectified diffusion. We have previously reported of alveolar damage to the immersed pig's lung exposed to high-frequency sound, increased number and larger gas bubbles in the mesenterium of rats after diving simulation while under high-frequency sound transmission, and similar observations in the prawn plasma (1-3). However, it is not yet known whether high-frequency sound-associated bubble enhancement might cause neurological damage. The purpose of the present study was to investigate the occurrence of possible neurological insult, as monitored by somatosensory evoked potential (SSEP) recording, secondary to high-frequency sound exposure in the rat model.

**Methods:** Anesthetized Sprague-Dawley rats were immersed in a customized designed temperature-controlled box filled with water inside a hyperbaric chamber. The animals were exposed to a diving profile on air for a bottom time of 60 minutes followed by 3ATA/min decompression. A pinger was placed 10 cm ventral to the animal's abdomen. Intermittent sound at an intensity of 184.5 dB re 1 Micropascal @ 1 meter and a frequency of 37kHz was transmitted in a duty cycle of 0.26%. Four groups, each containing 9 animals, were included in the study as follows:

Group	Depth	Sound exposure
1.	0 m	No
2.	0 m	Yes
3.	30 m	No
4.	30 m	Yes

Animals had a baseline somatosensory evoked potentials (SSEP's) recording the day before the immersion, and a second study within 30 minutes after it. SSEP's were obtained from the lumbar and cervical spinal cord, and from the parietal cortex via transcutaneous needle electrodes. The following parameters were collected and compared among the study groups: Waves latency, amplitude, and conduction times. Also, the number of animals in which SSEP waves disappeared on the second study was compared. One-way ANOVA was used to analyze the variance of the normalized differences in SSEP wave parameters between the two studies. The Fisher exact test was employed to evaluate the significance of SSEP waves disappearance.

**Results:** All SSEP waves were clearly recorded in the baseline study of all animals, and on the second study of animals belonging to groups 1 and 2. Some of the waves could not be identified after the diving simulation in 3 and



2 rats of groups 3 and 4 accordingly ( $p=0.09$ ). SSEP waves disappeared in a significantly larger number of animals from groups 3 and 4 when compared to groups 1 and 2 (5/18 and 0/18 accordingly;  $p=0.045$  2-tailed Fisher exact test). No differences in SSEP waves latency, amplitude and conduction time could be found.

**Conclusions:** The rat diving simulation model produced about 27% hits of SSEP monitored decompression illness. However, the high-frequency sound exposure employed did not contribute to the development of neurological insult. Differences in perfusion and tissular elastic pressures might explain lack of functional damage in the face of the previously described mesenterium bubble growth (2). Such differences might resist bubble growth or counterbalance possible local bubble-induced ischemia.

#### A09 SUBMARINE ESCAPE FROM 25 ATA USING HYPEROXIC ESCAPE GAS. Michael Gennser<sup>1</sup>, S. L. Blogg<sup>2</sup>, G. A. M. Loveman<sup>2</sup>, F. M. Seddon<sup>2</sup>, J. C. Thacker<sup>2</sup>, M. G. White<sup>2</sup>

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**Background:** Submarine escape from a submarine with raised internal pressure exposes the submariners to a high risk of decompression illness (DCI). It has been suggested that use of hyperoxic escape gas instead of air could reduce the risk of DCI. However, increased oxygen fraction in the breathing gas may cause oxygen convulsions. This experiment was undertaken to see if escape from 25 ata could be carried out with a 60/40 oxygen/nitrogen breathing gas mixture without causing oxygen convulsions.

**Methods:** 15 adult female or castrated male goats in the weight range 35-60 kg were used. Submarine escape profiles from 25 ata were carried out in a computer-controlled hyperbaric facility. During the escapes the animals were breathing either air or a 60/40 O<sub>2</sub>/N<sub>2</sub> mixture via a oro-nasal mask supplied with gas from the "hood-inflation system". Directly after the escape all animals were returned to air breathing. The animals were monitored with respect to breathing frequency, and end-tidal gas concentrations during the first 30 min after escape. Pre-cordial Doppler monitoring was carried out every 5 min during the first 30 min, every 15th min between 30 and 120 min, and then every hour.

**Results:** No hyperoxic convulsions were seen in any of the eight goats breathing nitrox gas during the escapes, despite a maximum inspired PO<sub>2</sub> of 1500 kPa, and PiO<sub>2</sub> > 400 kPa for 80 s. There were no cases of DCI. There was no difference in breathing frequency or gas exchange post-escape between the air and the nitrox group. The maximum bubble score was similar in the two groups of animals (air; median K-M score: 3, range 3 to 4, nitrox; median K-M score 3, range 3- to 4). However, the bubbles in the venous circulation disappeared faster in the nitrox group and the bubble score was lower in the nitrox group from 25 min post-escape and onwards until time to zero detectable bubbles (nitrox 43 min vs Air 110 min,  $p<0.05$ ).

**Conclusions:** Simulated submarine escapes can be carried out in goats from 25 ata while breathing 60/40 oxygen/nitrogen gas without causing oxygen convulsions. Bubbles disappeared faster after dives with hyperoxic gas. Further experiments will be carried out to see if breathing a hyperoxic gas mixture during escapes will reduce the risk of DCI after submarine escapes preceded by shallow air saturation.

#### A10 INFLUENCE OF RADICAL SCAVENGER VITAMINS ON ERYTHROPOIETIN (EPO) PLASMA CONCENTRATIONS IN TRAINED SUBJECTS AFTER OXYGEN DIVING. Till S. Mutzbauer<sup>1</sup>, Angelika Gruenes<sup>1,4</sup>, Birger Neubauer<sup>2</sup>, Ingrid Lorenz<sup>3</sup>, Manfred Weiss<sup>4</sup>, Marion Schneider<sup>4</sup>

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**Specific objective:** EPO constitutes a hypoxia induced gene product, the expression of which is negatively influenced by oxidative stress. It was tested whether radical scavenging vitamins C and E influence EPO plasma concentrations following short intervals of submersed physical activity under hyperoxic hyperbaric environmental conditions.

**Materials and Methods:** Eight trained divers took daily 1 g of ascorbic acid and 600 IU of d- $\alpha$ -tocopherol, and eight trained divers placebo, >3 h before diving with oxygen as breathing gas at average depths of 4 m for 30 min on 2 consecutive days, and additionally on day 3. Groups were comparable with respect to oxygen consumption during dives. Venous blood was obtained on days 1, 2 (pre-dive) & 3. EPO plasma concentrations were determined by a highly sensitive chemiluminescence immunoassay.

Group of Divers	day 1		day 2		day 3		EPO conc. mU/ml
	Median	Range	Median	Range	Median	Range	
Vitamins	7.4*	(3.4 - 10.4)	5.1*#	(3.4 - 9.4)	7.2#	(5.2 - 11.8)	mU/ml
Placebo	7.3*	(5.0 - 10.0)	5.2*	(2.8 - 8.8)	6.0	(2.4 - 11.8)	mU/ml

\* $p<0.05$  day 1 compared with day 2

# $p<0.05$  day 2 compared with day 3

**Statistics:** Wilcoxon signed-rank and Mann-Whitney U-test.

**Description of Results:** Decreasing EPO plasma concentration on day 2 compared to baseline was the leading feature in both groups ( $p=.046/.018$ ). Between days 2 & 3 an increase in EPO concentrations, in 50% even exceeding baseline, was observed in the vitamin ( $p=.009$ ) but not in the placebo group ( $p=.155$ ). No inter-group differences were found on days 1 and 2. EPO concentrations on day 3 differed at  $p=.06$ .

**Conclusions:** In the majority of divers, hyperbaric hyperoxia induced a persisting EPO decrease in the placebo, but a transient EPO decrease in the vitamin group. Radical scavengers may attenuate EPO decrease in response to repeated oxygen diving.

**A11 REALISATION OF A DOPPLER ULTRASOUND DEVICE FOR UNDERWATER ASSESSMENT OF BLOOD FLOW AND DETECTION OF BUBBLE FORMATION.** Raschied Schabana, Sven Hamich, Peter Radermacher<sup>1</sup>, Claus-Martin Muth<sup>1</sup>, Klaus Paulat. Abteilung Medizinische Regelungstechnik, Fachhochschule, and 1Sektion Anästhesiologische Pathophysiologie u. Verfahrensentwicklung, Universitätsklinik f. Anästhesiologie, Universität, Ulm, Germany

**Background:** Doppler ultrasound detection of intravascular bubble formation is used for the evaluation of decompression profiles. Up to now this technique has only been applied, however, after surfacing, since underwater bubble detection has not yet been possible. The absence of bubbles after diving does not per se exclude bubble formation during the dive. Therefore we modified a Doppler ultrasound device for underwater application in order to enable bubble detection already still underwater prior to surfacing. Furthermore, given the potential importance of a patent foramen ovale, we also sought to bubbles resulting from right-to-left shunting.

**Materials and Methods:** A commercially available ultrasound device for transcranial Doppler measurement of cerebral blood flow (Smart Dop®, DWL Elektronische Systeme GmbH) was modified for the use during diving while maintaining continuous signal recording and autonomous power supply. The device was tested in 5 healthy volunteers during the decompression phase of different diving profiles. The dives were performed in the wet tank of the pressure chamber of the Deutsche Lebens-Rettungs-Gesellschaft, Berlin, and consisted of simulated exposures to 33 – 50 m sw. Blood velocity was measured underwater in the subclavian and femoral vein as well as the middle cerebral artery.

**Results:** In each diver high quality signals were obtained in all vessels investigated. Doppler signal highly suggesting formation of multiple microbubbles was most pronounced during the respective decompression stops and occurred in particular after exposures to 50 msw. The Doppler signal recorded in this phase was similar to that obtained using classical ultrasound contrast medium. Hence, the size of the microbubbles detected probably did not exceed 5 – 7  $\mu\text{m}$  suggesting possible passage of the capillary vascular net without major bubble entrapment.

**Conclusions:** Using our device intravascular bubble detection is possible even during the decompression phase of a dive thereby potentially improving the safety evaluation of decompression procedures. In addition, the putative importance of a patent foramen ovale can even be assessed underwater.

**Acknowledgements:** Supported by the Deutsche Lebens-Rettungs-Gesellschaft and DWL GmbH

**A12 EFFECT OF HYPERBARIC OXYGEN (HBO) THERAPY ON NEUTROPHIL ACCUMULATION AND AREA OF ISCHEMIC TISSUE DAMAGE DURING PERMANENT CEREBRAL ISCHEMIA IN RATS.** Astrid Hjelde<sup>1,3</sup>, M. Hjelstuen<sup>3</sup>, O. Haraldseth<sup>2,3</sup>, S.R. Thom<sup>4</sup>, A. O. Brubakk<sup>1</sup>.

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**Background and aim:** Hyperbaric oxygenation (HBO) has been considered for many years as a treatment of severe brain ischemia, due to its ability to improve tissue oxygen delivery. However, its efficiency remains controversial. Studies to date suggest that HBO may interfere with the destructive neutrophil (PMN) infiltration response following ischemia/reperfusion (I/R). The aim is to study the effect of HBO on PMN accumulation in rats with permanent occlusion of one middle cerebral artery (MCA), and to identify the relation between PMN and the area of cerebral ischemic tissue damage.

**Materials and methods:** The effect of HBO therapy on PMN accumulation and area of ischemic tissue damage was investigated in a rat model of permanent ischemia. Permanent cerebral ischemia (4 hrs) was introduced in 15 rats using a MCA occlusion model. This was achieved using a nylon monofilament with a diameter of 0.27 mm and a rounded tip. The filament was introduced through the external carotid artery and advanced 19 to 20 mm, thus blocking the origin of the MCA. The nylon filament remained in place until all the animals were killed 4 hrs after the onset of MCA occlusion. The animals were randomly divided into two groups. In one group ( $n=7$ ) HBO treatment was performed 10 min after MCA occlusion (HBO group). The remaining 8 animals were not treated and served as a control group. Animals that underwent HBO were placed in a hyperbaric chamber that were pressurized to 2 atmospheres absolute (ATA) with 100% oxygen for 230 min. Compression and decompression was accomplished

during 1 min. The control animals were breathing room air at ambient pressure for an equivalent period of time. Magnetic resonance imaging (MRI) was used to assess the efficiency of HBO treatment on ischemic tissue damage. Myeloperoxidase (MPO) is an enzyme localized in PMNs, and by measuring its activity, the degree of PMN accumulation can be quantified.

**Results:** HBO had no effect on permanent ischemia, as no significant difference ( $P=0.817$ ) on the area of ischemic tissue damage was observed between HBO-treated ( $331.0 \pm 87.5 \text{ mm}^3$ ) and non-treated ( $321.7 \pm 111.3 \text{ mm}^3$ ) animals. However, an increase in MPO was found in the HBO-treated group vs. non-treated ( $5.4 \pm 4.1$  vs.  $2.4 \pm 1.2 \text{ pg/g}$  wet weight of brain), but this difference was not significant ( $P=0.224$ ).

**Conclusion:** The administration of HBO did not reduce cerebral tissue damage during permanent MCA occlusion. Thus, HBO may not be recommended as a therapy during permanent ischemia.

### A13 ALTERATIONS OF ARTERIAL BLOOD GASES AFTER HYPERBARIC OXYGEN THERAPY IN CRITICALLY ILL PATIENTS. Beatrice Ratzenhofer-Komenda<sup>1</sup>, Anton Offner<sup>1</sup>, Sabine Gabor<sup>2</sup>, Huberta Klemen<sup>2</sup>, Franz Quehenberger<sup>3</sup>, Paulus Spernbauer<sup>1</sup>, Josef Heydar Fadai<sup>1</sup>, Ulrike Romirer<sup>1</sup>, Gerhard Prause<sup>1</sup>, Freyja Maria Smolle-Jüttner<sup>2</sup>

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**Background:** Hyperbaric Oxygen (HBO<sub>2</sub>) modulates microvascular perfusion patterns of various organ systems like skin (1) or brain (2) the following study was based upon the clinical observation of a decline of the subjects' oxygenation state after the hyperbaric session. The study aimed at evaluating whether and to which extent blood gases were altered after the hyperbaric session in critically ill patients.

**Materials and methods:** After approval by the ethics committee of the university, 13 critically ill patients (11m, 2f; 19-66 yr) undergoing HBO<sub>2</sub> Therapy were included in the study (diagnoses: necrotizing fasciitis in 2pts., Postoperatively after major abdominal surgery in 7 pts., Burn injury in 2 pts. And carbon monoxide intoxication in 2 pts.). All subjects were mechanically ventilated in the simv mode (synchronized intermittent mandatory ventilation, peep: 5 mbar, mean inspiratory fraction of oxygen (fio<sub>2</sub>): 0.4) and received analgesia and sedation throughout the measuring period. Ventilator setting, fio<sub>2</sub> and catcholamine support before and after the hyperbaric session remained unchanged.

The hyperbaric protocol was run at 2.2 ata with an isopression phase of 50 min. Efficacy of ventilation (simv, peep: 5 mbar, fio<sub>2</sub>: 1.0) in the hyperbaric environment was monitored by blood gas analyses. On average, the subjects were studied after their 2<sup>nd</sup> hyperbaric session. Arterial blood gases (avl 995 blood gas analyzer, avl ges. M. B. H., Austria), mean arterial blood pressure (map), heart rate (hr) and body temperature were recorded before, one and two hours after the treatment. The subject's position was kept constant during the study period and no other intervention was scheduled for the day of the study.

**Statistical analysis:** repeated measure anova.

**Results:** see table 1. lower and upper limits of the 95% confidence interval are indicated within parentheses

PARAMETER	BEFORE HBO <sub>2</sub> T ABS. VALUES MEDIAN 95 % CI	1 H AFTER HBO <sub>2</sub> T % OF BASELINE MEDIAN 95% CI	2 H AFTER HBO <sub>2</sub> T % OF BASELINE MEDIAN 95% CI	P-VALUE
PH	7.42 (7.38 - 7.45)	99 (98.8 - 100.0)	99 (99.2 - 100.2)	0.0484
PACO <sub>2</sub> (MMHG)	37.7 (34.3 - 39.4)	113.7 (100.8 - 123.9)	105.6 (96.3 - 112.6)	0.1834
PAO <sub>2</sub> (MMHG)	98.2 (84.9 - 115.4)	77.04 (71.4 - 94.5)	85.02 (76.3 - 106.4)	0.0022
P(A-A)O <sub>2</sub> (MMHG)	125.0 (76.3 - 203.1)	116.0 (102.1 - 137.9)	108.2 (91.8 - 123.3)	0.0036

$N = 13$ . ANOVA. BASELINE VALUES ARE GIVEN IN ABSOLUTE NUMBERS AND CORRESPOND TO 100%. FURTHER VALUES INDICATE THE PERCENTAGE OF CHANGE FROM BASELINE.

PACO<sub>2</sub>: ARTERIAL CARBON DIOXIDE TENSION. PAO<sub>2</sub>: ARTERIAL OXYGEN TENSION. P(A-A)O<sub>2</sub>: ALVEOLOARTERIAL OXYGEN TENSION DIFFERENCE ( FORMULA:  $P(A-A)O_2 = FIO_2 \times (\text{AMBIENT PRESSURE} - 47) - PAO_2 - PACO_2 / 0.8$ ).

Base excess, MAP, HR and body temperature did not change significantly during the measuring period, median arterial oxygen tension decreased by 23% (1h) and by 15% (2h) of baseline after hbo<sub>2</sub> therapy. The dosage of norepinephrine had to be briefly increased in one patient due to a drop of blood pressure by more than 20% from baseline. in our collective, no subject required further therapeutic interventions.

**Conclusions:** HBO<sub>2</sub> is likely to modify the behaviour of the pulmonary microcirculation (3). in the clinical setting, arterial oxygen tension decreases and alveoloarterial oxygen tension difference increases in the very early period after exposure to hbo<sub>2</sub>. these data suggest a development of a ventilation-perfusion inequality which seems to be reversible as a trend towards baseline occurs within 2 hours. the duration of this effect and the underlying causes

(atelectasis formation? inhibition of the hypoxic pulmonary vasoconstriction?) have to be elucidated by further investigations.

#### **A14 EXTRACELLULAR SUPEROXIDE DISMUTASE CONTRIBUTES TO INCREASED CEREBRAL BLOOD FLOW AFTER HYPERBARIC OXYGEN.** I.T. Demchenko<sup>1</sup>, T.D. Oury<sup>2</sup>, J.D. Crapo<sup>3</sup>, C.A. Piantadosi<sup>4</sup>

Departments of <sup>1</sup>Anesthesiology and <sup>4</sup>Medicine, Duke University Medical Center, Durham NC 27710, Department of <sup>2</sup>Pathology, University of Pittsburgh Medical Center, Pittsburgh, PA 15261, Department of <sup>3</sup>Medicine, National Jewish Medical Center, Denver, CO 80206

**Background:** Extreme hyperoxia causes a sustained increase in the formation of superoxide anion ( $O_2^-$ ) that appears to contribute to oxygen convulsions. The first line of enzymatic defense against excess  $O_2^-$  is the superoxide dismutases (SOD). Two isozymes, SOD1 and SOD2, have been shown to be important in scavenging  $O_2^-$  produced in the intracellular space and have been linked to CNS  $O_2$  toxicity. The third isozyme, extracellular SOD3, is found only in the extracellular space in highest concentration between endothelium and smooth muscle. This suggests SOD3 may be critical in maintaining low extracellular superoxide concentration at this interface and thus prevent  $O_2^-$  mediated inactivation of smooth muscle relaxation by NO. Transgenic mice over expressing (+/+) or lacking (-/-) the SOD3 provide novel opportunities to isolate and examine the role of SOD3 in CNS  $O_2$  toxicity.

**Methods:** Anesthetized and artificially ventilated C57BL/6 wild type and SOD3+/+ or SOD3-/- mice were exposed to hyperbaric oxygen (HBO<sub>2</sub>) at 5 ATA for 60 min. Cerebral blood flow (CBF) and PO<sub>2</sub> in the right striatum were measured. NO metabolites (NOx), as indicator of brain NO bioavailability, was measured in the left striatum by microdialysis and analyzed by chemiluminescence. Blood pressure, EEG and body temperature were monitored. Blood gases were controlled before and rechecked after HBO<sub>2</sub> exposures.

**Results:** Steady state CBF, brain PO<sub>2</sub> values and blood pressure were the same in wild type and genetic mutants. During HBO<sub>2</sub> exposures wild type mice displayed initial decreases in CBF and NOx followed by gradual return to control values and then an increases by 26-128% above pre-exposure levels. Brain PO<sub>2</sub> values were 348-585 mmHg during vasoconstriction phase and rose by 2-3 fold during cerebral hyperemia. EEG spikes, as a sign of CNS  $O_2$  toxicity, were observed always after increases in CBF. Extreme hyperoxia also decreased CBF and NOx in SOD-/- mice and the responses were more pronounced and prolonged than in wild type animals. SOD3-/- mice showed slower increases in PO<sub>2</sub> and greater delay in onset of EEG discharges during HBO<sub>2</sub> exposure. However, unlike wild type and SOD-/- mice, HBO<sub>2</sub> increased CBF and NOx in SOD3+/+ mice. PO<sub>2</sub> in the striatum rose much faster and EEG spikes were observed earlier in SOD3+/+ mice compared to wild type.

**Conclusion:** SOD3 appears to be involved in sensitivity to CNS  $O_2$  toxicity by inhibiting  $O_2^-$  mediated inactivation of NO and altering cerebral blood flow.

#### **A15 HUMAN BEHAVIOURAL THERMOREGULATION DURING HYPOXIC EXPOSURE.** Petra Golja and Igor B. Mekjavic. Department of Automation, Biocybernetics and Robotics, Institute Jozef Stefan, Ljubljana, Slovenia & Institute of Biomedical and Biomolecular Sciences, University of Portsmouth, United Kingdom.

**Background:** Prolonged exposure to hypoxia initiates functional changes in neural activity. The present study tested the hypothesis that such changes in central neural activity may result in impaired perception of temperature and subsequently alter behavioural temperature regulation. In high heat loss ambients, appropriate behavioural thermoregulatory responses may prevent deleterious cooling of deep body temperature. Any hypoxia-induced impairment of behavioural thermoregulatory responses may therefore predispose individuals to enhanced core temperature cooling and ultimately hypothermia.

**Materials and Methods:** Eight healthy male volunteers participated in two separate trials. In each trial, following a 15 minute rest period they were immersed to the neck in 28°C water for 90 minutes, and inspired either room air (Air) or a hypoxic gas mixture (Hypoxia: 11.5 %O<sub>2</sub>, 88.5 % N<sub>2</sub>). During the immersion, the subjects' right hand was placed in a separate bath perfused by water. At regular intervals the subjects were instructed to adjust the temperature of the water perfusing the hand bath to a comfortable temperature, by manipulating a valve. The adjusted temperature of the water perfusing the hand bath was termed the 'preferred hand temperature' (Tpref). At minute intervals, esophageal (Tes), rectal (Tre) and mean skin (Tsk) temperatures, and ventilation (V<sub>I</sub>) were recorded. The observed responses between the two conditions were compared with a one-way ANOVA.

**Results:** Tsk was maintained at 28.1°C throughout the immersion in both conditions. During the immersion, Tes decreased from 36.7°C in both the Air and Hypoxia conditions to 35.7 and 35.9°C, respectively. Similarly, Tre decreased from 36.9°C to 36.2°C in both the Air and Hypoxia conditions. Concomitant with the decrease in deep body temperature was an increase in Tpref. There was a large inter-subject variability in the initial and final Tpref. The average increase in Tpref during the immersion was 2.3°C in the Air, and 2.6°C in the Hypoxia condition; the difference was not statistically significant. With the exception of V<sub>I</sub>, which was significantly higher in the Hypoxia

condition, there were no significant differences in any of the recorded variables between the Air and Hypoxia conditions.

**Conclusions:** Assuming that the selection of preferred hand temperature represents a behavioural thermoregulatory response, the present results suggest that normobaric hypoxia induced by inhalation of a gas mixture containing 11.5% O<sub>2</sub> does affect behavioural thermoregulation.

**Acknowledgements:** This work was supported by the Ministry of Education, Science and Sport (Republic of Slovenia).

#### **A16 HYPERBARIC OXYGENATION AND SPONTANEOUS ELECTRICAL ACTIVITY OF MONKEY BRAIN.** Nikola Dekleva, Beograd, Yugoslavia, Serbia

The results of these investigations suggest that differential HBO effects on the studies of subcortical and cortical structures might exist, as well as sensibility of the primary cortex to HBO action.

The present experiments have been undertaken to analyse the spontaneous electrical activity in the brain of monkeys subjected to the effects of hyperbaric oxygen (HBO). Experiments were carried out in monkeys (*Macaca Cynomolque*) of both sexes weighting 2 - 3 kg.

The electrodes were inserted under Nembutal anaesthesia, into hypothalamus, hippo-campus, motor, extrafriaire and striate cortex of one or both sides of the brain. Spontaneous electrical activities of the studied structures were recorded before, during and after the exposure of the monkeys to normobaric and hyperbaric oxygenation in the HBO chamber.

Primary EEG alterations were noticed in the posterior hypothalamus and then in hippocampus. In these structures the sequences were slow, the activities consisted of irregular waves showing variable amplitudes.

They were followed by progredient reduction in the amplitude of the electrical activity in the striate cortex, whose curve temporarily become more and more disorganised, as the time of monkeys exposure to 3 ATA oxygen grew. Essential changes of spontaneous electrical activity in motor and extrastriate cortex were not noticed, except for rare transitory isolated graphoelements of irritative character.

The results of these investigations suggest that there might be differential effects of HBO on the investigated subcortical and cortical structures, as well as selective sensibility of primary visual brain cortex of primates to the effect of hyperbaric oxygenation.

#### **A17 MONITORING OF BRAIN ENERGETIC METABOLISM UNDER NORMOBARIC NARCOSIS, COMPARISON BETWEEN UNIQUE AND MULTIPLE EXPOSURES: A MULTI-PROBE MICRODIALYSIS STUDY IN FREE-MOVING RAT.** Aurélie Moulins, Franck Escalettes, Joël Reybaud, Jean-Louis Méliet, Jean-Jacques Risso. Department of Neurochemistry of Extreme Environments, I.M.N.S.S.A., B.P. 610, F83800, Toulon Naval, France

In order to study the mechanisms involved in narcosis induction, neurochemical studies have been performed, using microdialysis technique. The results led to the evidence that extracellular dopamine decreases during both hyperbaric Nitrox and normobaric nitrous oxide exposure. Recent studies pointed out the involvement of glucose in the modulation of the dopaminergic neuronal activity in rat striatum.

The aim of the present study is to monitor brain energetic metabolism by microdialysis under nitrous oxide which is considered as a good model of inert gas narcosis inductor.

Extracellular glucose, lactate and pyruvate concentrations were followed after a double microdialysis probes stereotaxic implantation (length 4 mm, width 0,5 mm), in left and right striatum of male Spage Dawley rats. The first measurement was performed on one striatal side, on Monday, during a 2 hours exposure to a normoxic 60 % nitrous oxide mixture. The second measurement was performed on the other side, on Friday, after 8 bi-daily successive N<sub>2</sub>O exposures of 120 min each. In both situations, measurements of baselines as well as recuperation phases were also recorded. Animals were placed in a free-moving device specially designed for our 180 L pressure chamber used as a normobaric nitrous oxide respiratory box.

Analysis occurred on a specific CMA 600 microdialysis spectroscopic analyzer, after double enzymatic reactions. Results show, after the first exposure, a decrease in striatal glucose level, immediately visible in the dialysates following the submission of the animals to the narcotic breathing mixture. The repetitive narcotic exposure led to an amplification of the variation of glucose concentration. On the contrary, the lactate and the pyruvate levels increase just after the injection of the nitrous oxide in the chamber. The repetitive exposure emphasises the amplification phenomenon, previously observed with glucose. The lactate/pyruvate rate, which is a index of the extent of glycolysis and anaerobic metabolism in the tissue, increased dramatically as well. Moreover, glycerol which is thought to be an indicator of membrane disintegration in the brain, was also analyzed by the same method while amino acids, especially excitotoxic glutamate, were measured by HPLC with fluorimetric detection after o-

phthalaldehyde derivatization. Dopamine and its metabolites (DOPAC, HVA) were monitored by the same probe and analyzed by HPLC coupled with electrochemical detection.

Considering the diminution of the brain metabolism, correlated by the animals behavior under such a percentage of N<sub>2</sub>O, the decrease in cerebral glucose could be linked to a decrease of a central energetic input. The alteration of the striatal glucose concentration is correlated to the decrease of the extracellular dopamine level induced by all narcotic conditions. Although nitrous oxide can be considered, when used under hyperbaric condition, as an anesthetic, results obtained in our laboratory on pentobarbital effects highlight an increase in brain glucose as well as a decrease in lactate and pyruvate concentrations.

#### **A18 MECHANISMS OF HYPERBARIC OXYGEN (HBO) - INDUCED ADAPTIVE RESPONSE.**

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Hyperbaric oxygen (HBO) treatment is a well suited model for studying genetic consequences of oxidative stress. Our previous in vivo studies with human volunteers have shown that one consequence of HBO exposure is the induction of an adaptive response, which protects cells from the induction of further oxidative DNA damage. We can now show that an adaptive protection is also induced, when isolated human lymphocytes are exposed to HBO, which offers the possibility to perform investigations on mechanisms responsible for this protection. Analysis of the time course of the adaptive response indicated that human lymphocytes are already adapted 4h after an initial HBO and that this protection lasts for at least 24h. In parallel to this effect, an induction of the stress protein heme oxygenase-1 (HO-1) was found in the same cells. Comet assay experiments with the selective HO-1 inhibitor tin-mesoporphyrin (SnMP) revealed a complete abrogation of the HBO-induced adaptive response, indicating a close relationship between the induction of HO-1 and the occurrence of the adaptive protection in human lymphocytes. In contrast, a comparable protection was not found in V79 cells, suggesting a cell-specific difference in expressing the adaptive response. In order to gain further insight in the role of HO-1 in cellular protection, V79 cells were transiently transfected with a full-length human HO-1 cDNA construct (V79-HO1 cells) and exposed to HBO. Investigations with the comet assay and the micronucleus test show that overexpression of HO-1 resulted in a significant protection against genotoxic effects induced by a single HBO. Furthermore, V79-HO1 cells were able to express an adaptive protection following a repeated HBO exposure. However, the effect was less pronounced compared to that observed in human lymphocytes. These results indicate that other mechanisms such as induction of ferritin may also contribute to the cellular phenotype of the adaptive protection.

#### **A19 DOCUMENTATION OF THE EFFECT OF OXYGENATION FOR CEREBRAL PALSY AND THE BRAIN INJURED CHILD – SEQUENTIAL SPECT IMAGING.**

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**Purpose:** This manuscript will document the effect of hyperbaric oxygenation hyperbaric oxygenation on cerebral palsy and brain injured children using single photon emission computerized tomography (SPECT) scanning.

**Background:** Over one-thousand patients, worldwide, treated with hyperbaric oxygenation show compelling results. A recent Canadian study suggested positive effects may also be obtained from compressed air. Clinical reports remain mostly anecdotal; rigorous scientific documentation is required to make this become an acceptable, reimbursable therapy.

**Materials and methods:** 275 cerebral palsy or other brain insult patients, six weeks to fourteen years, were treated with 20 to 400 exposures of hyperbaric oxygenation at 1.1 to 1.75 ATA, one hour – twice daily. Seizure disorder was treated at 1.1 to 1.25 ATA. 250 patients had sequential SPECT scanning.

**Results:** Less spasticity and scissoring, removal of most G-tubes and trachs, improvement in fine and gross motor control, cognitive and visual improvement. SPECT scans showed positive changes in flow and metabolism in about 87 percent of the patients, with a 90 percent correlation between change in SPECT scan and patient's clinical response. Three representative cases will be presented: A) Anoxic Ischemic Encephalopathy, B) Traumatic Brain Injury, C) Severe Vaccination Reaction. SPECT scanning presentations parallel clinical results.

**Conclusion:** Utilizing each patient as individual control, and evaluating brain blood flow and metabolism with SPECT scanning associated with hyperbaric oxygenation documents the positive clinical effects noted worldwide. Recent data from Russia, Mexico and Cornell University compliments earlier studies showing that the earlier the intervention (i.e. from delivery room to the chamber), the more promising the results.

#### **A20 HYPERBARIC TREATMENT OF NECROTISING SOFT TISSUE INFECTIONS: HOW MANY SESSIONS ARE NECESSARY?**

Joachim Hencke, Ana Jakopin, Carl-Michael Grosse-Wietfeld,



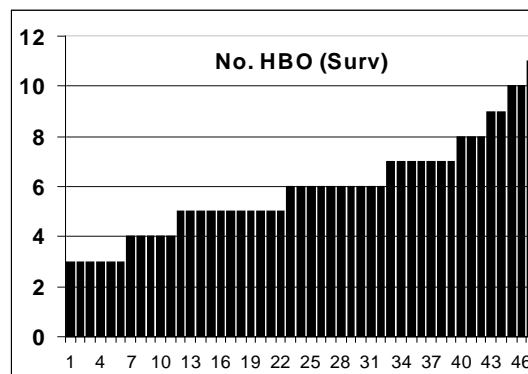
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**Introduction:** Hyperbaric oxygen therapy now for over 40 years has been part of the treatment regime in necrotising soft tissue infections (NSTI). Nevertheless there have been several different recommendations for the total number and frequency of hyperbaric treatments.

**Method:** All patients admitted to our hospital for NSTI received initial surgical debridement and daily wound inspection in the operating theatre, broad spectrum antibiotics and hyperbaric oxygenation of 90 minutes at 300 kPa given two times within the first 24 hours and then once a day. Based on the surgeon's judgement hyperbaric therapy was finished when infection was under control and no further necrosis occurred. This is a retrospective analysis from our patient data base.

**Results:** From January 1999 until March 2000 we treated 63 patients. The overall mortality was 22% (14 patients). 47 of the 49 survivors could be analysed. The total number of treatments given reached from 3 to 11, the mean value was 5,85 (SD 1,97). Maximum oxygen levels measured transcutaneously (PtcO<sub>2</sub>) were 1680 +/- 190 mmHg.

**Conclusion:** The efficiency of hyperbaric oxygen therapy in necrotising soft tissue infections mainly is due to the achievement of sufficient tissue oxygen tensions on the site of infection. In a former study we showed a correlation of mortality and maximum achieved systemic PtcO<sub>2</sub>. Using the treatment protocol presented in this study, infection control can be achieved by very few, in some cases even less than 5 hyperbaric treatments.



**A21 HYPERBARIC OXYGEN IN THE TREATMENT OF THERMAL BURNS – EXPERIENCES WITH CHILDREN AND ELDERLY PEOPLE.** Heiko Renner, Claudia May, Sabine Gabor, Huberta Klemen, Bengkt Hellbom, Udo Anegg, Alfred Maier, Hans Pinter, Freyja-Maria Smolle Jüttner. Division of Thoracic and Hyperbaric Surgery, Dept. of Surgery, University Medical School of Graz, Graz, Austria

**Background:** The use of HBO therapy as an adjuvant to established therapy of burned patients is already accepted. The positive effects of hyperbaric oxygen therapy in treatment of burns are bacterio-static and bactericidal effects on aerobic and anaerobic bacteria, improvement of microcirculation reduction of edema, less inflammatory response and reduction in the size and depth of lesions.

**Subject and methods:** We present the Austrian experience on HBO therapy in the treatment of burns during the last two years. We treated 31 patients, 21 males and 10 females, with an average age of 43,2 years. All patients were treated within the first 24 hours after trauma. The HBO therapy was done after lavage and wound debridement, if necessary. Each hyperbaric session took 60 minutes at 15 m depth. The mean number of sessions per patient was 9,4.

**Results:** 30 of these 31 patients are still alive. We observed a decreased infection-rate (20%), a reduction of surgical procedures per patient (1,75) as well as a decreased length of hospital stay (19d) in comparison to our past data.

**Conclusion:** HBO treatment is a secure additive treatment option for well equipped centers, with reproducible patients benefits, but needs a straight forward management.

**A22 APPLICATION OF THE HBO THERAPY BY CROHN DISEASE WITH FISTULA FORMATION.** Oliver Walid Jacobs<sup>2</sup>; J. Rebhen<sup>1</sup>; M. K. Müller<sup>1</sup>

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An alarming complication of the Crohn Disease (CD) is the formation of fistula lesions. There are no satisfactory and novel treatment modalities for such cases. There are 2 fistula types. The internal type formed a connection between colon, ileum, jejunum, stomach and urinary bladder. The external fistula flows into cutis. The fistula by CD occurs a weight reduction with reinforce diarrhoea with deficiency of minerals and vitamins. This lead to immune debility, which promote the inflammatory processes and abscesses.

Desirable is to break through this circular course. The cell messenger by CD is the tumor-necrose-factor-alfa (TNF- $\alpha$ ). Infliximab (Remicade) was described against TNF- $\alpha$ . Benefit of Infliximab is to inhibit the mucosal inflammation and to suppress the fistula. Disadvantage is the building of own antibodies HEMEC against TNF- $\alpha$ , which is making Infliximab inefficient. Additionally there are the high costs and side effects.

There are patients, who are not reacting on an immune suppression / glucocorticoids, even with rezidive fistula and in case of repeated surgical interventions.

The inflamed intestinal mucosa in experimental induced colitis and in CD show a positive reaction, when treated by HBO. Haemorrhagic and inflamed colon mucosa with early cell damage in ischemia was significantly reduced. The anti-inflammatory effects of HBO-therapy has been well documented in several studies.

Cytokines (IL1 and TNF- $\alpha$ ) are elevated in patients with perianal CD. Treatment by HBO decreases significantly these cytokines. HBO reduces the steroid agents.

The aim is to tender such preloaded patients a therapy with less side effects, with a short duration, an effective one with less expense. Such a demand could be justified by the HBO-therapy.

Several studies demonstrated by HBO the suppression of IL1, IL6 and TNF- $\alpha$ . Further more an important effect of the HBO is the angiogenesis with an increase of fibroblast production. These effects suppress the fistula building.

**Case report:** A 31 years old (1969) white female, CD diagnosis was 1992, rezidive perianal secreted fistula with multiple operations since 1997. Before the HBO we did a contrast fistula presentation. Medication was Mesalazin, Steroids and Azathiopren. Before starting the HBO-therapy we added Metronidazol to her medications. For the HBO-therapy we used a problem wound therapy schema (TS 240-90). The HBO-therapy duration was 14 days. Already after 5 days, she reported a reduction of pain symptoms and fistula secretion. After completing the HBO course were the fistula totally closed, the patient was painless and fully satisfy. Oxygen toxicity did not occur.

Thereafter we administered her once infusion of Infliximab to forward the inhibition of TNF- $\alpha$ . After a follow up periods of ¼ year, ½ year and 1 year demonstrated a stabile state with no recurrence and confirmed the efficiency of HBO therapy.

By utilization of high dosage combinations of drugs and such as Infliximab was the limited success less than 3 months till the reappearance of the fistula.

Furthermore we treated another 4 patients with similar symptoms of CD with fistula lesions and 2 of them had an acute attack. All of them become an improvement.

We recommend further studies to confirm statically the positive effect of HBO-therapy on CD- patients even with fistula lesions. HBO is a useful therapy form of CD with perianal lesions who are unresponsive to immunosuppressive or to metronidazole therapy or who suffers from the side effects under other medication/drugs.

### A23 HYPERBARIC OXYGEN THERAPY ON 17<sup>TH</sup> OF AUGUST EARTHQUAKE IN MARMARA.

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**Introduction:** Crush syndrome or traumatic rhabdomyolysis is the manifestation of muscle injury caused by prolonged limb compression sustained in crush injury. Hyperbaric oxygen therapy (HBO<sub>2</sub>) has an adjunctive role in the treatment of crush injury and other acute traumatic peripheral ischemias. It is reported that HBO<sub>2</sub> decreases edema with vasoconstriction, prevent from reperfusion injury and contributes wound healing. In this study, the patients whom developed compartment syndrome after crush injury followed Marmara earthquake on August 17 in 1999 and treated with decompressive fasciotomy and hyperbaric oxygen therapy (HBO<sub>2</sub>) was evaluated retrospectively.

**Materials and Methods:** After examination and prior treatment on admission, the patients who had sustained injuries by being compressed under collapsing buildings were identified as crush syndrome. Demographic data and anatomical injury site was recorded. Evaluations on admission include arterial blood gas analysis, whole blood count, serum concentration of potassium, calcium phosphate, creatine kinase (CK), urea, glutamine oxalacetic transaminase (SGOT), glutamic pyruvic transaminase (SGPT), lactate dehydrogenase (LDH), and radiographic and neurologic examinations. Performed medical and surgical treatments and related complications were also recorded. Records related with HBO therapy included the total number and duration of HBO<sub>2</sub> sessions.

**Results:** Six hundred and thirty patients were admitted to our hospital after the North Anatolian Earthquake. One hundred and forty six patients were underwent surgical procedures. Fasciotomy was performed to 92 patients. Fifty-two of them (23 patients in our hospital and 29 patients from various hospitals) were treated with HBO<sub>2</sub> therapy in postoperative period. These patients consisted of 32 male and 20 female.

On admission, in laboratory measurements; mean values for white cell count was 12350/mm<sup>3</sup>, for CPK was 75.61 U/L, for SGOT was 451.56 U/L, for SGPT was 1125.34 U/L, and for LDH was 2325.51 U/L. 12 patients had hyperpotassemia, 9 had hypocalcaemia, and 5 had hypophosphatemia. In 7 patients acute tubular necrosis was diagnosed on ultrasonographic examination. Myoglobinemia and myoglobinuria was detected in 10 patients. Debridement was performed to necrotising wounds if necessary. Wound cultures were collected from patients. Acinetobacter was 50,9%, S. aureus was 26,4% and Pseudomonas was 22,4% was grown from wound cultures. Specific antibiotic treatment was begun and then daily wounds cleaning were maintained. Mostly acinetobacter was isolated which were sensitive to imipenem and meropenem.

HBO<sub>2</sub> therapy have been started to 31 crushed patients who were saved from ruins in first 36 hours but their therapy couldn't be continued because of the our chambers capacity was insufficient to get many victims. To make enough room to the military personnel, civilian patients have been transported to the nearest hospitals. HBO<sub>2</sub> therapy was performed to 21 patients 3 to 10<sup>th</sup> days after disaster and all of them were completed the therapy, so HBO<sub>2</sub> were performed to totally 52 patients. Twenty-nine of them had fasciotomy of lower extremity, 14 had upper extremity fasciotomy, and 9 had upper and lower extremity fasciotomy. Hemodialysis was performed to 7 patients

because of acute renal failure and 3 of them were dead during the therapy due to acute respiratory distress syndrome and sepsis.

HBO<sub>2</sub> was performed twice a day at 2,5 ATA with 2 hours sessions. HBO<sub>2</sub> therapy was totally provided to patients in 946 sessions varying between 3 to 70 sessions.

**Conclusion:** HBO<sub>2</sub> therapy is advocated for the treatment of severe trauma of the limbs in association with surgery because of its effects on peripheral oxygen transport, muscular ischemic necrosis, compartment syndrome, and infection prevention. Its therapeutic efficacy and limitations are yet incompletely understood and appreciated, and continuing research in this area is warranted.

#### **A24 INFLUENCE OF SYMPATHIC INNERVATION ON THE HEART RATE DURING SCUBA DIVING. PRELIMINARY RESULTS.** Stefaan Deneweth <sup>1,2</sup>, Herman Vanbogaert <sup>3</sup>, Michel Lambrechts <sup>3</sup>, Alessandro Marroni<sup>3</sup>, Ramiro Cali Corleo <sup>3</sup>, Costantino Balestra <sup>1,2,4</sup>, Peter Germonpré <sup>2,5</sup>

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**Introduction:** Due to the popularity of diving we are confronted with a greater population of divers without knowing what their limitations are. Because many divers are older and physically not fit, an investigation of the physiological cardiocirculatory reactions during scuba diving is warranted. In this, we have to consider the real circumstances of this sport and therefore we may not limit ourselves to “dry” hyperbaric chamber dives. Previous studies to analyse the heart rate control during diving have mostly been performed in dry chambers. Here we show preliminary results of heart rate variability during “wet pot” standardized dives.

**Methods:** Eighteen healthy divers without any known cardiac or pulmonary disease (15 males and 3 females; age from 27 to 50 years - mean 36) performed a standardized dive while connected to a Holter monitoring device (Unolter, Novacor) sealed into the dry suit. Blood samples were taken before and after the dive to evaluate serum glycemia and hematocrite; other parameters measured were arterial blood pressure, body impedance, Borg subjective fatigue scale, Flicker Frequency Test (objective concentration evaluation), D2 Visual Attention Test, Cattell (anxiety test) and MMPI (personality test). The heart rate data were stored and were analysed afterwards for quantification of the sympatho-vagal balance activity. We looked at the power related to the low frequencies (LF) and high frequencies (HF) components expressed in absolute values as well as in their ratio (LF/HF, so-called autonomic index) and the related peak frequencies. The result of this computations were compared with the psychological screening for stress and the blood samples. We divided the recording period into 3 parts: just before the dive, during the dive and the third part included the preparation time.

**Preliminary results:** The spectral analysis of the heart rate variability showed marked differences between the recorded periods and demonstrated surprisingly that the dive period seems not to be the most stressing one. There did not seem to be a marked bradycardia during the dive, which was an unexpected result. The fatigue analysis did not show a difference before and after the dive. There was no significant change in hematocrit values, nor in the body impedance and glycemia levels.

**Conclusion:** This method could reveal, in a controlled environment, persons who could be more susceptible to stress related inadequate reactions that represent a risk factor for diving-related incidents. The preliminary results show that there is a certain stress factor, but surprisingly mainly not during the dive.

#### **A25 ENHANCED LYMPHATIC CAPTATION OF PROTEINS DURING NORMOBARIC OXYGEN BREATHING. A LYMPHOSCINTIGRAPHY STUDY.** Thyl Snoeck <sup>1,2,3\*</sup>, Mikel Ezquer <sup>1,2,3\*</sup>, O. Leduc <sup>2</sup>, A. Leduc <sup>2</sup>, F. Willeput <sup>2</sup>, Costantino Balestra <sup>1,2,3</sup>, Alessandro Marroni <sup>1</sup>, Ramiro Cali Corleo <sup>1</sup>, Peter Germonpré <sup>1,4</sup>

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**Introduction:** The use of normobaric oxygen (NBO) as a first aid tool for decompression sickness (DCS) has been advocated for a long time. Several beneficial effects of NBO have been demonstrated, one of which could be the faster elimination of tissular nitrogen bubbles. During DCS occurrence, a cascade of intravascular reactions has been demonstrated. These inflammatory reactions occur within minutes of the onset of DCS, and involve the precipitation of proteins on the gas-bubble interface, causing a stabilisation of the intravascular and intratissular bubbles. Little is known about the elimination of these protein-coated bubbles. As interstitial proteins are evacuated by the lymphatic circulation, we wanted to investigate if NBO enhances the lymphatic protein captation and elimination.

**Methods:** Ten healthy subjects received an injection of 0.2 ml of Tc99-marked Human Albumin, diluted with 2.3ml of S.S.P.P., into the first interdigital space. This injection produced a moderate subcutaneous edema. While remaining recumbent, each subject underwent a lymphoscintigraphy. On a separate day, the procedure was repeated, but the subject breathed NBO from immediately after the injection. The dynamics of the isotopic activity at the axillary ganglia was recorded as witness of the speed and quantity of the lymphatic protein drainage. In parallel, TcPO<sub>2</sub> in the edema region was constantly monitored.

**Results:** In 6 subjects out of 10, NBO produced a marked increase of the isotopic activity at the axillary level after 1 hour, starting 30 minutes after the (see figure). In these subjects, PTcO<sub>2</sub> levels at the site of edema showed a marked increase during the first 10 minutes, followed by a return to baseline.

**Conclusion:** Normobaric oxygen breathing enhanced protein captation by the lymphatic system in 6 out of 10 subjects. This may be related to peripheral vasoconstriction, but more likely to an independent enhancement of the lymphatic flow. Investigations are planned to further clarify this observation, using different sizes of proteins to explore possible macrophage activity interference.

**A26 THE USE OF A “PROPORTIONAL M-VALUE REDUCTION CONCEPT” (PMRC) CHANGING THE ASCENT PROFILE WITH THE INTRODUCTION OF EXTRA DEEP STOPS REDUCES THE PRODUCTION OF CIRCULATING VENOUS GAS EMBOLI AFTER COMPRESSED AIR DIVING. DSL SPECIAL PROJECT 01/2001.** A. Marroni <sup>1-2-6</sup>, R. Cali Corleo <sup>1 2 6</sup>, C. Balestra <sup>1 3 6</sup>, P. Longobardi <sup>4-6</sup>, P. Germonpre <sup>1-2-8</sup>, E. Voellm <sup>5-6</sup>, M. Pieri <sup>1-6</sup>, R. Pepoli <sup>6-7</sup>

1) Dan Europe Foundation, Research Division . 2) Division of Baromedicine, University of Malta Medical School. 3) Haute Ecole Paul Henry Spaak, Human Biology Dept. Bruxelles, Belgium. 4) Centro Iperbarico, Ravenna, Italy. 5) Dynatron Ag and Uwatec Ag, Switzerland. 6) Dan-Uwatec Diving Safety Laboratory. 7) Ravenna Sub, Ravenna, Italy. 8) Center for Hyperbaric Oxygen Therapy, Military Hospital Bruxelles.

The DAN Europe SAFE DIVE project showed that post dive High Bubble Grades are directly related to Fast to Medium Half Time Tissues, Computed Nitrogen Venous Partial Pressure (P<sub>venN<sub>2</sub></sub>) higher than 1100 mbar and Leading Tissue Nitrogen Partial Pressure (PltN<sub>2</sub>) higher than 80% of the allowed M Value. A specific research project was started to identifying bubble-safe dive profiles based on the above findings. Three square dive profiles were selected: a single dive to 20 m for 60 min, a single dive to 40 m for 10 min, a series of three repetitive dives to 30 m for 16 min with 75 min Surface Interval. Doppler Recording was performed every 15 minutes post dive. Grading was according to a variant of the Spencer method: Low Bubble Grade – occasional bubble detection; High Bubble Grade – frequent to continuous bubble detection. The dives were made according to the original ZH-L8 ADT model (Dive Series A) and repeated a first time with a new algorithm, modified in order to keep the PltN<sub>2</sub> within the above indicated limits (Dive Series B). 184 Doppler Recordings were made after 10 test chamber dives (90 man-dives) on 9 volunteers. After Dive Series A, 5 of the 9 divers presented High Bubble Grades for extended time and 1 Diver suffered a mild episode of Skin Bend. Because of the limited sample, this figure should not be considered as a DCS risk index higher than indicated by current epidemiological research, however we wanted to design dive profiles which could be safe even for “high bubble risk” divers. After Dive Series B only occasional Low Bubble Grades were registered. However the resulting ascent profiles were not considered “diveable” in the field. A third profile (Dive Series C) was calculated, based on a different concept, introducing a gradual reduction of the Leading Tissue M-Value, inversely proportional to the Tissue HT (*Proportional M-Value Reduction Concept – PMRC*). The fast to medium tissues M-Values were reduced by decreasing reduction factors, to reach correction factor 1 for the 80 minutes HT tissues. The set of experimental dives was repeated with the same group of 9 volunteers, plus an additional 3 new divers, known as “bubblers” from previously monitored dives (Dive Series C). 96 Doppler Recordings were made during 5 test dives (60 man-dives) on 12 volunteers. After Dive Series C, 8 of the 12 Divers produced only occasional Low Bubble Grade signals, however the 20 meter dive produced constant LBG readings in all the divers, over the entire 90 minutes post-dive monitoring period and this was considered as an index that the slow compartments M-Values were still too high. The PMR Concept was then extended to the 80 minutes HT tissues, reaching correction factor 1 for the 160 minutes HT. All the ascent profiles were re-calculated accordingly and were tested during a fourth series of chamber dives (Dive Series D) with 10 of the volunteers from the previous dives plus two female divers who had shown HBG Doppler readings during previous field exposures to “normal” dive profiles and had suffered multiple episodes of skin DCS. 108 Doppler Recordings were made during these last 5 test dives on 12 volunteers (60 man-dives). 6 of the 12 divers produced only minimal LBG Doppler readings, which were occasional in nature and did not show any clear pattern as to the post-dive time interval.

**Conclusions:** The modification of the ZH-L8 ADT algorithm by the introduction of a *Proportional M-Value Reduction Concept (PMRC)* to the fast and medium-slow HT Tissue compartments, without altering the original speed of ascent between any planned stop and resulting in a modified ascent slope and in the introduction of extra

deep stops during the ascent eliminated the occurrence of significant post-dive Doppler detectable Venous Gas Emboli in a sample of 14 volunteers, during 20 dry test dives and 210 man-dives monitored with 388 Precordial Doppler Readings. The PMRC model has been entered into a new dive computer prototype which is currently being tested during multiple unrestricted recreational dives within the DAN UWATEC Diving Safety Laboratory program.

**A27 THE SPEED OF ASCENT DILEMMA: “INSTANT SPEED OF ASCENT” OR “TIME TO SURFACE” – WHICH ONE REALLY MATTERS? INSTANT SPEED OF ASCENT VS. DELTA-P IN THE LEADING TISSUE AND POST-DIVE DOPPLER BUBBLE PRODUCTION. DSL SPECIAL PROJECT 02/2001.** A. Marroni <sup>1 2-6</sup>, R. Cali Corleo <sup>1 2-6</sup>, C. Balestra <sup>1 3-6</sup>, P. Longobardi <sup>4-6</sup>, P. Germonpre <sup>1-2-8</sup>e. Voellm <sup>5-6</sup>, M. Pieri <sup>1-6</sup>, R. Pepoli <sup>6-7</sup>

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Fast ascent from any dive is universally considered as dangerous, even if anecdotal reports from the field offer contradictory interpretations. The DAN-UWATEC Diving Safety Laboratory findings indicated that the introduction of extra deep stops during the ascent, without changing the actual speed of ascent between the stops, resulted in the elimination of any significant post-dive precordial Doppler bubble detection. To better understand the role of the actual “speed” of ascent, which we defined as “instant” speed, we developed a method to calculate this “instant” speed from the depth/time profile recorded with the DAN-UWATEC Diving Safety Laboratory “Black Boxes” (specially modified dive computers) every 20 seconds during the entire dive. The results showed no apparent relation between “instant” speed and post-dive Doppler bubble detection. Apparently contradictory results were actually observed during several test chamber dives (series of 3 repetitive dives to 30 meters and single dives to 40 meters) with “instant” speeds of up to 24 meters per minute and no bubble signal detection in the experimental dives, computed according to the new Proportional M-Value Reduction Concept and introducing extra deep stops during the decompression phase, versus significant high bubble grade signals after the control dives, made according to the standard ZH-L8 ADT algorithm, with “instant” speeds of ascent never faster than 10 meters per minute. These observations seem to indicate that the Delta-P imposed on the leading tissue, irrespective of the “instant” speed of ascent, is the critical factor for precordially detectable bubble production in this series of experimental dives. Further analysis on real unrestricted recreational dives is planned within the DAN-UWATEC Diving Safety Laboratory program.

**A28 INCIDENCE OF ASYMPTOMATIC CIRCULATING VENOUS GAS EMBOLI IN UNRESTRICTED, UNEVENTFUL RECREATIONAL DIVING. SKIN COOLING APPEARS TO BE RELATED TO POST-DIVE DOPPLER DETECTABLE BUBBLE PRODUCTION. AN UNEXPECTED FINDING. DSL SPECIAL PROJECT 03-2001.** A. Marroni <sup>1 2-6</sup>, R. Cali Corleo <sup>1 2-6</sup>, C. Balestra <sup>1 3-6</sup>, P. Longobardi <sup>4-6</sup>, P. Germonpre <sup>1-2-8</sup>, E. Voellm <sup>5-6</sup>, M. Pieri <sup>1-6</sup>, R. Pepoli <sup>6-7</sup>

1) DAN Europe Foundation, Research Division 2) Division of Baromedicine, University of Malta Medical School 3).Haute Ecole Paul Henry Spaak, Human Biology Dept. Bruxelles, Belgium. 4) Centro Iperbarico, Ravenna, Italy. 5) Dynatron AG and Uwatec AG, Switzerland. 6) DAN-UWATEC Diving Safety Laboratory 7) Ravenna Sub, Ravenna, Italy. 8) Center for Hyperbaric Oxygen Therapy, Military Hospital Bruxelles.

Between 1995 and 1999, DAN Europe collected 2105 fully monitored unrestricted recreational dives, during 75. Research Trips organized by 106 Research Field Operators and involving 575 volunteer Research Divers. All the dives were Doppler monitored at fixed intervals post-dive. The distribution by depth of the monitored dives showed that the relative majority.(33,15%) of the dives were made in the 20 to 30 meters depth range. The Overall depth range varied from 5 to 65 meters. Doppler monitored bubbles were detected in.37,4% of all the monitored dives. 25,4% of the dives produced Low Bubble Grade recordings only, while 12% produced High Bubble Grades and 2,4% produced Very High Bubble Grades. Repetitive dives showed a reversed incidence of post dive VGE, as only 15% of the repetitive dives were bubble-free. LBG were detected in 18% of the repetitive dives and HBG were recorded in 67% of the repetitive dives.. A further analysis of the dives has shown an unexpected finding, when considering the variable of the estimated skin cooling, calculated by the dive profile recorder as a function of time and water temperature. HBG doppler signals appeared to be directly related to estimated skin cooling, with higher signal for skin temperatures of.27 °C and low or absent signal for skin temperatures of 29 °C. The post-dive time to peak Doppler bubble detection varied between 30 and 45 minutes, with a trend to earlier peak-time for higher bubble grades. Although these data refer to estimated and not measured skin cooling, they suggest a more

important than previously considered role of the skin in the production and release of post-dive circulating venous gas emboli and a concomitant role of temperature and heat-loss.

It is hypothesized that diving in relatively cold water produces progressive skin cooling and vasoconstriction that may, by the end of the dive and during the post-dive period, produce a temporary trapping of excess inert gas in the skin compartment, producing significant local supersaturation and gas phase formation during the ascent and the immediate post-dive period. The subsequent post-dive skin re-warming, with removal of the vasoconstriction, may produce a significant release of venous bubbles and “showers” of bubbles invading the pulmonary circulation. This resembles the well known anecdotal post-dive “hot shower” effect. The role of these variables will be investigated during experimental as well as unrestricted field dives, including Doppler Monitoring and continuous skin and body temperature monitoring, within the DAN UWATEC Diving Safety Laboratory program.

#### **A29 CEREBRAL LACUNARY SPOTS IN RELATION TO PATENCY OF FORAMEN OVALE, AN MRI INVESTIGATION.** Brigitte Farkas<sup>1</sup>, Emilie Duboc<sup>1,3</sup>, C.Pietrons<sup>4</sup>, Jacques Widelec<sup>4</sup>, Frédéric Vanderschueren<sup>1</sup>, Costantino Balestra<sup>1,2,3</sup>, Alessandro Marroni<sup>1</sup>, Ramiro Cali Corleo<sup>1</sup>, Peter Germonpré<sup>1,5</sup>

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**Introduction:** Previous retrospective studies showed an increased incidence of cerebral type decompression sickness (DCS) in divers with patency of the Foramen Ovale of the heart (PFO). Magnetic Resonance Imaging (MRI) studies showed increased prevalence of cerebral lacunary images in divers with PFO. These necrotic spots are believed to be due to arterial embolisation of nitrogen bubbles through the PFO during the decompression phase. In order to test this assumption, we recently started a new comparative study, using MRI imaging, psychometric testing and trans-esophageal echocardiography (TEE). We present here the first preliminary results of the MRI investigations.

**Methods:** Thirteen healthy subjects (9 males and 4 females; 23-45 years old) underwent MRI testing. The subjects were fully informed and ascertained that none had a contraindication to MRI investigation. Three sequences were used: Sagittal T1 weighted, axial T2 weighted and axial FLAIR sequencing. T2 weighing is used to enhance the liquid-filled structures, whereas the FLAIR sequencing erases the circulating fluids and shows ischemic areas as hyperdense spots.

**Results:** None of the 13 tests showed any ischemic zone. In 3 of the subjects a large PFO was known to be present (previous TEE examination) yet no hyperdense cerebral zones were present even while every diver did more than 200 “belgian” deep and cold dives. To ensure the precision and repeatability of the MRI investigation, two divers passed a second MRI one week later using the same standardized slices. No difference was detected in the MRI images by two independent radiologists accustomed to MRI analysis.

**Conclusion:** These preliminary results do not consent us to conclude but the absence of cerebral lesions even with known patency of the Foramen Ovale in divers is interesting. The repetition of the test in two divers after one week seems to confirm the good reproducibility of the examination and does not support the hypothesis of transient hypoperfusion and variable “ischemic” images. The same control test will be performed in any subject showing lacunary images, in order to clearly ascertain the reproducibility of MRI.

#### **A30 INSPIRED AIR TEMPERATURE AND HUMIDITY DO NOT INFLUENCE BRAIN STEM TEMPERATURE: IMPLICATIONS FOR INHALATION REWARMING THERAPY OF HYPOTHERMIC VICTIMS.** Igor B. Mekjavic<sup>1,2</sup>, Klemen Rogelj<sup>3</sup>, Maja Radobuljac<sup>3</sup>, Ola Eiken<sup>4</sup>

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**Background:** Inhalation rewarming is an effective method for eliminating respiratory heat loss. Numerous studies have also proposed that it is effective in enhancing the rate of rewarming of hypothermic individuals, but this view has been challenged by studies which have demonstrated that both in laboratory and simulated field settings, inhalation rewarming provides no improvement over spontaneous rewarming. The present study tested the hypothesis that inhalation rewarming may provide a thermal increment to central neural structures adjacent to the nasopharyngeal region, specifically the brain stem, medulla and hypothalamus. In this manner, inhalation rewarming, though not capable of enhancing the rewarming rate of body core temperature, might act to stabilise the temperature and hence the function of vital structures in the central nervous system, responsible for respiration, cardiac function and temperature regulation.



**Materials and Methods:** This hypothesis was tested by monitoring the auditory evoked brain stem responses (AEBRs) of fourteen subjects (7 male and 7 female) inspiring room air (24°C) followed by hot air (41°C) saturated with water vapour and cold dry air. The order in which the latter two conditions were presented to the subjects was counterbalanced. The latencies of peaks I, III and V, and the interpeak latencies (IPLs) I-III, III-V, and I-V were compared between the three conditions with a repeated measures ANOVA. Changes in IPLs are sensitive makers of changes in brain stem temperature. The total duration of each condition was 25 minutes, and AEBRs were recorded during the last 10 minutes. Prior to the measurement of AEBRs tympanic temperature (Tty) was measured with an infra-red tympanic thermometer.

**Results:** There were no significant differences in Tty, peak latencies I, III, and V, and IPLs I-III, III-V, and I-V. The results indicate that inhalation of either hot or cold air does not influence Tty, nor does it influence the temperature of the brain stem.

**Conclusions:** We conclude that inhalation rewarming is not capable of warming the vital central neural structures adjacent to the nasopharynx in any significant manner. Consequently, it appears unlikely that inhalation rewarming is an effective means of reviving brain stem and hypothalamic function in either shivering or non-shivering hypothermic victims.

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**A31 HUMAN BEHAVIOURAL THERMOREGULATION DURING HYPOXIC EXPOSURE.** Petra Golja and Igor B. Mekjavic. Department of Automation, Biocybernetics and Robotics, Institute Jozef Stefan, Ljubljana, Slovenia & Institute of Biomedical and Biomolecular Sciences, University of Portsmouth, United Kingdom.

**Background:** Prolonged exposure to hypoxia initiates functional changes in neural activity. The present study tested the hypothesis that such changes in central neural activity may result in impaired perception of temperature and subsequently alter behavioural temperature regulation. In high heat loss ambients, appropriate behavioural thermoregulatory responses may prevent deleterious cooling of deep body temperature. Any hypoxia-induced impairment of behavioural thermoregulatory responses may therefore predispose individuals to enhanced core temperature cooling and ultimately hypothermia.

**Materials and Methods:** Eight healthy male volunteers participated in two separate trials. In each trial, following a 15 minute rest period they were immersed to the neck in 28°C water for 90 minutes, and inspired either room air (Air) or a hypoxic gas mixture (Hypoxia: 11.5 %O<sub>2</sub>, 88.5 % N<sub>2</sub>). During the immersion, the subjects' right hand was placed in a separate bath perfused by water. At regular intervals the subjects were instructed to adjust the temperature of the water perfusing the hand bath to a comfortable temperature, by manipulating a valve. The adjusted temperature of the water perfusing the hand bath was termed the 'preferred hand temperature' (Tpref). At minute intervals, esophageal (Tes), rectal (Tre) and mean skin (Tsk) temperatures, and ventilation (V<sub>I</sub>) were recorded. The observed responses between the two conditions were compared with a one-way ANOVA.

**Results:** Tsk was maintained at 28.1°C throughout the immersion in both conditions. During the immersion, Tes decreased from 36.7°C in both the Air and Hypoxia conditions to 35.7 and 35.9°C, respectively. Similarly, Tre decreased from 36.9°C to 36.2°C in both the Air and Hypoxia conditions. Concomitant with the decrease in deep body temperature was an increase in Tpref. There was a large inter-subject variability in the initial and final Tpref. The average increase in Tpref during the immersion was 2.3°C in the Air, and 2.6°C in the Hypoxia condition; the difference was not statistically significant. With the exception of V<sub>I</sub>, which was significantly higher in the Hypoxia condition, there were no significant differences in any of the recorded variables between the Air and Hypoxia conditions.

**Conclusions:** Assuming that the selection of preferred hand temperature represents a behavioural thermoregulatory response, the present results suggest that normobaric hypoxia induced by inhalation of a gas mixture containing 11.5% O<sub>2</sub> does affect behavioural thermoregulation.

**Acknowledgements:** This work was supported by the Ministry of Education, Science and Sport (Republic of Slovenia).

**A32 RESCUE LIFTING SYSTEM AND SARRRAH-PROJECT: RECOVERY DEVICE FOR THE RESCUE OF DIVERS AND MEN-OVERBOARD IN ACCIDENTAL HYPOTHERMIA.** Baumeier W.<sup>1</sup>, Schwindt M.<sup>2</sup>, Bahlmann L.<sup>1</sup>, Schmucker P.<sup>1</sup>

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**Objectives:** Rescue of helpless persons from the sea is very demanding for rescuers and the available rescue devices alike. The most difficult step in the technical rescue chain is to bring this person onto a safe and secure

platform. The devices which have been available for this purpose have been inadequate up to now, and their use has been associated with a high risk of death for the most seriously hypothermic accident victims. The following details of accidental hypothermia at sea, in conjunction with rescue measures, will be explained, as well as a double strap system (RLS – Rescue Lifting System) for gentle and safe rescue from the sea. The medical project ‘SARRRAH’ (Search and Rescue, Resuscitation and Re-warming in Accidental Hypothermia) studies the rescue and treatment process of severely hypothermic patients and also includes the specific details, which are affecting the patient’s health status within the scope of technical rescue and transport.

**Background:** An accident at sea including immersion will almost always lead to accidental hypothermia. Hypothermia has an enormous influence on the physiological functions of the human body. The metabolic turnover slows down and impairs the organic functions. The effects on brain, muscle and circulation functions, is of importance for the technical rescue of hypothermic patients. The circulatory collapse can come about as a result of a number of factors: loss of hydrostatic assistance to venous return, and re-imposition of the effects of gravity, hypovolaemia, increased blood viscosity, diminished work capacity of the hypothermic heart and reduced time for coronary filling, dulled baroreceptor reflexes, unmeetable demands to perfuse skeletal muscle, psychological stress and pre-existing coronary disease.

The rescue operation is only the first step of the emergency treatment of patients with severe accidental hypothermia. The correct and gentle handling of the victim must be followed by specialized medical procedures of First Aid and transportation to specialized centres, which have the facilities to warm up these patients even under the influence of cardiac arrest.

The optimization of all successive procedures determines the recovery and therefore the final outcome. Statements about the efficiency of a specific rescue procedure can only be made, if the treatment and healing process to the point of discharge from hospital is being monitored by scientific criteria. These kinds of studies are demanding and have not been made so far.

**Conclusions:** It is a basic fact that people with hypothermia have a much better chance of successful resuscitation than critically ill people with normal body temperature, but only if the hypothermia is sufficiently taken into consideration throughout rescue and medical intervention. It is therefore necessary to establish clear and realistic procedures for emergency response, rescue and medical treatment based upon up-to-date and proven knowledge. The first step in the rescue chain must be done perfectly. Golden (4) made it absolutely clear, that “removal from water in the horizontal posture is preferable, in all circumstances, if it can be achieved. It also appears very likely that any demand for physical effort on the part of the victim at the time of rescue carries a risk of precipitating collapse and death. With the inevitable “circumstances permitting”, immersion victims should be handled with the utmost gentleness and as the potentially critically ill patients that they are.”

On the other hand, if rough circumstances do not allow this gentle handling, no time should be wasted and risk to the life and health of rescuers or the loss of the victim should be minimised. If the person with severe accidental hypothermia is handled in a rough manner, a fatal circulation arrest can be the result. Trained rescuers will have little problem performing resuscitation procedures to the point of arrival at a hospital which is equipped with a device for rewarming under extracorporeal circulation (3, 6). The tolerance of the hypothermic brain against hypoxia is well known and the chance of survival is very high (1).

Normal sling arrangements can cause discomfort to the arms and chest, which may lead to cardiovascular stress and an increase in heart rate due to possible restricted breathing. The newly developed ‘Rescue Lifting System’ seems to prevent rescue stress and also rescue collapse. The SARRRAH project will help to find out which equipment and procedures will lead to the best outcome, i.e. which will help the most persons with severe hypothermia to survive.

### A33 ARTERIAL BLOOD GAS PARTIAL PRESSURES DURING DIVING IN ELITE APNEA DIVERS.

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**Background:** Unassisted elite apnea divers regularly achieve dive depths of 50 – 80 msw and diving times of 5 – 6 min. Several studies have been reported on *alveolar* gas partial pressures in apnea divers, both during simulated as well as free diving (1, 2, 3). Up to now data on *arterial* blood gas partial pressures, however, are only available from short-term dives to shallow depths (4). Therefore we measured arterial blood gases, acid-base-status and lactate concentrations in elite apnea divers during diving in a wet tank.

**Materials and Methods:** 2 members of the German apnea national team volunteered for the investigation. The dives were performed in the wet tank of the pressure chamber of the Deutsche Lebens-Rettungs-Gesellschaft, Berlin, and consisted of exposures to 20 msw. Descent and ascent times were 1 min each, bottom times were 2 and 3 min, respectively. Blood samples were taken from an indwelling arterial catheter before the dive, at the beginning and the end of the bottom period, and immediately, 10 and 30 min after the dive. Measurements were performed

using an ABL 720 blood gas analyzer (Radiometer). The measured blood gas partial pressures were compared with the values predicted by a mathematical model developed to simulate compression- and decompression-related gas exchange effects of apnea diving.

#### RESULTS:

	APNEA TIME MIN	PO <sub>2</sub> MMHG	PCO <sub>2</sub> MMHG	PH	LACTATE MMOL/L
<b>DIVER M. H.</b>					
BEFORE DIVING	0	127	23	7.53	1.0
START BOTTOM TIME	1:05	226	35	7.39	1.5
END BOTTOM TIME	4:09	158	43	7.36	1.8
SURFACING	5:05	37	40	7.39	1.7
<b>DIVER S. P.</b>					
BEFORE DIVING	0	104	29.5	7.47	0.7
START BOTTOM TIME	0:55	225	43.5	7.36	1.1
END BOTTOM TIME	2:51	103	45	7.36	1.1
SURFACING	3:51	25	43	7.38	1.3

**Conclusions:** The directly measured arterial PO<sub>2</sub> mirrors the theoretical values predictable from the combined effects of compression, decompression and oxygen uptake. The increase of the arterial pco<sub>2</sub> is less pronounced than expected from the measurements of alveolar gas concentrations reported in the literature for comparable dives (5) but confirms data reported for free-diving weddell-seals (6). This seemingly striking finding, however, is confirmed when mathematical modelling based on blood and gas volumes and solubilities is used for the prediction of alveolar-arterial co<sub>2</sub> exchange during the compression phase. In addition, the haldane-effect on co<sub>2</sub> solubility in arterial blood probably assumes importance during the ascent.

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#### A34 NEW METHOD FOR RESCUER ARTIFICIAL VENTILATION SUITABLE FOR ENVIRONMENTAL CONDITIONS WITH REDUCED AMBIENT PRESSURE. Till S. Mutzbauer<sup>1</sup>, Birger Neubauer<sup>2</sup>, Peter H.J. Mueller<sup>3</sup> Kay Tetzlaff<sup>4</sup>

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**Specific objective:** It has been suggested that emergency artificial ventilation without the use of emergency respirators may be limited by low oxygen concentrations in a victim's airway. Even lower oxygen partial pressures result under conditions with low ambient pressure. Several closed circuit rebreathing devices exist that facilitate application of high inspiratory concentrations of oxygen. Capabilities of non-anesthesiologists using such devices for controlled ventilation might be restricted, however.

**Materials and methods:** A disposable closed oxygen rebreathing circuit (Clear-Flo Circle System 2133, Intersurgical Ltd., Berkshire, UK) was modified replacing the breathing bag by a mouthpiece, which was connected to the soda lime absorber by a hose. All of the four authors ventilated an artificial lung that was connected to the mask port of the rebreathing circuit using a tidal volume of 600±50ml. A pressure in the system exceeding 10 mbar was avoided by integration of a PEEP-Valve (Ambu International A/S, Broendby, Denmark) into the hose downstream to the mouthpiece. At the beginning of the experiment the artificial lung contained 5 Liters of expired air simulating the content of a victim's lung. The rebreathing system was as well purged initially by exhaled air of the "rescuer" to obtain initial oxygen concentrations in the inspiratory hose (FiO<sub>2</sub>) of the system below 17% as measured by an oxygen sensor (HAUX OXIMETER, Haux, Karlsbad, Germany. The system's overpressure valve at the soda lime absorber inlet was adjusted to a semi closed position. Oxygen flow into the rebreathing device was either adjusted to 1 L/min or to .5 L/min. 10 cycles of cardiopulmonary resuscitation (CPR) ventilations according to the latest international guidelines were administered with the help of an audio tape where reproducible counts had been recorded. At the end of the two CPR ventilations within each cycle FiO<sub>2</sub> was recorded. 10 cycles were observed in each experiment.

**Description of results:** In the semi closed mode using an oxygen flow of 1 L/min FiO<sub>2</sub> exceeding 30% (median 31.7%) was achieved after 9 cycles by all rescuers. With an oxygen flow of .5 L/min FiO<sub>2</sub> values exceeded 25% (median 26.7%) after 9 cycles.

**Conclusions:** The modification of the system as described could be used for artificial ventilation of victims with cardiac arrest by less experienced rescuers who are capable of administering mouth to mask rescue breathing e.g. in situations when CPR is required at high elevation remote locations or in the cabin of operating aircraft.

**A35 ROLE OF PATENT FORAMEN OVALE (PFO) AND MRI LESIONS IN UNEXPLAINED DIVING ACCIDENTS (DCS II).** Andreas Koch, H. Kirsch, A. Rump, E. Bettinghausen, H. Rieckert. Schiffahrtsmedizinisches Institut der Marine; Kronshagen & Sportmedizin, Christian-Albrechts-Universität, Kiel, Germany

**Introduction:** It is known, that divers with a PFO have an increased risk for severe diving accidents due to paradoxical cerebral breathing-gas embolism through a Right-/Left- (R/L) Shunt. 18 cases of unexplained mild DCS II and 18 matched divers in a control group were examined for a PFO and for cerebral lesions in MRI, if in this DCS II-group, like in typical and more severe cases, an increased prevalence of PFO and typical lesions in MRI could be found.

50 completely asymptomatic divers ( $35 \pm 11$  ys., median of 500 dives) were studied, if this group also has accumulated cerebral lesions due to repetitive gas embolisms.

**Methods:** T1-, T2- and diffusion-weighted Brain-MRI to detect abnormalities, suspicious to be residua of small arterial gas-embolisms. PFO-detection with simultaneous Transcranial Doppler-ultrasound (TCD) and Transthoracic Echocardiography (TTE) with Echo-contrast.

**Results:** PFO found in 18 (36%) of the 50 divers, 10 shunting only after Valsalva-maneuver, 8 with spontaneous shunt. Two times R/L-Shunt in TCD with negative TTE, one time TTE clearly positive with negative TCD. 18 both negative in MRI and PFO-detection. 137 cerebral abnormalities were found, 56 in 10 of the 18 PFO-Positives and 81 in 14 of the 32 PFO-Negatives (56% vs. 44%, n. s.). Age and number of dives vs. number of MRI-abnormalities (both  $p < 0.05$ ) were significant correlated.

**Conclusions:** The Combination of TCD and TTE improves bubble-detection in R/L-Shunting. In the examined group of divers, an overall high prevalence and number of MRI-abnormalities was found, with a tendency towards an even higher prevalence of MRI-lesions in the PFO-positives.

**A 36 NO INCREASED INCIDENCE OF PATENT FORAMEN OVALE (PFO) IN UNEXPLAINED DIVING ACCIDENTS (DCS II) WITHOUT OBVIOUS MRI-LESIONS.** Andreas Koch, H. Kirsch, A. Rump, P. McCormack, E. Bettinghausen, H. Rieckert. Schiffahrtsmedizinisches Institut der Marine; Kronshagen, Sportmedizin Christian-Albrechts-Universität, Kiel, Germany

**Introduction:** Divers with a PFO have an increased risk for severe stroke-like diving accidents due to paradoxical cerebral breathing-gas embolism through the Right-/Left-Shunt, but the role of a PFO in mild DCS II with minor neurological symptoms is still unclear. 18 cases of unexplained mild DCS II ( $37 \pm 10.2$  ys.; mean of 888, median of 170 dives) and 18 matched divers in a control group  $40.3 \pm 12.7$  ys.; mean of 870, median of 440 dives) were examined for a PFO and for cerebral lesions in MRI, if in this DCS II-group, like in cases with severe neurologic symptoms, an increased prevalence of PFO and typical lesions in MRI could be found.

**Methods:** T1-, T2- and FLAIR- Brain-MRI to detect abnormalities, suspicious to be residua of arterial gas-embolisms, that caused the DCS II. PFO-detection with simultaneous Transcranial Doppler-ultrasound (TCD) and Transthoracic Echocardiography (TTE) with Echocontrast.

**Results:** PFO found in 6 (33%) of the 18 DCS-cases, 4 with a high-grade shunt ( $> 20$  signals in TCD), and in 8 (44%) divers of the control group, 6 presenting a high-grade shunt. 5 Divers in the DCS-group showed cerebral lesions, multiple (10 lesions) in 1 diver. 8 divers in the control group had lesions, multiple (5,5,10,12 lesions) in 4 divers.

**Conclusions:** In this group of mild, unexplained DCS II, neither the incidence of a PFO, nor the number of cerebral lesions was increased compared to the control group. We conclude, that in our DCS II-cases without obvious findings in MRI, PFO played no major pathophysiologic role.

**A37 THE RISK OF DECOMPRESSION SICKNESS CAUSED BY HYPOBARIC ENVIRONMENT EXPOSURE AFTER DIVING (DRIVING TO ALTITUDE AND FLYING).** N. Naraki and M. Mohri. JAMSTEC: Japan Marine Science and Technology Center, Yokosuka, Japan

**Introduction:** The risk of Decompression Sickness (DCS) concerning the hypobaric environment exposure by ascending in an automobile and flying after diving was well known. However, the risk is increasing in recent years in Japan by the advance of the diving equipment and transport technology and by the debasement of the air fares. The diving in west-coast of Izu Peninsula, about 100 km west of Tokyo and also at the foot of Mt. Fuji, is very popular for recreational scuba divers, living in the Tokyo metropolitan area. Many of them return to Tokyo, driving pass the mountainous area in Izu Peninsula. The diving in Nansei Islands, locate in southernmost tip of Japan and in the subtropical area, also becomes very popular in these several years, and many divers fly back to the mainland. Therefore, we measured the altitude of the expressway which was main route from Izu to Tokyo. And we measured the internal pressure of the aircraft cabin from the Nansei Islands to Tokyo. In addition, we tried to verify the risk of flying after diving by the animal experiments with rats, the effects of hypobaric exposure 700 hPa (equivalent altitude of 3,000 m) after the hyperbaric exposure 0.4 MPa (equivalent depth of 30 m).

**Methods:** The altitude of the expressway from Izu to Tokyo and of the aircraft cabin from Nansei Islands to Tokyo and also some other domestic and international air lines were calculated from the pressure, measured with the portable apparatus on the vehicle and on the passengers respectively. On the animal experiments, since the definition of the short term diving on rats is difficult, we used the profile of saturated diving (30 m depth for 150 minutes, compression and decompression rates were 10 m depth per min), after the decompression from diving, we put the five interval times (1, 5, 10, 20 and 30 min.) before the decompression start to the six hypobaric conditions (equivalent altitudes of 500, 1,000, 1,500, 2,000, 2,500 and 3,000 m, with the decompression rate of 3,000 m altitude per min. and the pressure was hold for 30 min. in each hypobaric conditions, then the rats were compressed or returned to the atmospheric condition, with the rate of 3,000 m altitude for 10 min. The DCS levels were evaluated with the observed signs and symptoms as follows; non DCS, slight DCS with scratching skin, middle DCS with respiratory distress and serious DCS with the death. The seven rats for each combined experimental conditions (5 intervals and 6 altitudes) were exposed to the hyper- and hypobaric environments, but we have not conducted the experiments for the conditions when we can estimate the non-DCS.

**Results and discussion:** The highest point in the expressway from Izu peninsula to Tokyo was only 466m (960 hPa), we arrived there after 20 min. drive from the near entrance of diving spots. Generally the internal pressure of the aircraft cabin were linearly reduced to 800 hPa-755 hPa (corresponding to the altitude of 2,000 m- 2,570 m), during about 15 min. from the take-off for the flights at around 10,000 m altitude. The cabin pressure was hold for the cruising flight, then it was returned to the atmospheric pressure for 15 min. during the descent. On the animal experiments, we found the high level DCS over 1,500 m altitude for short interval conditions, and no DCSs at longer intervals than 20 min.

From our results, it was confirmed that the aircraft utilization is very dangerous after diving and the interval time between the diving and flying is very important, as it was forbidden in textbooks of the diving technology.

**A38 MEDICAL PROBLEMS IN RECREATIONAL DIVERS IN YUGOSLAVIA: ASSESSMENT OF FITNESS TO DIVE AND ANALYSIS OF DIVING RELATED INJURIES DURING THE PERIOD 1994-1998.** Dragana Ivkovic, Nikola Dekleva, Nenad Dikic, Milos Zarkovic, Branislav Misa Zivojnovic, Milorad Djuknic, Zoran Vidovic and Alessandro Marroni.

**Background:** Medical problems in Yugoslav recreational divers have not been evaluated during last decade in spite of the fact that the population of recreational divers is enlarging.

**Purpose of the study:** Evaluation of initial medical exam results in novice divers. Analysis of diving related injuries during 5 years period. Risk estimation for later diving related injury in divers with "health abnormalities" on initial examination.

**Materials and methods:** 292 candidates for recreational diving were examined (221 men and 71 women). Medical examination included medical history, physical examination, Chest X-Ray, EKG, blood pressure, FEV1/FVC% and flow-volume loop, audiogram, laboratory tests. Five years follow up and recording of diving related injuries in 620 recreational divers (average age 27,3; SD 6,51; I=15-46) who performed 6775 dives in 220 days. 892/6775 (13%) dives were deeper than 30m and 936/6775 (14%) were repetitive dives.

**Results:** 19/292 (7%) candidates were unfit to dive. Fitness to dive was assessed in 273/292 (93%) divers. Among them 98/273 (36%) had some medical condition which was not absolute contraindication for diving (36% ENT problems, 26% CV, 14% abnormal laboratory tests, 13% respiratory problems, 6,5% CNS, 6,5% other). Frequency of predominant injury- middle ear barotrauma, recorded in 36% (225/620) divers, was compared between novice and experienced divers and between completely fit and those who had some medical problem on initial examination. (Fisher exact test and Risk estimate-Odds Ratio). Novice divers who have ENT problem on initial examination are more frequently injured ( $p < 0,001$ ) than other novice divers without problem and have 5 times greater risk to get middle ear barotrauma than expert divers who also have some ENT problem on initial exam.

**Conclusions:** Some health abnormality was recorded in 40% (117/292) candidates for recreational diving. Results of initial medical examination could estimate risk for certain diving related injuries. Good diving technique is essential for prevention of middle ear barotrauma. There were no decompression illness or pulmonary barotrauma. Risk for DCI is  $< 1/6775$  dives for all dive profiles.

**A39 THE INFLUENCE OF INCREASED PRESSURE AND INCREASED OXYGEN PARTIAL PRESSURE ON THE CORROSION OF DENTAL ALLOYS.** Frank Hebllich\*, Rudolf Lenz\*, K. Ludwig.

\* German Naval Medical Institute, Kronshagen, Germany, ° Clinic for Dental Prosthetics and Dental Materials of the Christian-Albrechts-University of Kiel

**Introduction:** During the regular medical test regarding their fitness for diving, navy scuba divers have repeatedly reported signs of wear on their dental restorations. Firemen, who frequently work with oxygen masks, also complain about defects on cast restorations and amalgam restorations. Based on these indications, we have tested various dental alloys for their corrosion resistance during excess pressure and increased oxygen partial pressure.

**Test method:** Proof samples of three dental alloys of the company Jensen GmbH in Metzingen (pd-based alloy „Reliance“ Pd64Ag25Zn9Ga2, low gold content alloy „Landmark“ Au59Ag29Pd8Zn2In2, high gold content alloy „JP-84“ Au84Pt8Pd5In2) were subjected to a saliva substitute (0.1mol/l lactic acid, 0.1 mol/l sodium chloride) for seven days in a statistic immersion test according to DIN EN ISO 1562. Afterwards, the amount of ions dissolved in the liquid was determined by atomic absorption spectrometry. The jars with the liquid and the test castings were stored as follows:

2 jars with three castings each in a „normal“ atmosphere (oxygen partial pressure: 0.21 bar)

2 jars with three castings each in compressed air at 6 bar (oxygen partial pressure: 1.26 bar)

2 jars with three castings each in „Nitrox B“ at 5 bar (breathing gas mixture of 60% oxygen and 40% nitrogen, oxygen partial pressure: 3.0 bar).

**Results:** A direct relationship between increased ion particle solubility as measure for corrosion and an increased atmospherical pressure could not be determined for either of the three alloys.

The solubility of zinc from the alloy „Reliance“ decreased noticeably with an increase of the overall pressure. When the pressure was increased from one to five bar, zinc solubility decreased to 2/5 of the initial value. A further increase of the pressure from 5 to 6 bar resulted in a further drop of zinc solubility by 1/3, so that there seems to be a linear correlation between pressure and zinc solubility following the formula  $y = -0.53x + 4.4$ .

For „Reliance“, we also noticed a linear increase of the solubility of gallium- and silver ion particles with an increase of the oxygen partial pressure. The amount of gallium ion particles measured at 0.21 bar ( $4.39 \mu\text{g}/\text{cm}^2 \times 7\text{d}$ ) increased to  $7.36 \mu\text{g}/\text{cm}^2 \times 7\text{d}$  at 1.26 bar and reached a maximum of  $10.01 \mu\text{g}/\text{cm}^2 \times 7\text{d}$  at 3.0 bar. Again, there seems to be a linear correlation between gallium solubility and the oxygen partial pressure (formula  $y = 2.8x + 1.63$ ). The amount of dissolved silver ion particles at an oxygen partial pressure of 0.21 bar doubled with an increase of the oxygen partial pressure to 1.26 bar and doubled again at a oxygen partial pressure of 3.0 bar (correlation formula  $y = 1.2x - 0.07$ ).

**Conclusion:** The results of our tests support the impression that the increased ion particle solubility resulting from an increased oxygen partial pressure is partly responsible for an increased corrosion of the palladium-based alloy we tested. Especially because of a potential toxicity of the dissolved gallium, we recommend to reconsider with caution the use of this alloy for professional divers.

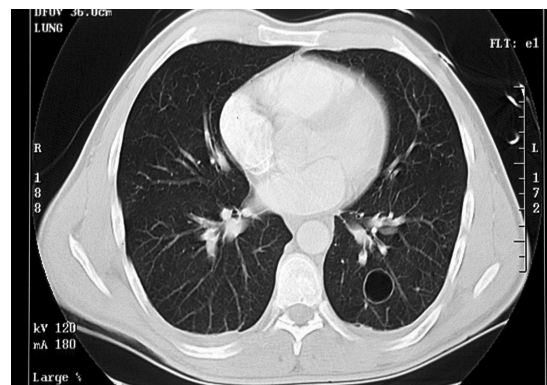
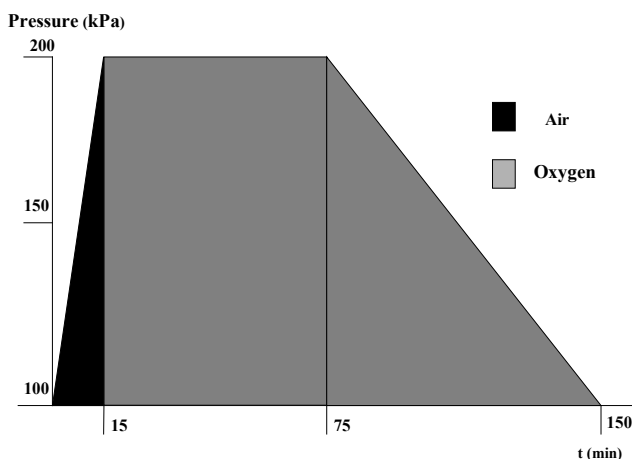
#### A40 MANAGEMENT OF A COMPLEX DIVING ACCIDENT - A CASE REPORT OF RELAPSED ARTERIAL GASEMBOLISM (Video-Presentation). Armin Kemmer<sup>1</sup>, Adel Taher<sup>2</sup>

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Scuba diving may cause neurological damage to the spinal cord. We report a case of severe decompression illness caused by arterial gas embolism due to pulmonary bullae unknown before the accident.

After a 25 metres dive in Egypt, the patient developed paraesthesia and weakness of all four extremities within the first minutes of surfacing. Normobaric oxygen was given immediately. Treatment acc. US Navy Tab. 6 began within 2 hours after the accident. During the hyperbaric treatment the patient became incomplete tetraplegic. Hyperbaric therapy was continued until he was repatriated by air ambulance on day two.

On admission in our clinic the MRI showed myelon oedema and gas bubbles in the cervical spinal cord. We continued hyperbaric treatment and saw an improvement of the neurological symptoms. Five minutes after the second treatment in our decompression chamber the patient developed symptoms of shock and paraesthesia of his



whole body. CT-scan of the thorax showed two bullae, but no pneumothorax.

We discontinued hyperbaric therapy for one week because of the high risk of further gas emboli. There was no improvement of the neurological symptoms in this time. The patient was incomplete tetraplegic up to TH3. After intensive discussion the patient agreed to continue decompression therapy, despite the high risk of further gas emboli.

We used a treatment protocol with 200 kPa over 150 minutes and prolonged decompression-time of 1½ hours. To reduce the risk of AGE the patient was breathing oxygen for the whole treatment time. Mild symptoms of AGE occurred twice during the treatments, but symptoms disappeared within few minutes after decompression. After 33 treatments and excessive physiotherapy, even during hyperbaric treatment the patient was able to walk without crutches. On discharge, there was a slight muscular weakness of the right leg and a low-grade urinary dysfunction.

**Conclusion:** Even in cases of repetitive AGE during hyperbaric oxygen treatment of severe diving accidents one should consider to continue treatment with a modified protocol. Decompression-time should be prolonged and there should be no air-breaks during the whole treatment. Excessive physiotherapy, even inside the hyperbaric chamber seems to have an additional positive effect.

#### **A41 PROJECT HORTIS: INVESTIGATING RADIOTHERAPY-INDUCED LATE EFFECTS OF NORMAL TISSUE WITH HYPERBARIC OXYGENATION.** Dick Clarke. The Baromedical Research Foundation, Columbia, South Carolina, USA

When evaluating therapeutic effectiveness, modern medical practice places great emphasis on high levels of clinical evidence. Hyperbaric medicine fares poorly in this regard. Much of its present application relies on laboratory data and uncontrolled clinical experience. The need to improve the level of supportive evidence is both paramount and immediate. An increasing number of those who are financially responsible for the provision of hyperbaric oxygen therapy are limiting or withdrawing support. Project HORTIS (Hyperbaric Oxygen Radiation Tissue Injury Study) has been introduced to address this shortcoming. As the first of several planned controlled and double-blinded clinical trials, HORTIS will concurrently investigate radionecrosis at seven anatomic sites. An eighth "prophylactic" arm is included. HORTIS is multicenter and international in scope. Patients are presently under protocol at one institution. It is intended that nine additional institutions will be enrolled by the end of 2001, in eight different countries. Should this level of recruitment be achieved, eight publications of a high level of evidence will be submitted for publication within three years. This presentation will overview HORTIS, with emphasis on entry criteria, the randomization process, therapeutic and placebo protocols, treatment algorithms, and the blinding process.

#### **A42 THERAPEUTICAL HYPERBARIC FACILITIES IN EUROPE ARE MEDICAL DEVICES: CONSEQUENCES.** Robert Houman, Centre for Hyperbaric Oxygen Therapy, Military Hospital Brussels, Belgium

The European Directive CE 93/42 is based on the concept of new approach and global approach. Free movement of products is a cornerstone of the Single Market. The mechanisms in place to achieve this aim are based on prevention of new barriers to trade, mutual recognition and technical harmonization.

A number of European Directives / Guidelines exists regarding hyperbaric equipment in general, that may have important consequences for the use of hyperbaric oxygen treatment (HBO) chambers in particular. There is necessity for those responsible for the operation of hyperbaric treatment chambers to conform to Standards regulating medical equipment, which in fact any HBO chamber is.

It has to be agreed that in the European legislation, there is a lack of uniformity regarding the standardisation and/or regulation of different aspects of HBO equipment and operation: safety of personnel, of patients, and technical safety. In response to this fact, a study has been undertaken to address the feasibility of a normalisation on a European level.

Keywords: European Directive CE 93/42, European Directive CE 97/23, Standards and Guidelines, Security of hyperbaric equipment, hyperbaric oxygen therapy – safety,

#### **A43 NECROTISING FASCIITIS, TRUNCAL AND NECK INVOLVEMENT, SUCCESSFULLY TREATED SURGICALLY, ANTIBIOTICS AND HYPERBARIC OXYGENATION: A CASE REPORT.**

Dusko Micevic, Dragan Micic, Voican Stanic, Vladislav Stepic, Ivanko Bojic, Slobodan Rudnjanin (Yugoslavia)

**Background:** Necrotising fasciitis is a progressive, rapidly-spreading, inflammatory infection located in the deep fascia, with secondary necrosis of the subcutaneous tissues. Because of the presence of gas forming organisms, sub-cutaneous air is classically described in necrotising fasciitis. This may be seen only on X-ray, MRI or not at all. The speed of spread is directly proportional to the thickness of the subcutaneous layer. It moves along the deep



fascial plane. These infections can be difficult to recognize in their early stages, but they progress rapidly. The causative bacteria may be aerobic, anaerobic, or mixed flora, and the expected clinical course vary from patient to patient. Treatment is difficult, often irrational, and almost “one step behind the facts”, because early recognition is difficult, and aetiology, bacteriology, and the clinical course are sometimes poorly understood and expected to evolve in a different and more favorable way. We describe a case of necrotising fasciitis with neck and truncal involvement, successfully treated with surgery, antibiotics and finally with hyperbaric oxygen.

**Materials and methods:** Patient (28 years, male) was admitted to Military Medical Academy, Department of Infectious Diseases. The disease started suddenly two days before he came to hospital, with high fever which was 40 degree of Celsius, dehydration, headache, severe systemic reaction of toxemia. The patient was admitted to hospital with painful movement of the right arm, erythema in the right axilla that quickly spread in the next hours. The pain of the right shoulder and axilla increased. Laboratory findings, in the first 36 hours after admittance, there was progressive decreasing in the count of erythrocytes and hemoglobin, leucocytosis, granulocytosis, trombocytopenia, decrease of sodium and potassium and rapid increase creatin kinase. We made sonographic findings and revealed increased lymph gland in the right axilla 1,1 cm and after 24 h we made CT of the thorax and we found edema of subcutaneous layers in the right axilla and around tissues, while muscles of the lateral thorax were edematous and divided into layers. Thanks to the data from literature, and our little experience specialist of Infectious disease made assumption that it was necrotising fasciitis and started therapy with antibiotics as listed below.

**Description of results:** After the first surgery the diagnosis confirmed. The cause of this necrotising fasciitis was streptococcus hemolyticus group A. The thoracic surgeon made three debridements in the next three days. We continued with antibiotics therapy (cefpirome 4g/24h, clindamicine 2400 mg/24h, gentamicine 240mg/24h + cilastatin, metronidazole 1500mg/24h) followed with IV fluids and nutrition and also with correction of electrolytes. Three days later gentamicine was substituted with imipenem. HBO started after the second debridement and used as recommended the first European consensus conference on hyperbaric medicine (Lille 1994).

**Conclusions:** We have described a case of severe necrotising fasciitis, with neck and truncal involvement. Aggressive surgical debridement was the cornerstone in the management this case of necrotising fasciitis. There were three debridements and every day inspection of the whole infected area under general anaesthesia. Antibiotics treatment had important place, started immediately after admittance of the patient to the hospital. Hyperbaric oxygenation timely added to the whole treatment after the second surgery. Treatment scheme was: three times in 24 h for the first day; two times in the second and in the third day, and after that once in a day until infection was taken under control. There were twelve HBO treatments on 2,5 ATA, duration 90 minutes in the monoplace hyperbaric chamber BLK 301 produced by Space and Research Center Khrunicheva Moscow. Surgery, antibiotics and timely HBO treatments were successful in treating this severe case of necrotising fasciitis.

#### A44 HYPERBARIC OXYGEN THERAPY AS A TREATMENT MODALITY FOR CALCIPHYLAXIS.

Andreja Marn Pernat<sup>1</sup>, Radoslav Kveder<sup>1</sup>, Andrej F. Bren<sup>1</sup>, Rafael Ponikvar<sup>1</sup> and Igor B. Mekjavic<sup>2,3</sup>

<sup>1</sup>Department of Nephrology, University Medical Center Ljubljana; <sup>2</sup>Institute Jozef Stefan, Ljubljana, Slovenia &

<sup>3</sup>Institute of Biomedical and Biomolecular Sciences, University of Portsmouth, UK.

**Background:** Calciphylaxis is a rare, but potentially devastating ulcerative cutaneous disease in humans, described in the setting of advanced renal failure associated with secondary hyperparathyroidism and high calcium x phosphorus product. Typical pathologic findings include small vessel calcifications within the dermis and subcutaneous tissue. These vascular changes promote tissue ischemia resulting in tissue necrosis. Treatment is often unsatisfactory and mortality rates are between 50 and 100% (1). Within the last 10 years, the Department of Nephrology in Ljubljana has treated 4 cases of calciphylaxis (1 dialysis and 3 post-transplantation patients). Two patients are still alive. Recently, two reports (2, 3) have proposed that hyperbaric oxygen therapy (HBOT) may be successfully used to treat skin ulcers in calciphylaxis. We report a case of calciphylaxis-induced skin ulcerations that did not respond to conventional therapy until HBO was included as an adjunctive therapy.

**Materials and Methods:** A 50-year old woman with end-stage renal disease due to unknown primary renal disease had undergone haemodialysis for 15 years. Due to severe secondary hyperparathyroidism total parathyroidectomy with parathyroid autotransplantation was performed in 1997. After cadaveric kidney transplantation in June 1999 moderate renal failure persisted. In March 2001, she presented with a 3-week history of two small spontaneous ulcerations on the right lateral calf, which progressed to a large ulceration covered with thick necrotic eschar. The diagnosis of calciphylaxis was based on clinical presentation and evidence of small vessel medial calcifications revealed by skin biopsy. Despite normalized calcium x phosphate product, careful local therapy including surgical wound debridement and excluding calciphylactic challengers such as corticosteroids, the ulcerations continued to increase in size and number. HBOT was initiated two weeks after hospitalization, and comprised daily 90 min



sessions at 2.5 ATA, 5 times weekly. During each 90 min HBOT, three 25 min sessions of breathing 100% O<sub>2</sub> were separated by 5 min of breathing ambient air. To date, we have completed 35 treatments.

**Description of Results:** Within four weeks of commencing HBOT, the ulcers began to decrease in size and the base of the skin lesions became moist instead of dry and necrotic. The surrounding induration softened and the livedo resolved. Despite the successful healing of the primary ulcers, two necrotic lesions developed on the toe and 2<sup>nd</sup> finger of the other leg within 5 weeks of HBOT. Presently, these subsequent lesions are not responding to HBOT and are deteriorating.

**Conclusions:** HBOT offers effective symptomatic treatment of skin lesions in calciphylaxis. It does not, however, affect the underlying pathogenetic mechanism.

**Acknowledgements:** This work was supported by the Ministry of Education, Science and Sport (Republic of Slovenia)

**A45 NEUROPROTECTION BY THE USE OF HYPERBARIC OXYGENATION AFTER PERMANENT FOCAL CEREBRAL ISCHEMIA.** Holger Schade, W.-R. Schäbitz, R. Kollmar, J. Bardutzky, S. Heiland, S. Schwab, U. Carl. Department of Neurology and Neuroradiology, Ruprecht-Karls-University Heidelberg, Germany

**Background and purpose:** Hyperbaric oxygenation (HBO) treatment has been shown to have trophic and neuroprotective effects *in vitro* and *in vivo* in different lesion models. HBO has potent neuroprotective effects after hypoxic-ischemic injury and global ischemia. The role of HBO in focal cerebral ischemia is only partially understood. In the present study, we therefore evaluated by applying MRI-monitoring whether a clinically relevant systemic treatment with HBO can achieve a long lasting neuroprotective effect.

**Methods:** Male Wistar rats underwent permanent respectively transient (2h) occlusion of the right MCA by using the suture occlusion model. Two hours after MCAO the animals were treated with HBO (100% O<sub>2</sub> / 2,0 atm) for an hour. Classification of groups: A= permanent occlusion – HBO treatment (n 8), B= permanent occlusion - control (n 8) D=transient occlusion – HBO treatment (n 8), E= transient occlusion - control (n 8). Physiological parameters were measured during treatment. After 24h animals were sacrificed and TTC-stained.

Subsequently another two groups (permanent occlusion – HBO treatment resp. permanent occlusion – control) underwent MRI studies (DWI, PI, T2) beginning 90min after MCAO, as well as 3h, 5h, 24h, 48h, 72h and 120h after ischemia. After 120h animals were sacrificed and TTC-stained. The animals were weighed and neurologically assessed before each measurement (rating scale: 0=no deficit to 5=death).

**Results:** 30 min after HBO-treatment the physiological parameters showed no differences between the therapy and the control group except an 46% increase of the pO<sub>2</sub> level by the HBO treated animals (p<0.05, t-test). With HBO therapy a reduction of the mean infarct volume of 33% was achieved by permanent MCAO but there was no significant reduction by animals which underwent transient MCAO. This neuroprotective effect was proved by the use of MR-monitoring: The T2-weighted derived ischemic lesion volume at 24h after occlusion was 258,855±14,828 mm<sup>3</sup> versus 215,726±18,505 mm<sup>3</sup> (p<0.05), decreased to 236,511±30,629 mm<sup>3</sup> versus 180,147±13,932 mm<sup>3</sup> (p<0.05) at day 5 for controls and HBO treated animals, respectively. ADC derived lesion volume at 5h after MCAO was 211,17±39,904 mm<sup>3</sup> versus 175±22,15 mm<sup>3</sup> (p<0.05) and then decreased to 145,099±35,683 mm<sup>3</sup> versus 125,967±24,661 mm<sup>3</sup> (p<0.05) at 24h for controls and HBO treated animals, respectively. rrCBF showed no significant difference between HBO treated and control animals in basal ganglia and anterior cortex. Hyperbaric oxygen treatment increased rrCBF in the temporal cortex for 45,3% (24h) and in the parietal cortex for 22,1% (24h) compared to controls (p<0.05, ANOVA, F-test).

There was no significant difference in weight loss. There was less neurological deficit after ischemia in HBO treated animals with permanent MCAO compared to controls (p<0.05).

**Conclusion:** Treatment with hyperbaric oxygenation achieved a long lasting neuroprotective effect after permanent but not after transient occlusion as early as 5h after ischemia as measured by MRI. HBO may therefore represent a new approach to the treatment of permanent focal cerebral ischemia.

**A46 LEIPZIG'S HBO AMBULANCY: FINANCIAL ASPECTS.** Harald Englisch, Diethelm Kühnert, Leipzig, Germany

In the last year 7 HBO ambulancies were working in East Germany and additional chambers were affiliated at stationary institutions. The health insurances used to reimburse the treatment in Germany for many indications generally, the compulsory insurances paid e.g. 1,7 mill. DM in 1999 [1].

According to the decision of the German board of the insurances and physicians (NUB) the HBO therapy will not longer be paid by the majority of insurances [1]. Only private insurances and the labour co-operatives reimburse the HBO therapy any longer. Patients in Germany are emotionally and financially not prepared for paying important medical therapies from their own budget [2].

The German organisation of HBO chambers (cf. [www.vdd-hbo.de](http://www.vdd-hbo.de)) went to law against the NUB decision but a final result can be expected in several years only. In the meanwhile all but the Leipzig HBO ambulatory had been forced to close down in East Germany.

In the following we describe the Leipzig approach to continue its work in a difficult surrounding. The team of the Leipzig ambulatory started working on a bundle of measures, some of them only 1 or 2 month ago:

1. A full-time director is responsible for the commercial aspects, the chief physician has a high reputation.
2. The income of the employees is connected with the success of the ambulatory.
3. A communication trainer investigates the processes in the team.
4. An optimised information structure is mapped into the IT system.
5. The ambulatory is presented in the internet ([www.hbo-leipzig.de](http://www.hbo-leipzig.de)) and at professional flyers.
6. It is paid for advertisement in regional newspaper and at cars.
7. A regional football club is sponsored.
8. Physicians, representatives of patients organisations and the press are invited to trial diving tour.
9. Remuneration of private doctors according to the table GOÄ for private patients.
10. We are engaged to convince the insurances that from a health economic point of view the HBO therapy is for some indications very effective in comparison to the standard therapy.

#### Results:

1. The regional TV "mdr" (responsible for a region with about 10 mill. inhabitants) has reported about the ambulatory, cf. [www.mdr.de/hauptsache-gesund/sendungen/sendung20000403.html](http://www.mdr.de/hauptsache-gesund/sendungen/sendung20000403.html).
2. The newspaper for the region of Leipzig "LVZ" (responsible for a region of about 1 million inhabitants) has reported in a longer article.
3. The number of patients paying from their own pocket increased remarkably.
4. A cooperation contract with the municipal hospital of Leipzig ([www.sanktgeorg.de](http://www.sanktgeorg.de)) is signed.
5. A chamber associated with this hospital will be opened in the next year.
6. A Scientific Board for the ambulatory was established.
7. During the summer 2 diving tours each day took place.

#### Conclusions:

1. The health market as a market with very unstable boundary conditions needs a very flexible reactions.
2. The private HBO chambers can only survive if the economic experiences are shared as well.
3. The establishment of a non-profit organisation to support the chambers is discussed.

At the beginning of this month the HBO ambulatory in Dresden was reopened. You are invited to the corresponding Scientific Symposium at the end of September.

#### A47 STRESS -INDUCED HORMONAL AND MOOD RESPONSES IN SCUBA DIVERS: A FIELD STUDY. Udo Anegg<sup>1</sup>, Gabriele Dietmaier<sup>2</sup>, Alfred Maier<sup>1</sup>, Florian Tomaselli<sup>1</sup>, Sabine Gabor<sup>1</sup>, KlausWalter Kallus<sup>3</sup>, Frejya Maria Smolle-Jüttner<sup>1</sup>

Department of Surgery, Division of Thoracic and Hyperbaric Surgery<sup>1</sup>, University Medical School Graz, Department of Psychology, Division of Clinical and Health Psychology<sup>2</sup>, University Graz, Department of Psychology, Division of Applied Psychology<sup>3</sup>, University Graz, Austria.

**Introduction:** The majority of injuries in scuba-divers are attributable to inappropriate behavior under stressful diving conditions, predominantly involving panic reactions emerging from elevated levels of anxiety. Divers with an elevated level of anxiety are at higher risk to develop panic reactions than those possessing more adequate stress-coping-mechanisms.

**Patients and Methods:** In a comparison of two extreme groups with diametrically different coping strategies we determined the hormonal response (serum levels of prolactin, epinephrine, norepinephrine and saliva cortisol levels) and psychological parameters before and after immersion in a recreational and stressful.

**Results and Conclusions:** Prolactin was found to be a hormonal marker with a significant increase in the subgroup of the stress-controllers. The other observed somatic parameters showed significant increases during and after the dive, however without any significant group difference. Along with the self-reported emotional conditions under immersion these data suggest that an increased prolactin level reflects a state of elevated physical and mental activation and vigilance corresponding to the concept of "eustress".

#### A48 MEASUREMENT OF THE RADIOACTIVITY OF THE ADRIATIC SEA IN BOMBED AREA OF CAPE ARZA, BOKA BAY, MONTENEGRO, YUGOSLAVIA. Nenad Dikic, Branislav Misa Zivojnovic, Dragana Ivkovic, Snezana Pavlovic, Radojko Pavlovic, Marko Savovski, Predrag Radivkovic, Snezana Crnkovic, Bratislav Markovic, Nikola Dekleva. Yugoslavia

**Background:** On Sunday 30. 05. 1999. At 11:00am, NATO attacked Radanovici and Lustica near Herceg Novi and Tivat (Montenegro). Two years latter announced that Cape Arza has been bombed by depleted uranium on the area of 3500 m<sup>2</sup>. At the same time Montenegro.com reported that about 3 tones of depleted uranium has been dropped on Cape Arza and that government of FRJ proceed this information to the Government of Montenegro with one year delay. Federal Government stated that the Committee to monitor depleted uranium emissions is to be set up, but Representative of Federal Biro for Measurement announced that there is no equipment to measure uranium in Yugoslavia. The fact is that about 3 tones of depleted uranium are dropped to Montenegro. Two years latter there is no result about radioactivity in the area of Cape Arza. A problem of the contaminated sea and its shore is the special problem and nobody mentioned it. Community of divers are the most sensitive one, but the problem is much deeper since the seafood could be contaminated as well as people in that region. More then 2000 divers are doing their activities there. The most popular beach Zanjice in the whole Boka Bay is hundred meters from the potentially contaminated area.

**Aim of the project:** To measure the radioactivity of the sea, soil, sea flora and fauna as well as the land in the mentioned area of 3500 m<sup>2</sup>.

**Materials and methods:** Members of diving clubs Sebastian, Herceg Novi and Amphora, Belgrade have done several consecutive diversings in order to get sample of the sea, soil, sea flora and fauna. Ten samples have been provided. Specialists from VINCA, the only Yugoslav authorised laboratory, are doing all measurements and results should be publicly announced. After primary measurement, in the second step some medical examination could be done on the divers who dived two consecutive seasons in the bombed area.

**Description of results:** Results will be attached separately since VINCA doesn't have mass spectrometry and all the measurement are done on slow and difficult way. Final results will be done at the end of May 2001.

**Conclusion:** We don't want to predict the results since some of the well known specialist for the field of the radioactivity (Prof Dr James McLaughlin, Dublin, Ireland) could not say much about problem of the radioactivity and sea, but we are sure that measurement need to be done. All kind of help of diving population and organisation from abroad will be appreciated since this is totally new problem without significant experience. In the same time effects should be followed in long-term period. We considered this at least as a pilot study for some future necessary investigations.

#### **A49 EARLY 20<sup>th</sup>-CENTURY HYPERBARIC RESEARCH IN HAMBURG – THE WORKS OF ARTHUR AND ADELE BORNSTEIN.** Karl-Peter Faesecke. HyperMedConsult, Hamburg, Germany

On the occasion of the first international hyperbaric and diving medical meeting in the City of Hamburg, it seems appropriate to outline the contributions to the field that originated from the banks of the river Elbe early this century. The construction of the first underwater twin road tunnel in continental Europe was started here in July 1907; it was opened to the public exactly 90 years ago, on Sep.7<sup>th</sup>, 1911 and is still in full use today - its copper-covered escalator building being one of the characteristic land-marks along the northern riverside.

The digging of the two 426-m-tubes required intensive human labour under pressures up to 3.4 bar abs. After 232 cases of DCS within the first two months the Hamburg Medical Office looked for a qualified doctor to take care of the "tunnel cases". They appointed a 27-year old neurologist from Göttingen, Arthur Bornstein, born in Berlin of Jewish parents, who took his housing on the construction-site and initiated an all-inclusive occupational safety program from the first day on, performing strict physical exams on the workers, prolonging decompression times considerably, intensifying the use of the recompression chamber on site and experimenting on animals in it, together with his doctor-wife Olga-Adele.

Their work led to a considerable amount of "firsts", which hardly anybody in the field today is aware of, since they were published in German right before and during the first Great War. After 1918 German medicine had lost its world-wide influence, never to regain it...

To honour their contributions to the development of hyperbaric medicine and to again underline the importance of this meeting-site to the attendants, the most important ones are mentioned:

- 1<sup>st</sup> experimentally induced oxygen seizure in man (A.B. himself),
- 1<sup>st</sup> "life without blood" on dogs (carried out and published by O.A.B.),
- 1<sup>st</sup> application of oxygen in workers' decompression,
- 1<sup>st</sup> application of oxygen in hyperbaric recompression therapy,
- 1<sup>st</sup> calculation of nitrogen uptake via circulation,
- 1<sup>st</sup> identification and description of aseptic bone necrosis in caisson workers,
- 1<sup>st</sup> forensic post-mortems in decompression victims,
- 1<sup>st</sup> experimental investigation and explanation of "diver's squeeze".

After the tunnel's completion Bornstein was to become the first professor of pharmacology at the newly founded Hamburg University – but only after surviving more than four years as an army medical officer on the western front in WW I... His wife had died in childbed already in 1912. He followed her only 20 years later, thus being spared later Nazi-oppression, from which their only son, after having received his M.D. in Hamburg, emigrated to the United States of America in 1934, later serving in the Pacific theatre, then moving to Texas, where the family is still present.

The latest road tunnel (appr. 2.3 km) under the river Elbe is still under construction today. The boring process under pressure took more than three years and was completed in December of last year. The maximum pressure reached 5.5 bar (= 45 m); information on the applied preventive safety measures plus medical results will be presented during the current “2<sup>nd</sup> Arthur-Bornstein-Workshop” and in ensuing publications.

#### **A50 FIRE FIGHTING COURSE IN THE HYPERBARIC ENVIRONMENT.** Peter Germonpré, R. Houman. Advisory Committee for Hyperbaric Oxygen Therapy in Belgium (ACHOBEL)

The recent interest for hyperbaric oxygen therapy has resulted in a development of hyperbaric centres everywhere in Europe. Contrary to most of the previous hyperbaric centres, the personnel of these new hyperbaric centres are mostly not issued from the professional world of diving. This has often as consequence a sub-optimal technical formation, a lack of specific knowledge to the hyperbaric environment, a less than optimal perception and evaluation of the possible dangers of the hyperbaric system.

Fire and its consequences surely represent the biggest danger in a hyperbaric environment. Indeed, the increase of the partial pressure of oxygen will quickly lead to an uncontrollable extension of the fire, and this in a closed space without exits. In case of fire in the hyperbaric chamber, three parameters will quickly change: the temperature, the volumetric mass, provoking a pressure increase in a confined space, the partial pressure of the different composing gases with concomitant increased possible toxicity of these gases. Emergency intervention in case of fire is not limited to actions directed at extinguishing the flames but implicates a series of actions which must be executed in a quick and timely manner.

If it is true that this type of accidents are rare, never the less we must be aware that the consequences of fire in a hyperbaric oxygen chamber are almost always fatal for the occupants. From a analysis of the causes of fire inside hyperbaric chambers, (*P. Sheffield and D. Desautels*, Hyperbaric and hypobaric chamber fires: a 73 year analysis, *Undersea Hyperb Med*, 24 (3), 153) it is known that a large number of fires are the consequences of the objects taken inside the chamber by the patients. Moreover, the oxygen concentration in the hyperbaric chamber environment too often surpassed 28 percent! From this survey, casualties amounted to 60 dead and only 8 wounded, indicating that when a fire does happen, survival chances for inside occupants are small. So it is necessary to focus on a politic of prevention, rather than remedy. Two possible solutions can be outlined in order to cope with the fire safety problem: Active means for the fight against fire and preventive security.

It is obvious that, whereas all the means of active fight against fire can be provided, they can never be optimised without the creation and application of a preventive fire safety policy. Certainly in the case of preventive measures against fire, it is necessary that the personnel knows about all the aspects and dangers in the hyperbaric environment. In order to optimise the assimilation of all these measures by the personnel, a Course in Fire Fighting in the Hyperbaric Environment is proposed.

The theory part of the Course will focus on acquiring a profound knowledge of all the phenomena related to fire, a knowledge of the different risk factors and their consequences, and to create an awareness of the dangers. It aims at the sensibilisation of the hyperbaric personnel to the preventive fire safety measures. The practical training serves to demonstrate and acquire the correct reflex actions in case of fire, and this by creating the real conditions of fire in an hyperbaric chamber as an exercise. These exercises for the fight against fire will involve the use of all active means available for the extinguishment of the fire, in real time, and the organisation of the evacuation of the patients out of the hyperbaric chamber. This lesson is unique in Europe and is organised by the Advisory Committee for Hyperbaric Oxygen Therapy in Belgium (ACHOBEL). It is accessible for all categories of hyperbaric personnel (medical doctors, nurses and technicians). A certification of the Advisory Committee for Hyperbaric Oxygen Therapy in Belgium will be issued to the participants upon completion of the Course.

#### **A51 HYPERBARIC OXYGEN THERAPY: TREATMENT RESULTS IN THERAPY RESISTANT POSTIRRADIATION COMPLICATIONS IN PROSTATE TUMOURS.** Heiko Renner, Ramona Mayer, Sabine Gabor, Huberta Klemen, Alfred Hackl, Freya-Maria Smolle Jüttner. Division of Thoracic and Hyperbaric Surgery, Dept. of Surgery, University Medical School of Graz, Graz, Austria

**Background:** Chronic proctitis and cystitis are well known complications of external radiotherapy for prostate cancer. The result of hyperbaric oxygen (HBO) used in the treatment of these late and often therapy resistant effects are reported.

**Subject and methods:** Between March 1998 and September 1999, nine selected male patients (age, median 71 years; range, 69-76 y) with radiation induced proctitis/cystitis received HBO treatment within a median time of 26 months (range, 11-39) after megavoltage external radiotherapy for prostate cancer. Patients either suffered from radiation induced cystitis (3), proctitis (4), or both (2) RTOG/EORTC grade 2 (2) and grade 3 (7). Three patients suffered from unrelated second disease like myelodysplasia, plasmocytoma and amyloidosis. Three further patients had a history of diabetes; one of them and further two patients had anticoagulant therapy. Hyperbaric oxygen treatment was performed at the department of Thoracic Surgery and Hyperbaric Surgery in a multiplace chamber. Compression to 2.2-2.4 ATA was done while the patients breathed 100% oxygen over masks. The treatment was given for 60 minutes on a once-a-day basis, 7 days a week in one or two treatment courses; one treatment course consisted of 30 sessions of HBO treatment.

**Results:** HBO treatment was well tolerated in all patients. In two out of three patients with cystitis, two out of four patients with proctitis and both patients with combined proctitis/cystitis substantial improvement was obtained with complete remission of all symptoms in three patients. Further three patients had partial improvement of their symptoms, but these patients had stopped the HBO therapy before the planned end.

**Conclusion:** HBO treatment is a secure additive treatment option for those patients when conventional treatment has given unsatisfactory results.

## **A52 LATE RADIATION SEQUELAE IN WOMEN AFTER BREAST CONSERVING CANCER THERAPY: EFFECTS OF HYPERBARIC OXYGEN THERAPY (HBO).** Ulrich M. Carl<sup>1</sup>, J.J. Feldmeier<sup>2</sup>, G. Schmitt<sup>1</sup>, K.A. Hartmann<sup>1</sup>

<sup>1</sup> Department of Radiation Oncology, University Duesseldorf, Germany, <sup>2</sup> Department of Radiation Oncology, Medical College of Ohio, Toledo, USA

**Introduction:** Persisting symptoms after breast-conserving surgery and radiation are frequently reported. In most cases symptoms in the breast resolve without further treatment. In some instances, however, pain, erythema and edema can persist for years and can impact the patient's quality of life. Hyperbaric oxygen therapy (HBO) was shown to be effective as treatment for late radiation sequelae. The objective of this study was to assess the efficacy of HBO in symptomatic patients after breast cancer treatment.

**Patients and Methods:** Forty-four patients with persisting symptoms after breast-conservation therapy were prospectively observed. Thirty-two women received HBO in a multiplace chamber for a median of 25 sessions (7-60). One hundred percent oxygen was delivered at 240kPa for 90-minutes sessions, five times per week. Twelve control patients received no further treatment. Changes throughout the irradiated breast tissue were scored prior to and after HBO using modified LENT-SOMA criteria.

**Results:** Seven out of 32 women were completely free of symptoms after HBO. Only fibrosis and telangiectasia were not significantly affected by HBO. Treated patients showed a significant reduction of pain-, edema- and erythema-scores as compared to untreated controls ( $p < 0.001$ ). All 12 patients in the control group had persisting complains.

**Conclusion:** HBO should be considered as a treatment option for patients with persisting symptoms following breast conserving therapy. Fibrosis and telangiectasia are mainly considered important from a cosmetic point of view. In contrast HBO has a high impact on palliation from pain, edema and erythema, which are basic endpoints for life quality. Data are promising and prospectively randomised studies according to rules of evidence based medicine (EBM) are in preparation.

**Keywords:** HBO; breast cancer; breast injury; radiation sequelae

## **A53 EFFECTS OF HYPERBARIC OXYGEN (HBO) AND NORMOBARIC CARBOGEN ON THE RADIATION RESPONSE OF THE RHABDOMYOSARCOMA R1H.** Ulrich M. Carl<sup>1</sup>, K. A. Hartmann<sup>1</sup>, A. J. van der Kleij<sup>2</sup>, P. Sminia<sup>3</sup>

<sup>1</sup> Department of Radiation Oncology, University Duesseldorf, Germany, <sup>2</sup> Academic Medical Center, University of Amsterdam, The Netherlands, <sup>3</sup> Dept. of Radiation Oncology, Section Radiobiology, Free University, The Netherlands

**Introduction:** Experimental and clinical studies give evidence that clonogenic hypoxic tumour cells are an important factor of radioresistance. Hyperbaric oxygen (HBO) and normobaric carbogen (95% oxygen; 5% carbon dioxide) increase the oxygen delivery to tumours and were both directed against chronic hypoxia. This study was performed to explore the effects of HBO and carbogen on the the tumour oxygenation after multiple X-ray fractions and to detect differences between both modalities to modulate radiosensitivity when used only for the last part of the radiation treatment.

**Material and methods:** Experiments were performed on the rhabdomyosarcoma R1H transplanted subcutaneously in the flank of WAG/Rij rats. All animals were treated with 20 X-ray fractions of 2 Gy in 4 weeks. Measurements

of oxygen partial pressure ( $pO_2$ ) were performed using the Eppendorf oxygen electrode under ambient conditions and with normobaric carbogen and hyperbaric oxygen at a pressure of 240 kPa. A top-up dose of 10-50 Gy was applied in 2-10 fractions of 5 Gy within two weeks resulting in an overall treatment time of six weeks for the whole radiation experiment.

**Results:** Hyperbaric oxygen significantly increased the median  $pO_2$  of 122mmHg compared with 9mmHg and 6mmHg for the control group (0 Gy) and R1H-tumours after 20 fractions of 2 Gy, respectively. For HBO the difference of median  $pO_2$  readings for individual tumours was significantly different between the control (0 Gy) and tumours (40 Gy) irradiated under ambient conditions (control vs. 40 Gy-HBO:  $P=0.003$ ; 40 Gy air vs. 40 Gy-HBO:  $P=0.005$ ). The radiation doses to control 50% of the tumours ( $TD_{50}$ ) in the top-up experiment were 38.0Gy, 29.5Gy and 25.0Gy for air, carbogen and HBO, respectively. Oxygen enhancement ratios were 1.3 for normobaric carbogen and 1.5 for HBO. Dose response relations of the three groups were significantly different (Cochran-Mantel-Haenszel-Test: air vs. carbogen:  $P=0.44$ ; air vs. HBO:  $P=0.02$ ; carbogen vs. HBO:  $P=0.48$ ).

**Discussion:** These results suggest that HBO significantly improves the oxygenation status and the radiation response in a rodent tumour model after multiple X-ray fractions. With regard to both tumour oxygenation and radiation enhancement HBO was more effective than normobaric carbogen.

**Keywords:** HBO; carbogen; tumour oxygenation; polarographic oxygen electrode; radiation

#### A54 HYPERBARIC OXYGEN FOR THE TREATMENT OF IATROGENIC ARTERIAL GAS EMBOLISM: CASE REPORT. Michaela Ignatescu<sup>5</sup>, P Longobardi<sup>1</sup>, F. Sfogliarri<sup>1</sup>, F. De Pasquale<sup>2</sup>, S. Maitan<sup>2</sup>, C. Olivieri<sup>3</sup>, M. Baccanelli<sup>4</sup>

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**Background:** Iatrogenic gas embolism can occur during various medical procedures such as insertion of a central venous catheter, during cardiac operations or during extracorporeal circulation.

**Case report:** At the Hyperbaric Centre in Ravenna in July 2000 we treated a patient with gas embolism following an endoscopic pyeloneolithotomy operation to remove nephrolithiasis from the right kidney. During the operation the patient showed symptoms of air embolism and the procedure had to be interrupted. 8.5 hours later, the patient suffered a bilateral amaurosis preceded by vomiting and mild headache. A cerebral CT pointed out small hyperdense areas in the right subcortical frontal site with no evidence of cerebral oedema.

**HBO treatment:** 15 hours after the incident the patient started the compression treatment at our centre where we applied table CX30 which prescribes the respiration of first heliox and then oxygen for 7.02 hours at a maximum absolute pressure of 4.0 bar. The table was extended by two further cycles of 25 minutes of pure oxygen and 5 minutes of air at 2.2 and 1.9 bar respectively. At the end of the first hyperbaric treatment the patient reported a partial recovery of the visus with distinction of light and shadow.

The patient completed a series of ten treatments of hyperbaric therapy at 1.8 bar for 90 minutes over ten consecutive days. A complete resolution of the symptoms was observed after the fourth treatment with hyperbaric oxygen. A follow up at 6 months using other instrumental tests and clinical evaluations confirmed the improvement.

**Rationale:** We believe that during the surgery gas had entered the blood vascular system. We know that an amount of gas more than 0.3 cc/kg/min could bypass the lung filter and reach the cerebral vascular system. An endothelial damage due to the gas bubbles could lead to the harming of the hemato-encephalic barrier. From this results an oedema of the cerebral tissue, a reduced oxygen intake by the tissues as well as an ischemic damage of the cells.

We have preferred to use the table Cx 30 with heliox because it is known that  $O_2$  creates a gradient around the bubble especially in tissues with low oxygen consumption. Initially, the  $O_2$  entering the bubble will cause the expansion of the volume of the bubble instead of its reduction, determining an initial worsening of the symptoms. Helium distributes more efficiently than nitrogen and has a smaller tissue solubility, therefore displacing nitrogen in the bubble. The nitrogen is eliminated faster especially if it is located in the adipose tissue, in the cerebral and medullary substance or in the tendons.

**Conclusion:** Table CX30 when extended by 60 minutes applied even 15 hours after the acute event, with a further ten 90 minute treatments at 1.8 bar with daily frequency was effective in promoting the complete resolution of the complicated symptomatology.

#### A55 A PILOT STUDY OF THE USE OF HYPERBARIC OXYGEN THERAPY IN PATIENTS WITH FAECAL INCONTINENCE ASSOCIATED WITH PUDENDAL NERVE NEUROPATHY: EARLY RESULTS. Gerard Laden\*, J. Cundall, A. Gardiner, K. F. Chin, P. Grout\*, G. S. Duthie. Academic Surgical

Unit, Castle Hill Hospital, Cottingham, Humberside, \*Hyperbaric Medicine Unit, BUPA Hull and East Ridings, United Kingdom.

**Introduction:** Idiopathic faecal incontinence due to pudendal neuropathy has no adequate treatment. Multiple sclerosis patients with urinary and faecal incontinence have been treated with hyperbaric oxygen with mixed results. Initial improvements in function were followed by a reversal. We planned to see if the improvement could be sustained in patients with a chronic pudendal neuropathy.

**Method:** 7 patients (5 female and 1 male, age range 47-73) with chronic pudendal neuropathy and faecal incontinence were identified. They received 30 standard Marx treatments over 6 weeks. Pudendal latencies were performed consequentially throughout the treatment and 1 month after it had finished.

**Results:** There was a consistent improvement in the latencies seen in all of the patients at the end of the treatment which was maintained at 1 month follow up (see graph1). A visual analogue scale of global bowel symptoms has shown a mixed picture of both dramatic and no improvement.

**Discussion:** Early results suggest that hyperbaric oxygen may be of value in the treatment of pudendal neuropathy. The cause of this improvement at this time is unclear. If the results are maintained in further patients and at 6 month follow up then a randomised control trial must be performed.

#### **A56 HYPERBARIC OXYGEN THERAPY IMPROVES CLINICAL EFFECTS OF TRANSMYOCARDIAL LASER REVASCULARISATION AND CABG.** Jacek Kot<sup>1</sup>, Zdzislaw Sicko<sup>1</sup>, Lech Anisimowicz<sup>2</sup>, Mirosława Narkiewicz<sup>2</sup>, Wojciech Pawliszak<sup>2</sup>, Piotr Betlejewski<sup>2</sup>

<sup>1</sup>National Center of Hyperbaric Medicine, Institute of Maritime and Tropical Medicine, Gdynia, Poland,

<sup>2</sup>Department of Cardiac Surgery, Institute of Cardiology, Medical University of Gdansk, Poland

**Objective:** The stimulatory effect of the HBO therapy on the fibroblast growth and neo-angiogenesis is well documented, especially concerning promotion of ischemic wounds and bone-fractures healing. The similar mechanism is postulated for the transmyocardial laser revascularisation (TMLR) which involves the creation of channels in the myocardium with a laser to relieve angina. The main objective of this study was to verify the additive or synergistic effect of both methods applied to patients with refractory angina.

**Methods:** The prospective, randomized, open-label study with one-year follow-up was conducted. Sixty eight patients after CABG-TMLR operations were randomly assigned to receive the HBO therapy (HBO group, 10 HBO sessions once-a-day) or standard recovery protocol (NON-HBO group, without HBO therapy). The results of TTE, SPECT, stress-test, angina CCS classification, and quality of life based on the Seattle Angina Questionnaire (SAQ) were collected before operation and after 3, 6, and 12 months after the CABG-TMLR. The comparison between groups and between time points was performed using the repeated-measures ANOVA test after logarithmic transformation of parameters.

**Results:** There was statistically significant improvement of the quality of life (namely of the physical limitation, angina stability, and disease perception in the SAQ), angina CCS class and results of stress-tests in time in both groups. This improvement of the clinical status was significantly higher in the HBO group. However, ejection fractions and wall motion score indexes in TTE measurements did not show statistically significant differences between both groups, regardless of significant improvement over the time course. The results of SPECT studies did not differ between groups with the exception of the reduction of the number of hypoperfused segments which was significantly higher in the HBO group.

**Conclusion:** During the one-year follow-up there was improvement of the clinical status of patients after TMLR-CABG operations, and this improvement was more expressed in the HBO group.

#### **THE ROLE OF HBO-THERAPY IN THE TREATMENT OF OSTEOMYELITIS OF THE JAW. A RETROSPECTIVE ANALYSIS OF PATIENTS TREATED AT THE DEPARTMENT OF CRANIO-MAXILLOFACIAL SURGERY IN ZURICH (1988-2000).** Marc Baltensperger, Olivier Keller, Ahmet Gönen, Klaus Grätz, Jörg Schmutz. Switzerland

**Introduction:** Hyperbaric oxygen therapy (HBO) is an important part of the therapeutic armamentarium used in maxillofacial surgery to deal with osteoradionecrosis (ORN), improvement of surgical reconstruction's with critical vascularization and osteomyelitis. While the use of HBO is well established for treatment of ORN and irradiated tissue, it's role in osteomyelitis therapy is to date still insufficiently documented and controversial. The purpose of this study is to evaluate the role of HBO in our patient data.

**Materials and methods:** Records from patients with osteomyelitis treated with adjunctive HBO-therapy were reviewed from 1988-2000. Patient with less than 20 HBO sessions were from the study. Only patients with acute osteomyelitis (AO) and secondary chronic osteomyelitis (SCO) were included. Due to the very controversial results of HBO and other therapeutic modalities in primary chronic osteomyelitis (PCO), these patients were excluded. 43 patients met these criteria and showed sufficient documentation. A control group with an equal number of patients

(43) with AO and SCO, treated in the same time period without adjunctive HBO-therapy was randomly picked. The 86 cases consisted of 60 male and 26 female patients with a mean age of 45.6 years (range 6-89 years), the mean follow up time was 1.62 years (range 0.25 - 9 years). Both groups demonstrated homogenous demographic features with exception of their medical history. In the group with HBO-therapy there were significantly more patients with a history of alcoholism and cigarette-smoking (18 respectively 25 patients) compared to the group without HBO-therapy (12 respectively 17 patients). Treatment modalities were reviewed regarding surgery and HBO-therapy. Surgical procedures were divided into two groups, procedures involving major bone debridement (e.g. decortication, resection or extensive local revision) and minor procedures (incision and drainage and removal of the dental focus). One HBO session consisted of 2x45min at 2.0 atm pressure. The outcome of therapy was evaluated regarding the symptoms at the end of the follow-up period. Outcome with preoperative, postoperative and combined (at least 10 sessions HBO pre- and postop.) HBO-Therapy was analyzed. Antibiotic treatment was administered in all 86 cases and considered a relatively constant parameter.

**Results:** 43 patients received 25.4 HBO sessions in average (range 20-52). 7 patients received preoperative, 19 postoperative and 8 pre- and postoperative HBO-therapy. Of the patients without adjuvant HBO-therapy 36 (84%) underwent surgical procedures involving gross bone debridement, while only 28 patients receiving HBO-therapy (65%) needed such surgery, whereas in the HBO group 12 patients (28%) received minor surgical procedures compared to only 5 patients (14%) in the group without HBO-therapy. The mean number of major surgical procedures in the HBO group was 1.36 (range 1-4) and higher compared to group without HBO-therapy (mean 1.17; range 1-3). 28 patients (65%) with HBO-therapy compared to 31 (72%) without HBO were completely free of symptoms at the end of the follow up period. 10 patients (23%) with, and 8 patients (19%) without HBO therapy had minor residual symptoms, only in 5 (12%), respectively 4 patients (9%) the symptoms remained unchanged. None of the patients experienced a worsening of symptoms.

4 patients (57%) with preoperative HBO (mean 28.3 sessions; range 20-39), 19 patients (68%) with postoperative (mean 25.1 sessions, range 19-52) and 5 patients (62.5%) with combined HBO therapy (mean 11 sessions preop., range 11-15; mean 14.8 sessions postop., range 10-30) were considered free of symptoms at the end of the follow-up period. In 3 patients (43%) with preoperative HBO, 4 patients (14%) with postoperative HBO and 3 patients (37.5%) with combined HBO treatment a significant reduction of symptoms was observed. In 5 patients (18%) with postoperative HBO no change of symptoms was observed.

**Conclusion:** The outcome of patients treated with adjunctive HBO-therapy aside from antibiotics and surgery was slightly worse than in patients without HBO-therapy. This may be explained by the fact that patients with a history of alcoholism and cigarette-smoking often show a worse compliance to therapy. Further the effects of cigarette-smoking are clearly documented to antagonize the effects of HBO-therapy to a certain extent. Patients who received adjunctive HBO-therapy experienced significantly less major surgical procedures than patients without HBO-therapy, whereas more minor procedures were performed in the former group. This result is consistent with our experience, that in cases with limited extent of the disease, local rinsing, antibiotics and HBO-therapy are sufficient, hence avoiding decortication or resection. Combined HBO-therapy pre- and postoperative seems to show the best outcome, whereas postoperative HBO-therapy is least beneficial. However the number of cases examined is too small to be conclusive on this question. Prospective studies on a big population (multi center trials) are necessary to collect more accurate data.

## REMINDER

All members who have not yet paid their membership dues for this year are reminded to do so instantly. The Society needs the funds to finance her responsibilities!

You are also reminded to report any changes in your address to the Membership Secretary and to the Newsletter Editor. Otherwise we can not guarantee that the Journal and the correspondence from the Society's secretariat do reach you in time.

**Thank you very much for your co-operation.**



# MEETING ANNOUNCEMENTS

## WORLD CONGRESS OF DROWNING, 2002 BREATH-HOLD, SCUBA AND HOSE DIVING Amsterdam on 26, 27 and 28 June 2002

Recreational scuba diving is recognised as a safe sporting activity. There are relatively few accidents compared with other sports although, when an accident does occur in the water, it happens in a very unforgiving environment. What might be an insignificant incident at the surface can start a sequence of events that quickly escalates to become life-threatening. The environment in which this happens is also the probable reason why up to some 60 per cent of in-water diving fatalities meet their deaths by drowning. Drowning is the *mode* of their deaths, but not the cause. In examining the *causes* of drowning in divers, one must look at the way in which people dive. To reduce the risk of drowning in divers one must address not only their in-water procedures but also basic issues such as fitness, training and equipment.

For this reason the diving community has been invited to participate in the **World Congress of Drowning** to be held in Amsterdam on 26, 27 and 28 June 2002. This Congress was initiated by *The Society to Rescue Persons from Drowning* that was founded there in 1767. Partners in this venture include the International Federation of Red Cross and Red Crescent, ILS (International Life Saving) and DAN.

The aims of the Congress are

- to make recommendations on the prevention, rescue and treatment of drowning victims;
- to stimulate and facilitate initiatives to further promote the prevention of drowning;
- to reduce the number of drowning victims;
- to improve the survival rate and outcome of drowning victims.

"Breath-hold, scuba and hose diving" is thus just one of around 10 task forces convened to review particular aspects of this vast topic. Other task forces (and Chairpersons) include

- Epidemiology (Christine Branche, CDC, Atlanta)
- Rescue from the water (Chris Brewster, International Life Saving Federation, USA)

- Resuscitation (Paul Pepe, Emergency Medicine, University of Texas)
- Hospital treatment I (Jean Louis Vincent, Erasmus Hospital, Brussels)
- Immersion hypothermia (Beat Walpoth, University Hospital, Insel, Switzerland)
- Brain (David Warner, Duke University Medical Center, USA)

The diving task force covers the hazards associated with all types of diving. This includes recreational diving of every variety. It also covers subsistence fishermen-divers in the third world divers, most of whom have inadequate equipment and no proper training and who have an unknown rate of in-water incidents. The other large group is military and working divers who follow procedures that for them should make the risk of drowning negligible.

A number of drowning fatalities in divers occurs among divers who may have made an avoidable error or who may have been subjected to one. After reviewing such accidents the task force has prepared draft recommendations and reviewed those submitted by others. The following topics are among the questions that they consider deserve discussion at the World Congress.

Should diver certification last a lifetime, or is there a need for re-certification after a few years? What changes can be recommended in the training of divers and diving instructors that might enhance diving safety? Should a once-only medical declaration that was made before training potentially last for a lifetime? Is there a minimum age for diving as one of a buddy-pair? Should there be a greater emphasis at all levels of recreational diver training on the causation of known in-water fatalities?

Visit the web site for more details about the Congress, its task forces and the arrangements. Some 60 task force members from 20 nations have prepared formal presentations and reviewed the many recommendations for the Congress. Each

task force has a summary of its proposed agenda, each will have a plenary session for all and then a number of sessions on selected diving topics. So look through the recommendations in the diving section. Because they come from a wide range of sources, some appear worthwhile but others may not be universally acceptable. These will be discussed and, where appropriate, their implementation will be reviewed at the Congress in Amsterdam, 2002.

You can also write for more information to the

**World Congress of Diving 2002 Secretariat**  
**c/o Consumer Safety Institute**  
**PO Box 75169**  
**1070 AD Amsterdam**  
**The Netherlands**  
**or e-mail <Secretariat@drowning.nl>**

## **THE MEDICAL ASSESSMENT OF FITNESS FOR RECREATIONAL AND WORKING DIVERS**

**The Sheraton Airport Hotel, Amsterdam**

**1<sup>st</sup> & 2<sup>nd</sup> December, 2001**

Last year's meeting "Medical Assessment of Fitness to Dive" at The Royal Society of Medicine, London was run by Biomedical Seminars in association with the Medical Subcommittee of the European Diving Technology Committee. A request was made there that Biomedical Seminars run a course in this series on mainland Europe. This year the meeting will be held at the 5-star Sheraton Hotel in Schiphol Airport. The hotel is just 5 minutes by covered walkway from the Amsterdam airport terminal that also has its own station with frequent international trains, so this should be an easy place for everyone to reach.

The programme will follow the format of previous years with one or more special topics in the spotlight. Among those joining Dr McIver and Prof Elliott as speakers will be Dr Feenstra (otolaryngology); Dr van Hulst (PFTs); Dr Sterk (on occupational problems of tunnellers and divers); Dr Wendling (EDTC proposed harmonisation of standards for working divers); Dr Mueller (on the German-language meeting on diver fitness). In addition to a general review of some clinical decisions relating to diver fitness in the UK, recent policy changes in the HSE approval of doctors living in Europe but outside the UK and more so for those doctors living outside Europe will be explained by Dr Williams (HSE). The role of the International Marine Contractors Association (IMCA) will be reviewed. The newly revised RSTC medical guidance on recreational diver fitness reveals other issues that also deserve discussion in this context.

The 2-day registration fee is being maintained at last year's UK pounds 210 (Nfl 760) for early registrants. This includes lunch on Saturday and Sunday, and tea, coffee and biscuits throughout each day. From 1<sup>st</sup> October the fees will become UK pounds 230 (Nfl 830) and non-cancellable. Cheques made payable to Biomedical Seminars in UK pounds or Netherlands guilders are acceptable but we regret that we cannot accept credit card payment. A special weekend rate of around UK pounds 90 per night (approx Nfl 300) will be offered by the Sheraton to registrants quoting 'Biomedical Seminars'. Full details are available from Karen Reeves, Biomedical Seminars, 7 Lyncroft Gardens, Ewell, Surrey, England KT17 1UR. Fax (+44)208 7867036. Email: Karen@biomedseminars.demon.co.uk

REGISTRATION for "The Medical Assessment of Fitness for Recreational and Working Divers" at The Sheraton Airport Hotel, Amsterdam, 1st & 2nd December 2001. I enclose a cheque for UK pounds 210 (or Nfl 760) made payable to Biomedical Seminars.

**Name**

**Address**

**E-mail address**

**Mail to:**

Mrs Reeves, Biomedical Seminars, 7 Lyncroft Gardens, Ewell, Surrey, England KT17 1UR

# INSTRUCTIONS TO AUTHORS

The **Europ J Underw Hyperb Med** welcomes contributions (including letters to the Editor) on all aspects of diving and of hyperbaric medicine. Manuscripts must be offered exclusively to the **Europ J Underw Hyperb Med**, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review, with feedback to the authors. Accepted contributions will be subject to editing.

Manuscripts are accepted in English, and also in major European languages (French, Spanish, Italian and German) when accompanied by an English abstract and a letter of recommendation of a member of the **International Editorial Board**.

## Contributions should be sent to

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## Requirements for Manuscripts

The **Europ J Underw Hyperb Med** is composed on a PC using Word processing. Documents are acceptable on disc or by e-mail. Illustrations and tables should **NOT** be embedded in the Word document, only their position indicated. All tables are to be separate documents. Illustrations should be separate documents in Word or TIFF, clearly marked with the format used. **References should be in the correct format, shown in the next column.** Submissions must be accompanied by two printed copies of all text, tables and illustrations.

The printed copies should be double-spaced, using both upper and lower case, on one side of the paper only, on A4 paper. Headings should conform to the format in the **Journal**. All pages should be numbered. No part of the text should be underlined. These requirements also apply to the abstract, references, and legends to figures. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included. All tables should be double spaced on separate sheets of paper. **No vertical or horizontal rules are to be used.**

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Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, e.g. decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is 2,500 words or less. Inclusion of more than 5 authors requires justification. Original articles should include a title page, given the title of the paper and the first names and surnames of the authors, an abstract of no more than 200 words and except in unusual situations be subdivided into Introduction, Methods, Results, Discussion and References. After the references the authors should provide their initials and surnames, their qualifications, and the positions held when doing the work being reported. One author should be identified as Correspondent for the Editor and for readers of the Journal. The full current postal address of each author, with the Telephone, facsimile numbers and e-mail address of the corresponding author, should be supplied with the contribution. No more than 20 references per major article will be

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Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to 5 per letter).

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1. Thorsen E, Risberg J, Segadal K, Hope A. Effects of venous gas microemboli on pulmonary gas transfer function. *Undersea Hyperbaric Med* 1995; 22:347-353.
2. Hempleman HV. History of decompression procedures. In: Bennett PB, Elliott EH, eds. *The physiology and medicine of diving*. London: WB Saunders, 1993:324-375.
3. Kindwall EP, Goldmann RW. *Hyperbaric medicine procedures*. Milwaukee, WI: St. Luke's Medical Center, 1970.

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