# REUNION 2013

TRICONTINENTAL SCIENTIFIC MEETING ON DIVING AND HYPERBARIC MEDICINE

22-29 SEPTEMBER 2013

ORGANISED BY EUBS - SPUMS - SAUHMA - ARESUB

# Abstract & Conference Book

39th Annual Meeting of the European Underwater and Baromedical Society (EUBS)
42nd Annual Scientific Meeting of the South Pacific Underwater Medical Society (SPUMS)
Conference of the Southern African Underwater and Hyperbaric Medical Association (SAUHMA)
3ième Journée de médecine de plongée de l'Océan Indien (ARESUB)


**Editors:**
M Bennett
M Davis
JJ Brandt Corstius
P Germonpré


Congress Secretariat, Organisation and Logistic Support coordinated by the Scott Haldane Foundation





# REUNION2013
## Tricontinental Scientific Meeting on Diving and Hyperbaric Medicine

Organised by EUBS, SPUMS, SAUHMA and ARESUB
www.reunion2013.org

*Tamarun Conference Centre, La Saline-les-Bains*
*St.Gilles, Reunion Island, Indian Ocean*
*September 22–29, 2013*


**Organising Committee**
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# Conference Week Overview

| TIME | Sunday 22nd | | | Monday 23rd | | | Tuesday 24th | | Wednesday 25th | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Conference | Diving | Other | Conference | Diving | Other | Conference | Other | Conference | Diving | Other |
| 08:00 to 08:30 | | | | | | | **Registration** | **Registration** | | | |
| 08:30 to 08:40 | | **2 dives (package)** | | | **2 dives (package)** | | Opening ceremony | | | **2 dives (package)** | |
| 08:40 to 09:00 | | | DAN HIRA Meeting (Private) | | | IDAN Meeting (Private) | Richard Fitzpatrick | open all morning | | | IDAN Meeting (Private) |
| 09:00 to 09:20 | | | | | | | | | | | |
| 09:20 to 09:40 | | **Port of St.Gilles** | | | **Port of St.Gilles** | | Session 1 Diving Medicine | | | **Port of St.Gilles** | |
| 09:40 to 10:00 | | | | | | | | | | | (until 15:20) |
| 10:00 to 10:20 | | | | | | | | | | | |
| 10:20 to 10:40 | | | | | | | **COFFEE - TEA** | | | | |
| 10:40 to 11:00 | | | | | | **COFFEE - TEA** | Session 2 Hyperbaric Oxygen Therapy | | | | |
| 11:00 to 11:20 | | | | | | | | | | | |
| 11:20 to 11:40 | | | | | | | | | | | |
| 11:40 to 12:00 | | | | | | | | | | | |
| 12:00 to 12:20 | | | | | | | | | | | |
| 12:20 to 13:00 | | | | | | | **LUNCH** | | | | |
| 13:00 to 13:30 | | | | | | | | | | | |
| 13:30 to 14:00 | | | | **ARESUB** Scientific Meeting (in French) | | | Jacek Kot | | | | |
| 14:00 to 14:20 | | | | | | IDAN Meeting (Private) (continued) | Session 3 Diving Medicine | | | | EDTC Medical SubCommittee (Private) |
| 14:20 to 14:40 | | | | | | | | | | | |
| 14:40 to 15:00 | **ECHM** Workshop Diagnosis and treatment of mild DCS | | | | Society ExComs | | | | **Special Session 1** | | |
| 15:00 to 15:20 | | | | | | | | | | | **COFFEE - TEA** |
| 15:20 to 15:40 | | | | | | | | | Asthma and diving | | |
| 15:40 to 16:00 | | | | **COFFEE - TEA** | | | | | | | ECHM BR Meeting |
| 16:00 to 16:20 | | | | | | | **COFFEE - TEA** | | | | |
| 16:20 to 16:40 | | | | ARESUB (cont.) | | | Session 4 Diving Medicine | | | | |
| 16:40 to 17:00 | **COFFEE - TEA** | | | | | | | | | | |
| 17:00 to 17:20 | | | | | DHM Editorial Board Meeting | **Registration open** | | | **Visit of the Aquarium** | | |
| 17:20 to 17:40 | ECHM (cont.) | **Registration open** | | | | | | | | | |
| 17:40 to 18:00 | | | | | | | | | | | |
| 18:00 to 18:20 | | | | | | | | | | | |
| 18:20 to 18:40 | | | | | | | | | | | |
| 18:40 to 19:00 | | | | | | | | | | | |
| 19:00 to 19:30 | ECHM EB Meeting | | | | | **Welcome Reception** | **Happy hour at Tamarun** | | Richard Fitzpatrick at Aquarium | | |
| 19:30 to 20:00 | | | | | | | | | | | |
| 20:00 to 20:30 | | | | | | | | | | | |
| 20:30 tot 21:00 | | | | | | **location TBD** | | | **Drinks at the Aquarium** | | |
| 21:00 to 21:30 | | | | | | | | | | | |
| 21:30 to 22:00 | | | | | | | | | | | |
| 22:00 to 22:30 | | | | | | | | | | | |

| TIME | Thursday 26th | | Friday 27th | | | Saturday 28th | | Sunday 29th | |
|---|---|---|---|---|---|---|---|---|---|
| | Conference | Other | Conference | Diving | Other | Conference | Other | Conference | Diving |
| 08:00 to 08:30 | | | | 2 dives (package) | | | | | 2 dives (package) |
| 08:30 to 08:40 | | | | | | | | | |
| 08:40 to 09:00 | | | | | | | | | |
| 09:00 to 09:20 | Jacek Kot HBO | IDAN Instructors Meeting (Private) | | Port of St.Gilles | | SOCIETY AGMs | | DAN Divers Day | Port of St.Gilles |
| 09:20 to 09:40 | | | | | | | | | |
| 09:40 to 10:00 | Special Session 2 Controversies in | | | | | | | | |
| 10:00 to 10:20 | | | | | | Jacek Kot | | International DAN | |
| 10:20 to 10:40 | | | | | | Session 7 Hyperbaric Oxygen Therapy | | | |
| 10:40 to 11:00 | COFFEE - TEA | COFFEE - TEA | | | | | | | |
| 11:00 to 11:20 | Hyperbaric Medicine | | | | | | | | |
| 11:20 to 11:40 | | | | | | | | | |
| 11:40 to 12:00 | | | | | | | | COFFEE - TEA | |
| 12:00 to 12:20 | | | | | | | | | |
| 12:20 to 13:00 | LUNCH | LUNCH | | | | LUNCH | | | |
| 13:00 to 13:30 | | | | | | | | | |
| 13:30 to 14:00 | Richard Fitzpatrick | | | | | Session 8 Diving Medicine | | | |
| 14:00 to 14:20 | Session 5 Diving Medicine | | Special Session 3 PHYPODE Young investigators in diving research | | | | | | |
| 14:20 to 14:40 | | | | | | | | | |
| 14:40 to 15:00 | | | | | | | | | |
| 15:00 to 15:20 | | | | | | Closing ceremony | | | |
| 15:20 to 15:40 | | | COFFEE - TEA | | | Happy hour at Tamarun | | | |
| 15:40 to 16:00 | | | | | | | | | |
| 16:00 to 16:20 | COFFEE - TEA | | PHYPODE Special Session 3 Continued | | | | Young investigators in diving research PHYPODE Administrative meeting | | |
| 16:20 to 16:40 | Session 6 Hyperbaric Oxygen Therapy | | | | | | | | |
| 16:40 to 17:00 | | | | | | | | | |
| 17:00 to 17:20 | | | | | | | | | |
| 17:20 to 17:40 | | | | | | | | | |
| 17:40 to 18:00 | | | | | | | | | |
| 18:00 to 18:20 | | | | | | | | | |
| 18:20 to 18:40 | | | CONFERENCE DINNER at Hotel Lux* | | | | | | |
| 18:40 to 19:00 | | | | | | | | | |
| 19:00 to 19:30 | Happy hour at Tamarun | | | | | | | | |
| 19:30 to 20:00 | | | | | | | | | |
| 20:00 to 20:30 | | | | | | | | | |
| 20:30 tot 21:00 | | | | | | | | | |
| 21:00 to 21:30 | | | | | | | | | |
| 21:30 to 22:00 | | | | | | | | | |
| 22:00 to 22:30 | | | | | | | | | |

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# Websites

Conference Website: www.reunion2013.org
European Underwater and Baromedical Society: www.eubs.org
South Pacific Underwater Medicine Society: www.spums.org.au
Southern African Underwater and Hyperbaric Medical Association: www.sauhma.co.za
Association Réunionnaise de Médecine Subaquatique et Hyperbare: www.aresub.org
Scott Haldane Foundation: www.scotthaldane.org
Diving and Hyperbaric Medicine Journal: www.dhmjournal.com

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## Tricontinental Scientific Meeting on Diving and Hyperbaric Medicine

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*Tamarun Conference Centre, La Saline-les-Bains*
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## Scientific Programme

### Sunday, September 22nd, 2013

**ECHM Workshop**: "Diagnosis and Treatment of Mild Decompression Sickness"
*Location: "Le Voilier" room, Tamarun*

| | |
|---|---|
| 14:30 | **Introduction** |
| 14:40 | **Definition of mild DCS, clinical manifestations, differential diagnosis issues and the threshold between mild and serious DCS**<br>*Speaker: Simon Mitchell (New Zealand)* |
| 15:10 | Discussion |
| 15:20 | **Natural progress and evolution of DCS**<br>*Speaker: Nick Bird (USA)*<br>**A clinical perspective on delayed vs. early treatment & final outcome**<br>*Speaker: Jordi Desola (Spain)* |
| 15:50 | Discussion |
| 16:00 | **Telemedicine triage and decision-making issues - The issue of "remote locations", its impact on hyperbaric treatment delay, on-site treatment vs. Medevac risk-benefit evaluation and related decision making**<br>*Speakers: Jack Meintjes (South Africa), Ramiro Cali-Corleo (Malta)* |
| 16:30 | Discussion |
| 16:40 | **Break** |
| 17:10 | **Immediate non-hyperbaric treatment: what, when, how, by whom and the issue of in-water recompression**<br>*Speaker: Peter Germonpre (Belgium)*<br>*Discussants: Nick Bird, Jack Meintjes, Simon Mitchell* |
| 17:40 | Discussion |
| 17:50 | **Non-Hyperbaric medical treatment: pros, cons, possible complications and when to Medevac to HBOT facility**<br>*Speaker: Jack Meintjes (South Africa)*<br>*Discussants: Simon Mitchell, Peter Mueller* |
| 18:20 | Discussion |
| 18:30 | **Cost-benefit evaluation and possible liability implications of local non-HBTx vs. standard Medevac & HBOTx**<br>(Panel Discussion and Workshop Conclusions)<br>*Panel: Jacek Kot, Ramiro Cali-Corleo, Alessandro Marroni* |
| 19:10 | **Close of the Workshop** |

## Monday, September 23rd, 2013

**ARESUB Symposium : « 3° Journée de médecine de plongée de l'Océan Indien »**
*Location: "Le Voilier" room, Tamarun*
This symposium will be in French only.

| | |
|---|---|
| 13 :30 | **Accueil des participants** |
| 13 :45 | **Modification de bilan hydrique pendant la plongée - retentissement physiologique**<br>*Orateur : Pr J.Regnard (Besançon)* |
| 14 :20 | **Hématome épidural rachidien déclenché par une séance d'apnée**<br>C.D'Andrea, K.Doe, J-D.Harms<br>*Orateur : Dr C.D'Andrea (Réunion)* |
| 14 :35 | **Dégénérescence discale cervico-dorsale et risque d'accident de décompression médullaire chez le plongeur: étude cas-témoins**<br>E.Gempp, P.Louge, T.Lafolie, S.De maistre, M.Hugon, J-E.Blatteau<br>*Orateur : Dr E.Gempp (Toulon)* |
| 14 :55 | **Evaluation du stress oxydant et de l'état inflammatoire chez le plongeur présentant un ADD médullaire**<br>P.Louge, E.Gempp, J-M.Pontier, S.De Maistre, N.Vallée, J-G.Steinberg, J-E.Blatteau.<br>*Orateur : Dr P.Louge (Toulon)* |
| 15 :15 | **Historique des centres hyperbares dans l'Océan Indien**<br>J-D.Harms, E.Szalay-Bonnans, P.Durasnel<br>*Orateur : Dr J-D.Harms (Réunion)* |
| 15 :30 | **Prise en charge et bilan des accidents de décompression sur Mayotte**<br>*Orateur : Dr P.Durasnel (Mayotte)* |
| 15 :45 | **Pause Café** |
| 16 :15 | **La prise de Viagra avant plongée favorise l'accident de décompression chez le rat**<br>J-E.Blatteau, A-O.Brubakk, E.Gempp, O.Castagna, J-J.Risso, N.Vallée<br>*Orateur : Dr J-E.Blatteau (Toulon)* |
| 16 :35 | **Bulle intra-oculaire chez un plongeur porteur d'implants multifocaux**<br>V.Poncin, B.Grandjean, N.Alfonsi<br>*Oratrice : Dr V-Poncin (Dax)* |
| 16 :55 | **Pneumothorax spontané diagnostiqué tardivement au décours d'une plongée**<br>*Orateur : Dr E.Szalay-Bonnans (Ile Maurice)* |
| 17 :10 | **Intérêt des paliers profonds en plongée loisir à l'air**<br>*Orateur : Dr C.D'Andrea (Réunion)* |
| 17 :30 | **Identification des espèces de requins en fonction des morsures**<br>*Orateur : Dr F.Landron (Réunion)* |
| 17 :50 | **Clôture de la journée** |
| 18 :00 | **Départ** |

**19:00 Tricontinental Scientific Meeting - Welcome Reception**
*Location: Le Nautile Hotel, La Saline des Bains*

## Tuesday, September 24th, 2013

**Tricontinental Scientific Meeting, Day One**
*Location: "Le Voilier" room, Tamarun*

| | | | | |
|---|---|---|---|---|
| 08:30 | **Opening Ceremony** | | | |
| 08:40 | | **Keynote Lecture:** R Fitzpatrick : | TROPICAL REEF SHARKS | |
| | **Session 1: Diving Medicine** | | **Chair: Costantino Balestra** | |
| 09:20 | O-01 | P Louge | OXIDATIVE STRESS AND PROINFLAMMATORY INTERLEUKIN-6 RELEASE IN RECREATIONAL DIVERS WITH SPINAL CORD DECOMPRESSION SICKNESS | |
| 09:40 | O-02 | E Gempp | SERUM NEURON-SPECIFIC ENOLASE AND S100B PROTEIN LEVELS IN NEUROLOGICAL DECOMPRESSION SICKNESS: A CONTROLLED STUDY. | |
| 10:00 | O-03 | S Theunissen | DARK CHOCOLATE PREVENTS ENDOTHELIAL DYSFUNCTION AFTER A SCUBA DIVE | |
| 10:20 | **Coffee / Tea** | | | |
| | | | | |
| | **Session 2: Hyperbaric Oxygen Therapy** | | **Chair: Jack Meintjes** | |
| 10:40 | O-04 | D Lévigne | HYPERBARIC OXYGEN THERAPY: WHAT TYPE OF WOUND BENEFITS MOST? | |
| 11:00 | O-05 | P Baroni | ACCELERATED WOUND HEALING USING PLATELET GEL, SKIN GRAFT AND HYPERBARIC OXYGENATION | |
| 11:20 | O-06 | G Bosco | BIOLOGICAL EFFECTS OF A HYPERBARIC OXYGEN PRECONDITIONING (HBO-PC) IN PANCREATODUODENECTOMY: RESULTS OF A RANDOMIZED SINGLE-BLIND TRIAL IN HUMANS | |
| 11:40 | O-07 | M Bennett | THE TREATMENT OF NECROTISING FASCIITIS WITH HYPERBARIC OXYGEN THERAPY – A META-ANALYSIS OF OBSERVATIONAL DATA | |
| 12:00 | O-08 | A Anão | HYPERBARIC OXYGEN THERAPY IN ADVANCED PERIPHERAL ARTERIAL DISEASE – THERAPEUTIC EVALUATION IN CRITICAL LIMB ISCHEMIA | |
| 12:20 | **Lunch** | | | |

# O-01 OXIDATIVE STRESS AND PROINFLAMMATORY INTERLEUKIN-6 RELEASE IN RECREATIONAL DIVERS WITH SPINAL CORD DECOMPRESSION SICKNESS

P Louge[1], E Gempp[1], JM Pontier[2], S De Maistre[1], N Vallée[3], JG Steinberg[4], and JE Blatteau[3].

[1]Department of Diving and Hyperbaric Medicine, Ste Anne's Military Hospital, Toulon, France; [2]Department of diving medicine, French Navy Diving School, Toulon, France; [3]ERRSO, Institute of Biomedical Research of the Armed Forces Health Service, Toulon, France; [4]UMR MD2, Faculty of medicine, Marseille, France.

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**Introduction:** Oxidative stress and inflammation following hyperbaric exposure have been mainly reported in healthy divers or in experimental models of decompression sickness (DCS) but there are no data available in divers experiencing neurological DCS [1-4]. We aimed to evaluate the level of oxidative stress and inflammatory response in a cohort of injured divers, and compare these results with those obtained in otherwise control divers.

**Methods:** Thirty recreational divers (47 ± 11 years) referred for spinal cord DCS following scuba diving (40 ± 10 msw; 38 ± 10 min.) and 10 healthy military divers (34 ± 6 years) exposed to a routine air dive (30 msw; 41 min) were recruited. Blood measurements for thiobarbituric acid-reactive substances (TBARS), reduced ascorbic acid (RAA) and interleukin-6 (IL-6) were determined between 1-4 h post-dive. The initial severity of DCS was assessed with a clinical score proposed elsewhere [5].

**Results:** TBARS and RAA, known as indicators of oxidative stress and antioxidant activity respectively, were significantly elevated in DCS divers (Mann-Whitney test; $P = 0.0012$ and $P = 0.018$ respectively). Higher levels of IL-6 were also observed in injured divers compared with the control group (Mann-Whitney test; $P = 0.016$). We did not find significant changes between DCS divers with severe presentation (n = 13) and those with benign or mild symptoms (n = 17).

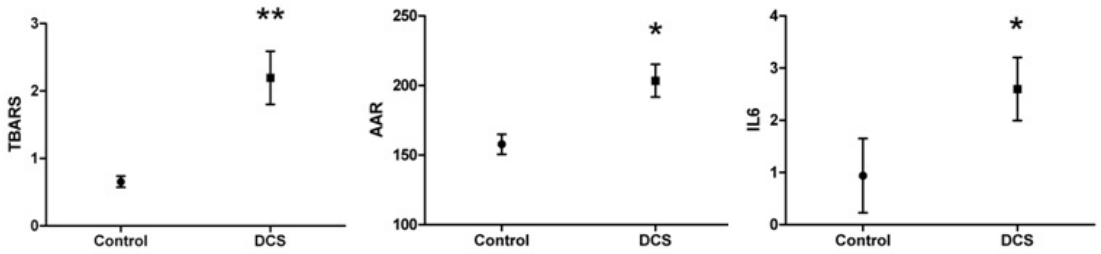


**Figure 1:** Mean values ± SEM of TBARS, RAA and IL-6 between controls and DCS divers. * denotes $p < 0.05$ and ** denotes $p < 0.01$

**Conclusion:** For the first time, our findings may provide evidence that there is a strong relationship between neurological DCS, increased level of oxidative stress and release of inflammatory mediators in scuba divers.

**References**

1 Ersson A, Walles M, Ohlsson K, et al. Chronic hyperbaric exposure activates proinflammatory mediators in humans. *J Appl Physiol* 2002; 92:2375-80.
2 Sureda A, Batle JM, Ferrer MD, et al. Scuba diving activates vascular antioxidant system. *Int J Sports Med* 2012; 33:531-36.
3 Bigley NJ, Perymon H, Bowman GC, et al. Inflammatory cytokines and cell adhesion molecules in a rat model of decompression sickness. *J Interferon Cytokine Res* 2008; 28:55-63.
4 Obad A, Marinovic J, Ljubkovic M, et al. Successive deep dives impair endothelial function and enhance oxidative stress in man. *Clin Physiol Funct Imaging* 2010; 30:432-38.
5 Blatteau JE, Gempp E, Simon O, et al. Prognostic factors of spinal cord decompression sickness in recreational diving: Retrospective and multicentric analysis of 279 cases. *Neurocrit Care* 2011; 15:120-27.

**Keywords:** Decompression sickness, cytokines, oxidative stress.

## O-02 SERUM NEURON-SPECIFIC ENOLASE AND S100B PROTEIN LEVELS IN NEUROLOGICAL DECOMPRESSION SICKNESS: A CONTROLLED STUDY.

E Gempp[1], P Louge[1], S De Maistre[1], and JE Blatteau[2].

[1]Department of Diving and Hyperbaric Medicine, Ste Anne's Military Hospital, Toulon, France; [2]ERRSO, Institute of Biomedical Research of the Armed Forces Health Service, Toulon, France.

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**Introduction:** Neuron-specific enolase (NSE) and S100B protein are proteins originating in the brain and commonly measured to assess the presence and severity of neurological damage after global cerebral ischemia, stroke and traumatic brain injury. To date, there are limited data examining the influence of scuba diving on these biomarkers [1-3], particularly when symptoms of decompression sickness (DCS) occur [4,5]. The purpose of this prospective observational study in a large cohort of divers was to determine whether serum NSE and S100B protein levels could be used as 1) indicators of neurological DCS and 2) predictors of incomplete recovery.

**Methods:** Fifty-nine divers with neurological DCS (46 ± 10 years; M/F, 45:14) and 37 asymptomatic divers (49 ± 12 years, M/F, 27:10) admitted for inadequate decompression who served as controls were consecutively enrolled between 2010 and 2012. Blood samples were collected at initial presentation up to 6 hours after dive completion (controls) or onset of symptoms (DCS divers). Biomarkers were quantified in non-hemolyzed samples only. Clinical outcome was assessed at 6 months post-injury.

**Results:** Both groups did not differ regarding age, sex, diving parameters and delay for blood collection. NSE, but not S100B protein, was higher in the DCS group than in controls (12.5 ± 4.3 µg/l vs. 8.8 ± 3.2 µg/l; $P < 0.0001$). An NSE level > 15.8 µg/l determined by ROC analysis predicted DCS development with a specificity of 100% (95%CI = 90-100) and a sensitivity of 24% (95%CI = 14-36). There was a trend towards a poorer outcome above this cutoff value (OR = 3.5, 95%CI = 0.99-12.3; $P = 0.08$).

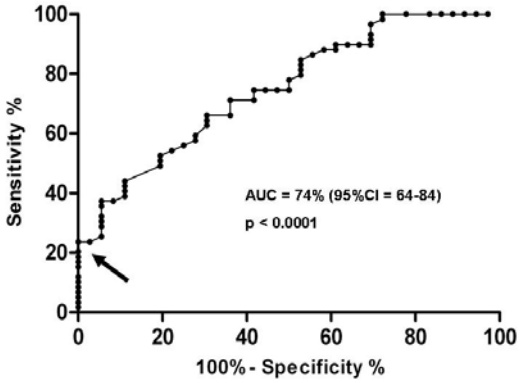


**Figure 1**. Receiver operating characteristics curve for NSE levels to predict neurological DCS development. Black arrow means cutoff value of 15.8 µg $l^{-1}$ for the highest specificity.

**Conclusion:** Early determination of NSE was found to be useful for the diagnosis of neurological DCS with a high specificity, but its prognostic value remains to be established. Reliability of S100B protein was not demonstrated in the present study.

**References**

1. Jurd KM, Parmer K, Seddon F, et al. Serum S100 and Neuron specific enolase: potential markers for cerebral events following decompression. *Undersea Hyperb Med* 1998; 25 (Suppl):40.
2. Stavrinou LC, Kalamatianos T, Stavrinou P, et al. Serum levels of S100B after recreational scuba diving. *Int J Sports Med* 2011; 32:912-15.
3. Bilopavlovic N, Marinovic J, Ljubkovic M, et al. Effect of repetitive SCUBA diving on humoral markers of endothelial and cerebral nervous system integrity. *Eur J Appl Physiol* 2013
4. Van Hulst RA, Klein J. Serum S100 in divers with neurological symptoms of decompression illness. *Undersea Hyperb Med* 2001; 28 (Suppl):51.
5. Poff DJ, Wong R, Bulsara M. Acute decompression illness and serum S100B levels: a prospective observational pilot study. *Undersea Hyperb Med* 2007; 34:359-67.

**Keywords:** Decompression sickness, diving**,** Neuron-specific enolase, S100B protein

## O-03 DARK CHOCOLATE PREVENTS ENDOTHELIAL DYSFUNCTION AFTER A SCUBA DIVE

S Theunissen[1,2], F Tillmans[1], J Schumacker[1], W Hemelryck[1], A Boutros[1], G Obeid[4], C Balestra[1,3], P Lafère[4], F Guerrero[2], and P Germonpré[4]

[1]Haute Ecole Paul-Henri Spaak, Environmental & Occupational Physiology Lab., Brussels, Belgium; [2]Université de Bretagne Occidentale, UFR Sciences et Techniques, 6 avenue Le Gorgeu, CS 93837, Brest; [3]DAN Europe Research, Brussels, Belgium; [4]Centre for Hyperbaric Oxygen Therapy, Military Hospital Queen Astrid, Brussels, Belgium.

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**Objectives:** The aim of the study is to observe the effects of ingestion of 30g of dark chocolate (86% cocoa) before a single scuba, dive knowing that flavonoïds contained in cocoa are able to improve endothelial function in a healty subject.

**Methods:** Forty-two volunteer male divers were divided in two groups. The chocolate group (21 divers) ate chocolate 2 h before the dive. All divers performed a dive to 33 meters for 20 minutes in a calm pool (NEMO 33 Brussels, Belgium). Water and air temperature were respectively 33°C and 29°C. Flow-mediated dilation (FMD), nitric oxide (NO) and peroxinitrites (ONOO-) were measured before and after the dive.

**Results:** A reduced FMD was observed in the control group whereas FMD was increased after ingestion of dark chocolate (respectively 90.87 +/- 6.7% of pre-dive values; $P < 0.001$ and 105.1 +/- 4.49% of pre-dive values; $P < 0.001$). No variation of NO was observed after the dive in controls (102.6 +/- 18.06% of pre dive values; $P > 0.05$) whereas an increase is found in the chocolate group (154.4 +/- 72.92% of pre-dive values; $P < 0.05$). A reduced ONOO- is observed in the control group (84.25 +/- 12.44% of pre dive values; $P < 0.05$) but no variation after ingestion of chocolate (100.1 +/- 27.83% of pre dive values; $P > 0.05$).

**Conclusion:** Endothelial dysfunction associated to diving seems to be prevented by ingestion of a moderate quantity of dark chocolate. Since hyperoxia reduces tetrahydrobiopterin (BH4) level (Fismen et al. 2013) endothelial nitric oxide synthase (eNOS) is down-regulated, leading to a decreased FMD. With addition of antioxidants, BH4 levels are higher allowing an activation of eNOS to produce NO; FMD is then increased.

**References:**

1. L Fismen, T Eide, A Hjelde, AM Svardal, R Djurhuus. Hyperoxia but not ambient pressure decreases tetrahydrobiopterin level without affecting the enzymatic capability of nitric oxide synthase in human endothelial cells. *Eur J Appl Physiol* 2013 113: 1695-704

**Keywords:** Flavonoids, oxidative stress, tetrahydrobiopterin, flow-mediated dilation, nitric oxide

The study is part of the Phypode Project, financed by the European Union under a Marie Curie Initial Training Network programme.

## O-04 HYPERBARIC OXYGEN THERAPY: WHAT TYPE OF WOUND BENEFITS MOST?

D Lévigne[1], A Modarressi[1], R Pignel[2], F Atashi[1], and B Pittet-Cuénod[1]

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**Introduction:** Chronic wounds constitute a growing health problem, predominantly due to the spreading pandemic of diabetes mellitus, and their treatment is still inconsistent and empirical. Hyperbaric oxygen therapy (HBOT) is a promising method to improve wound healing but there is still a lack of understanding of its exact mechanisms of action and its indications are yet to be clearly defined. We aimed to study the effects of HBOT in different wound conditions using an animal model.

**Materials and Methods:** Four different wound conditions (n = 20 per group) were studied: non-ischemic or ischemic wounds in normoglycemic or streptozotocine-induced hyperglycemic rats. Bilateral wounds were inflicted on the dorsal aspect of the hind foot. To create an ischemic condition, the left femoral artery of all animals was resected. Forty animals were treated with hyperbaric oxygen at 254 kPa for 95 minutes, five times a week until complete wound closure. Wound healing in these animals was compared to 40 rats receiving a standard semi-occlusive dressing. Wounds were assessed until complete wound closure by macroscopic planimetry and digital photography.

**Results:** Non-ischemic wounds in normoglycemic rats showed no significant benefit from HBOT. In contrast, wounds in ischemic or hyperglycemic conditions closed faster when treated with HBOT. Ischemic wounds in hyperglycemic animals benefited most from HBOT, these wounds closed significantly faster (29.2 days, SD 5.45) compared to standard wound dressing (43.5 days, SD 6.72, $P < 0.01$).

**Conclusion:** We conclude that HBOT constitutes a promising therapeutic approach for wounds in the context of diabetes and/or ischemia while simple non-ischemic wounds are less likely to benefit from it.

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**Keywords:** Hyperbaric oxygen therapy, wound healing, ischemic ulcer, diabetic ulcer, diabetes mellitus

## O-05 ACCELERATED WOUND HEALING USING PLATELET GEL, SKIN GRAFT AND HYPERBARIC OXYGENATION.

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**Objectives**: To verify if homologous platelet gel, autologous graft and HBOT allows 40% reduction of the wound after 4 weeks from inclusion and healing in 60 days. In PubMed only one publication could be found [1].

**Methods**: Nine patients suffering from arterial insufficiency and venous stasis lower limbs wounds (from > 6 weeks; size from 5 $cm^2$ to the whole leg circumference; Falanga score ≥ B2) were treated with: mechanical ultrasound debridement (at inclusion); homologous platelet gel; autograft (at the fourth week); HBOT [2]. Platelet gel was prepared following a validated method and activated, at clinical use, in 1:1 ratio with gluconate calcium and thrombin on a support of modulating matrix proteases (55% collagen, 45% oxidized regenerated cellulose). The final product, so gelled, was topically applied once a week for 4 weeks. Autograft was soaked for 5 minutes in Platelet Rich Plasma (PRP) and applied to the wound bed (pre-treated with PRP).

**Results**: Six wounds healed after 60 days from the inclusion and the area of the other 3 wounds was reduced by 72% (Falanga score A2). There were no side effects (Fig.1).

**Discusssion**: The delayed wound healing is due to high levels proinflammatory cytokines TNF-α and IL-1; alteration of the microcirculation; elevated levels of proteolytic elastase and protease; reduced synthesis of extracellular matrix (hyaluronic acid, fibrin, fibronectin); inhibition of growth factors (GF). PRP is known to enhance wound healing and survival rate of skin grafts [3]. It contains antibacterial peptides and a high number of platelets which, activated, release over 300 proteins, including GFs. PDGF and TGF-β stimulates proliferation of fibroblasts and production of new extracellular matrix. HBOT induces platelet activation and protein release [4]; stimulates vasculogenic stem cell mobilization from bone marrow [5] by stimulating nitric oxide synthase (NOS) in patients' platelets (NO remains elevated for 20 hours after HBOT).

**Conclusion**: Our experience suggests clinical efficacy of Platelet gel; autograft and HBOT in accelerating problem vascular wound repair.

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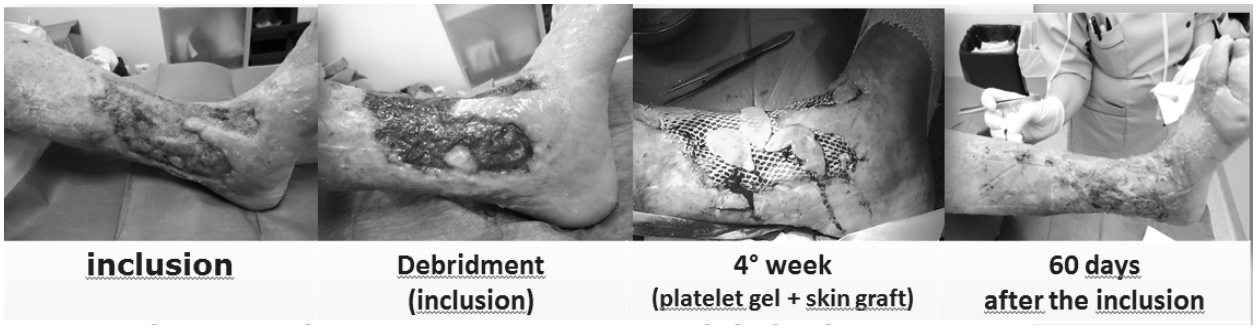


**Fig 1.** N.G., female, aged 85, mixed vascular ulcers (arterial and venous). Impairment Factor: results of radiation therapy for melanoma left lower limb. Therapy: platelet gel counterpart, autograft, compression therapy, hyperbaric oxygen therapy (20 sessions).

**Keywords:** Hyperbaric oxygenation, wound repair, platelet rich plasma, platelet gel, skin graft

## O-06 BIOLOGICAL EFFECTS OF HYPERBARIC OXYGEN PRECONDITIONING (HBO-PC) IN PANCREATODUODENECTOMY: RESULTS OF A RANDOMIZED SINGLE-BLIND TRIAL IN HUMANS.

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**Introduction:** In abdominal surgery, pancreaticoduodenectomy (PD) represents the intervention mostly burdened by various local and systemic complications (30 ± 60%). At the present time the adjuvant role of hyperbaric oxygen therapy (HBOT) in human surgery has been demonstrated only in cardiovascular or orthopaedic surgery and in liver transplantation.

In our study we evaluated post-operative biological and clinical effects of a preoperative (HBOT-PC) approach in patients undergoing surgery for a pancreatic ductal adenocarcinoma.

**Methods:** The study was a prospective, randomized single-blind study lasting 12 months. Twenty-one patients were randomized to two groups: 10 to a HBOT-PC group and 11 to a placebo group. The HBOT-PC group received HBOT (3 x 24 minutes of 100% $O_2$ via a tight-fitting oro-facial mask with 2 x 5-minute air breaks) at 2.5 ATA the day before PD intervention, whilst the placebo group patients breathed air for ≥40 minutes in a hyperbaric chamber pressurized to 115 kPa. In all patients, blood samples were taken before HBOT treatment or placebo-procedure ($T_0$), at the end of HBOT session or placebo-procedure ($T_1$), on first post-operative day (pod) ($T_2$) and on seventh pod ($T_3$). We measured interleukin (IL)-1,IL-6,IL-8,IL-10,IL-12p70 and TNF-α. For outcome evaluation, we considered the presence of postoperative pancreatic fistula (POPF), biliary fistula, fever, intra-abdominal abscess, bleeding, pulmonary complications, delayed gastric emptying and post-operative antibiotic needs.

**Results:** Patients undergoing HBOT showed a reduction in postoperative pulmonary infections vs. Placebo ($P = 0.023$). HBOT-PC modulates IL-6 synthesis ($P = 0.009$) and IL-10 one ($P = 0.03$), and does not cause more bleeding compared to placebo ($P = 0.450$). A statistically significant relationship between IL-6 and biliary fistula was seen ($P = 0.009$). IL-1 ($P = 0.006$) and TNF-α ($P = 0.04$) were correlated with hyperpyrexia and TNF-α appeared to be related to POPF ($P = 0.019$).

**Conclusion:** Further studies have to investigate how factors such as pressure, frequency, or lag time could be better tailored to the different major surgery procedures. HBOT-PC is safe in well-assessed candidates. They require a careful pre-HBOT clinical evaluation to avoid HBOT contraindications.

**Keywords:** Hyperbaric oxygen, preconditioning, surgery, pancreatoduodenectomy

## O-07 THE TREATMENT OF NECROTISING FASCIITIS WITH HYPERBARIC OXYGEN THERAPY – A META-ANALYSIS OF OBSERVATIONAL DATA

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**Introduction:** Hyperbaric oxygen therapy (HBOT) has been used in the treatment of necrotising infections since the 1960s [1], but the adjunctive treatment of necrotising fasciitis (NF) with HBOT remains controversial [2]. We have undertaken a meta-analysis of the comparative trials in this area in order to improve our understanding of the best available evidence.

**Methods:** In January 2013 we searched multiple electronic databases using a wide variety of terms used to describe NF. We included all comparative trials that reported the outcome of NF with and without HBOT. Our primary outcome was case fatality during the presenting admission. We also collected data on other clinical outcomes and cost when available. Data were analysed using RevMan 5.2 (Cochrane 2012) using odds ratio (OR) to measure treatment effect.

**Results:** Combining all searches yielded a total of 11 clinical reports for inclusion in this review (Fig 1). These reports included data on 208 patients receiving HBOT and 357 control (total 565). Seven of the 11 reports included patients receiving treatment over the same time period, while the other four were historically controlled.

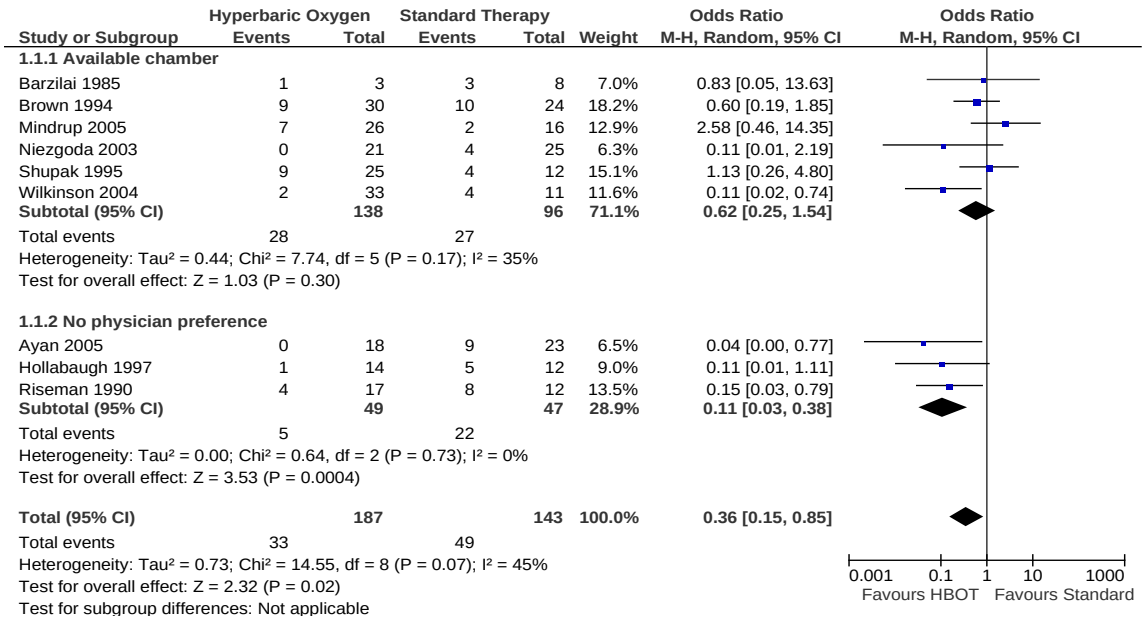


Figure 1. Forest plot for death with and without HBOT.

35 (16.8%) of cases treated with HBOT died, versus 75 (21%) receiving standard care. The OR of dying with HBOT was 0.42, (95%CI 0.20 to 0.89), P = 0.02. This analysis suggests that we might expect to treat 24 patients with NF in order to prevent one death.

**Discussion:** Our analysis suggests a benefit from the use of HBOT for the treatment of NF. These data represent the best clinical evidence available, but the overall estimate of effect should be interpreted with caution. Because RCTs may be impossible, the best course may be to establish a national or multinational prospective cohort including centres both with and without adjunctive HBOT. We urge the establishment of such a study.

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## O-08 HYPERBARIC OXYGEN THERAPY IN ADVANCED PERIPHERAL ARTERIAL DISEASE – THERAPEUTIC EVALUATION IN CRITICAL LIMB ISCHEMIA

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**Introduction:** Hyperbaric oxygen (HBO) is frequently used as complementary therapy for advanced degrees of peripheral artery disease (PAD), usually in critical limb ischemia with difficult healing ulcers and/or associated gangrene. Multiple different processes have been proposed for its healing action, such as local anti-hypoxic effects, microcirculatory regulation, neo-angiogenesis, infection limitation and oedema reduction.

**Objective:** Evaluation of therapeutic efficacy of HBO in advanced PAD and its characterization in a national reference centre.

**Methods:** We retrospectively included all patients (pts) evaluated between 2005 and 2011 with PAD, with the exclusion of cases attributed to diabetic neuropathy or venous aetiology. The following variables were evaluated: demographic data, risk factors, type and severity of lesion(s), revascularization and/or amputation history, duration of the clinical presentation (> or < 3 months), number of HBO sessions, clinical evolution until the end of HBO (grade I: worsening, II: no apparent benefit, III: mild improvement, IV: significant improvement), dropout pts and complications during HBO.

**Results:** Total number of pts referred with PAD: 98 (24 females), Leriche-Fontaine class: IV = 84, III = 14. Proposed to HBO: 87 pts; age 64.6 ± 12.3; Risk factors: Type I diabetes mellitus (DM): 11 (12.6%), Type II DM 31 (35.6%); arterial hypertension 57 (64.0%); smoking 26 (29.8%), active smoking: 12 (13.7%); dyslipidemia 39 (44.8%). Sixty-one pts (62.2%) initiated HBO, average number of HBO sessions: 49.2 ± 33; dropout pts during HBO: 12 (19.6%), with 3 cases of minor complications (4.9%). Clinical evolution in treated patients ($\chi^2$, $P$ = 0.106): Grades: I – 5 (10.2%), II – 15 (30.6%), III – 13 (26.5%), IV – 16 (32.6%). The presence of recent amputation or revascularization (< 3 months) was associated with better clinical outcome ($\chi^2$, $P$ = 0.0102).

**Conclusions:** The majority of pts referred with PAD presented with severe ischemic limb lesions with ulcer or distal gangrene (Leriche-Fontaine grade IV). HBO therapy was associated with variable results, with the best outcomes presenting in cases of recent amputation or revascularization as complementary treatment.

**Keywords:** Peripheral artery disease, Leriche-Fontaine classification, hyperbaric oxygen therapy

## Tuesday, September 24th, 2013

**Tricontinental Scientific Meeting, Day One (continued)**
*Location: "Le Voilier" room, Tamarun*

| | | | | |
|---|---|---|---|---|
| 13:30 | | **Keynote Lecture:** | | |
| | | J Kot | SATURATION DECOMPRESSIONS – IT'S OXYGEN THAT DRIVES, NOT INERT GAS | |
| | **Session 3: Diving Medicine** | | **Chair: Martin Sayer** | |
| 14:00 | O-09 | JE Blatteau | VALIDATION OF A SUBMARINE RESCUE DECOMPRESSION PROCEDURE FROM SATURATION EXPOSURES UP TO 6 ATA IN MAN | |
| 14:20 | O-10 | M Sayer | DECOMPRESSION MANAGEMENT BY 43 MODELS OF DIVE COMPUTER: SINGLE SQUARE-WAVE EXPOSURES TO BETWEEN 15 AND 50MSW | |
| 14:40 | O-11 | AO Brubakk | JENCODEC – THE DIGITAL DIVER | |
| 15:00 | O-12 | J Witte | OXYGEN-DIVER PBMCs SHOW SIGNIFICANT LESS DNA DAMAGE AFTER AN EXPOSURE TO HYPERBARIC HYPEROXIA, THE GENERATED DNA DAMAGE IS DUE TO THE OXYGEN CONCENTRATION | |
| 15:20 | O-13 | D Linnarsson | STRATIFIED INHOMOGENEITY REVISITED: LUNG DIFFUSING CAPACITY WITH INCREASED GAS DENSITY | |
| 15:40 | O-14 | D Madden | EXERCISE AFTER SCUBA DIVING INCREASES THE INCIDENCE OF ARTERIAL GAS EMBOLISM | |
| 16:00 | **Coffee / Tea** | | | |

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|---|---|---|---|---|
| | **Session 4: Diving Medicine** | | **Chair: Cyril D'Andrea** | |
| 16:20 | O-15 | R Arieli | EVOLUTION OF BUBBLES FROM GAS MICRONUCLEI FORMED ON THE LUMINAL ASPECT OF OVINE LARGE BLOOD VESSELS | |
| 16:40 | O-16 | JP Imbert | DOES THE OXYGEN WINDOW CONTROL THE SIZE OF PRE-EXISTING MICRO-BUBBLES? | |
| 17:00 | O-17 | K Lambrechts | EFFECT OF A SINGLE, OPEN-SEA, AIR SCUBA DIVE ON HUMAN MICRO- AND MACRO-VASCULAR FUNCTION | |
| 17:20 | O-18 | A Anão | HEART RATE VARIABILITY DURING HYPERBARIC EXPOSURE – A PHYSIOLOGICAL MODEL WITH APPLICATION IN THE STUDY OF AUTONOMIC FUNCTION DURING DIVING | |
| 17:40 | O-19 | A Schuster | PILOT STUDY: UNDERWATER OSCILLOMETRIC BLOOD PRESSURE MONITORING WITH A $H_2O$ INFLATED CUFF | |
| 18:00 | O-20 | A Schuster | WIRELESS MONITORING OF DIVERS IN A WET CHAMBER DURING LONG TIME EXPOSURE EXPERIMENTS | |
| 18:20 | O-21 | D Ofir | HOW MUCH OF A RISK MIGHT BE INVOLVED IN PERFORMING A YO-YO DIVE? A COMPARISON OF TWO DIFFERENT ANIMAL MODELS. | |
| 18:40 | O-22 | D Smart | FIELD VALIDATION OF YO-YO DIVING SCHEDULES USING DOPPLER ANALYSIS (DA) OF DECOMPRESSION STRESS IN AQUACULTURE DIVERS | |
| 19:00 | **Happy Hour @ Tamarun** | | | |

# O-09 VALIDATION OF A SUBMARINE RESCUE DECOMPRESSION PROCEDURE FROM SATURATION EXPOSURES UP TO 6 ATA IN MAN.

JE Blatteau[1], J Hugon[2], O Castagna[1], C Meckler[1], N Vallée[1], Y Jammes[3], M Hugon[4], J Risberg[5], and C Pény[6].

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**Background:** Recent advances in submarine rescue systems have allowed a transfer under pressure of crew members being rescued from a disabled submarine. The choice of a safe decompression procedure for pressurised rescuees has been discussed for a long time, but no schedule has been validated when the internal submarine pressure is significantly increased, i.e., exceeding 2,8 bar absolute pressure. This study tested a saturation decompression procedure from hyperbaric exposures up to 6 bar, the maximum operating pressure of the NATO submarine rescue system. The objective was to investigate the incidence of decompression sickness (DCS) and clinical and spirometric indices of pulmonary oxygen toxicity (POT).

**Methods:** Two groups were exposed to a nitrogen-oxygen atmosphere ($PO_2$ = 0.5 bar) at either 5 bar (N = 14) or 6 bar (N = 12) for 12 h followed by 56 h 40 min or 60 h respectively of decompression. When chamber pressure reached 2.5 bar, the subjects breathed oxygen intermittently, otherwise compressed air. Repeated clinical examinations, ultrasound monitoring of venous gas embolism and spirometry were performed during decompression.

**Results:** No case of DCS was observed, however three subjects had minor subjective symptoms, i.e., sensation of joint discomfort, regressing spontaneously. After surfacing, two subjects experienced joint discomfort disappearing without treatment. Only three subjects had detectable intravascular bubbles during decompression (low grades). No bubbles were detected after surfacing. Eleven subjects felt chest tightness when inspiring deeply during the initial phase of decompression. Precordial burning sensations were reported during oxygen periods. During decompression, vital capacity decreased by 8-9% and forced expiratory flow rates decreased significantly. After surfacing, changes in the peripheral airways were still noticed; lung diffusion for carbon monoxide was slightly reduced by 1% while vital capacity was normalized.

**Conclusion:** The procedure did not result in significant cases of DCS or POT and may be considered when the internal submarine pressure is significantly increased.

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**Keywords:** Diving, saturation, submarine rescue, decompression sickness, venous gas embolism, bubble, ultrasound, spirometry, pulmonary oxygen toxicity.

## O-10 DECOMPRESSION MANAGEMENT BY 43 MODELS OF DIVE COMPUTER: SINGLE SQUARE-WAVE EXPOSURES TO BETWEEN 15 AND 50MSW

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**Introduction:** Dive computers can be accepted in some occupational diving sectors as tools for managing decompression. There are few comparative studies to guide managers when choosing models of computer that best suit their operational needs. In addition, in Europe, standards and normatives that underpin CE marking for dive computers do not stipulate operational limits for decompression management [1].

**Methods:** Single examples of 43 models of dive computer that are in common use in the UK were set to default settings and compressed to five simulated depths (nominally 15, 20, 30, 40 and 50 msw). They were maintained at those depths until all computers had registered over 30 minutes of decompression. At each depth, and for each model, downloaded data were used to collate the times at which the unit was still registering "no decompression", and the times at which 3, 5, 8, 10, 12, 15, 20 and 30 minutes of decompression were indicated or exceeded. Each depth profile was replicated three times for most models.

**Results:** In general, the decompression isopleths generated for no-stop dives indicated that computers tended to be more conservative than standard decompression tables at depths shallower than 30 msw, but less conservative between 30-50 msw. There was considerable variation between models in the times permitted at all of the depth/decompression combinations; for all tests, the differences between maxima and minima times, expressed as a percentage of the maxima, ranged from 16.7 to 62.5%. Differences in results from replicated trials were minor.

**Conclusion:** The scale of variation in how decompression is managed by the tested dive computers suggests that there may be significant inconsistencies in how decompression stress is being calculated combined with different acceptance levels for DCS probability. Knowing the model of computer used may indicate the level of potential risk to a diver in cases of missed decompression.

**References:**

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**Keywords:** Decompression management, dive computer

## O-11 JENCODEC – THE DIGITAL DIVER

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**Introduction:** Free radicals/oxidative stress and pro-inflammatory injury changes from micro-particles play an important role in decompression.(1-3) Micro-particles are associated with virtually every major category of human disease. Diving and decompression offer well-controlled sets of pathophysiological conditions for modeling. Once the variables in diving are well established, they may provide insight into more complex or less well controlled clinical situations. Thus, diving can be an important contributor to understanding serious human diseases.

**Methods**: Ultrasound shows vascular bubble formation after a dive. Other non-invasive methods enable description of human physiology and pathophysiology in increasing detail. Genomics and biochemistry further improve interpretation of the results. These methods produce vast amounts of very complex data. Mathematical models are very useful for understanding complex biological mechanisms. Their predictive capability can provide insight for interpretation of experimental data and help formulate testable hypotheses.

By combining previous models developed by our group (*Jenny (4), Copernicus (5))* into a new model, ***"JENCODEC; the digital diver"***, individually adapted procedures that also consider the effects of the environment can be developed.

**Results & Discussion:** The Digital Diver is a super-sophisticated computer program that will be capable of generating a virtual living version of the individual diver. When this is achieved, it will be possible to run 'simulations' of the diving processes on the virtual or 'digital' diver, and use the results to make predictions about the outcome of the dive and its effect on the diver. If accidents happen, it will also be possible to determine the best treatment specifically for him/her. This can be termed 'personalized dive procedures (PEDIP)', the basis of future diver healthcare.

This is the same method that is used for developing the Virtual Physiological Human and the International Union of Physiological Sciences' Physiome projects (*EU project DISCIPULUS; The digital patient, FP7/2007-2013).*

**References:**

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**Keywords:** Decompression models; virtual reality; diver health care, DISCIPULUS

## O-12 OXYGEN-DIVER PBMCS SHOW SIGNFICANT LESS DNA DAMAGE AFTER AN EXPOSURE TO HYPERBARIC HYPEROXIA, THE GENERATED DNA DAMAGE IS DUE TO THE OXYGEN CONCENTRATION

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**Introduction:** With the Comet assay, an oxygen-dose and exposure-time dependence of DNA damage could be shown for ex vivo hyperoxia-exposed cells that have never been exposed to hyperoxia before (1). For the ex vivo oxygenation model, it is uncertain whether DNA damage is induced by the oxygen partial pressure ($PO_2$) or by pressure exposure per se. Also, knowledge is very limited about the severity of ex vivo oxygen-induced DNA damage in peripheral mononuclear blood cells (PBMCs) of pure-oxygen divers.

**Methods:** Fresh isolated PBMCs of non-divers (n = 12) and pure-oxygen divers (n = 18) of the German Navy were exposed to hyperbaric hyperoxia of 100% oxygen at a pressure of 4.0 bar (405 kPa) in an experimental hyperbaric pressure chamber on a 96 µl microwellplate at 37-38°C up to 6.5 hours of exposure-time. Previously PBMCs of non-divers (n = 10) were exposed to 100 % oxygen at normobaric pressure (1.0 bar) in comparison to 100% oxygen prepared by ambient air (21% oxygen) at a pressure of 5.0 bar (507 kPa). The DNA damage was analysed hourly by using the alkaline Comet assay and the alternative measuring method – Yes/No Comet – out of 200 cells (Comet assay IV- software).

**Results:** The Comet developement of non-diver PBMCs after exposure to the same $PO_2$ at different exposure pressures showed no significant differences (Figure 1, means and standard deviations, SD, shown). There was significantly less ex vivo induced DNA damage in PBMCs of oxygen divers compared to non-divers (Figure 2, means +/- SD shown).

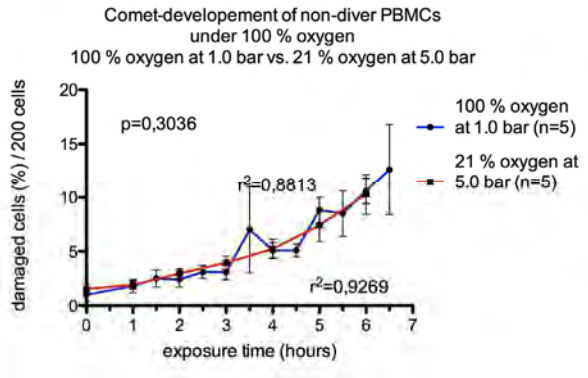


Figure 1

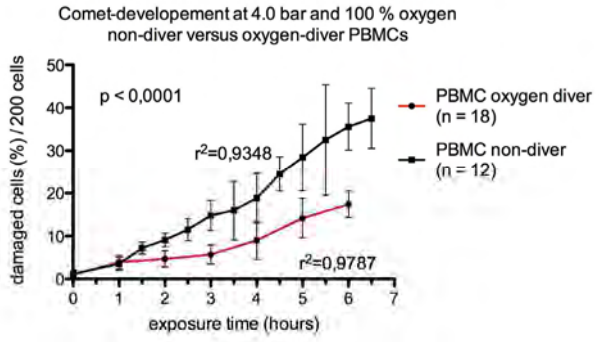


Figure 2

**Conclusion:** DNA double strand breaks after ex vivo hyperbaric oxygenation of PBMCs were induced by the actual $PO_2$, regardless of the exposure-pressure. The results of up to 50 % less DNA damages in PBMCs of oxygen-divers compared to non-divers may be an indication of protective adaptation processes (2,3).

**References:**

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**Keywords:** Hyperoxia, DNA damage, Comet assay, oxygen-divers

## O-13 STRATIFIED INHOMOGENEITY REVISITED: LUNG DIFFUSING CAPACITY WITH INCREASED GAS DENSITY

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**Background:** It is generally recognized that gas exchange in the respiratory zone of the lung takes place by diffusion rather than by convection. Theoretically therefore, breathing a dense gas with reduced diffusivity for metabolic gases should result in stratified inhomogeneity, that is, increased partial pressure differences of metabolic gases within the respiratory zone of the lung. The same should be true for a tracer gas like nitric oxide (NO), when used to determine lung diffusing capacity ($DL_{NO}$).

**Methods:** $DL_{NO}$ was determined in 8 subjects at ambient pressures of 505, 1,015, and 4,053 hPa (379, 761 and 3,040 mmHg) while they breathed normoxic gases.

**Results**: Mean values for $DL_{NO}$ were 116.9 +/- 31.4 (SD), 113.4 +/- 31.3 and 99.3 +/- 28.5 $ml \cdot min^{-1} \cdot hPa^{-1}$ at 505, 1,015, and 4,053 hPa, with a 13 % difference between the two higher ambient pressures ($P$ = 0.002). The data were applied to a model in which $DL_{NO}$ was considered to comprise two serially coupled conductances; the gas phase (Dg), and the alveolo-capillary membrane (Dm). The former should be inversely proportional to ambient pressure and gas density, whereas the latter was assumed to be constant across ambient pressures. The data fitted the model well and we conclude that diffusive transport of NO in the peripheral lung is inversely related to gas density. A Dm of 119.9 $ml \cdot min^{-1} \cdot hPa^{-1}$ could be estimated representing the theoretical maximum for $DL_{NO}$, should there be an infinite Dg. At normal ambient pressure $DL_{NO}$ was 5% lower than Dm and at 4,053 hPa (equivalent depth 30 msw.) this difference was 17%.

**Conclusion:** $DL_{NO}$ underestimates Dm substantially if measured in a hyperbaric environment, for example, during studies of pulmonary oxygen toxicity.

**Keywords**: Diffusivity, hyperbaric, gas exchange, nitric oxide

## O-14 EXERCISE AFTER SCUBA DIVING INCREASES THE INCIDENCE OF ARTERIAL GAS EMBOLISM

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**Introduction**: Arterialization of venous gas emboli (VGE) after decompression from scuba diving has traditionally been associated with pulmonary barotrauma or cardiac defects such as a patent foramen ovale (PFO). Recent studies have demonstrated the right-to-left passage of emboli through intra-pulmonary arterio-venous anastomoses (IPAVA) that allow blood to bypass the pulmonary microcirculation [2, 4]. These passages open up during exercise [1] and the aim of this study is to see if exercise in a post-diving period increases the incidence of arterialization. Further studies show that application of 100% oxygen can close these shunts [3]. A secondary goal was to observe arterialization after the application of oxygen.

**Methods:** After completing a dive, PFO-negative test subjects were monitored via trans-thoracic echocardiography (TTE) for bubble grade at rest. Subjects then completed an incremental cycle ergometry test to exhaustion under continuous TTE observation. Exercise was suspended if arterialization was observed and resumed when the arterialization cleared. Exercise continued and supplemental oxygen was provided and the time for arterialization to clear was compared to the first round when subjects were breathing room air.

**Results**: Out of 23 subjects, 3 arterialized at rest, 12 arterialized with exercise and 8 did not arterialize at all even during maximal exercise. The time for arterialization to clear with oxygen was significantly shorter than without ($P$ = 0.035). Exercise after diving increased the incidence of arterialization from 13% at rest to 52%.

**Discussion:** This study shows that individuals are capable of arterializing through IPAVA and that the degree to which these open varies between individuals. Basic activities associated with scuba diving such as surface swimming or walking with heavy equipment may be enough to allow the passage of VGE through IPAVA.

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**Keywords:** SCUBA diving, exercise, VGE, arterialization, IPAVA

## O-15 EVOLUTION OF BUBBLES FROM GAS MICRONUCLEI FORMED ON THE LUMINAL ASPECT OF OVINE LARGE BLOOD VESSELS

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**Introduction**: It has been shown that gas nanobubbles form spontaneously on a smooth hydrophobic surface submerged in water. These nanobubbles were shown to be the source of gas micronuclei from which bubbles evolved during decompression of silicon wafers. We suggest that the hydrophobic inner surface of blood vessels may be a site of nanobubble production.

**Methods**: Sections from the right and left atria, pulmonary artery and vein, aorta, and superior vena cava of sheep ($n$ = 6) were stretched on microscope slides and exposed to 1.013 MPa for 18 h.

**Results**: Hydrophobicity was checked in the six blood vessels by advancing contact angle with a drop of saline of 71 ± 19 degrees, with a maximum of about 110 ± 7 degrees (mean ± SD). Tiny bubbles ~30 μm in diameter rose vertically from the blood vessels and grew on the surface of the saline, where they were photographed. All of the blood vessels produced bubbles over a period of 80 min. The number of bubbles produced from a square cm was: in the aorta 20.5; left atrium 27.3; pulmonary artery 17.9; pulmonary vein 24.3; right atrium 29.5; superior vena cava 36.4. More than half of the bubbles were present for less than 2 min, but some remained on the saline-air interface for as long as 18 min.

**Conclusion**: Nucleation was evident in both the venous (superior vena cava, pulmonary artery, right atrium) and arterial (aorta, pulmonary vein, left atrium) blood vessels. This newly suggested mechanism of nucleation may be the main mechanism underlying bubble formation on decompression.

**Keywords:** nanobubble, hydrophobic surface, arterial bubbles, nucleation

## O-16 DOES THE OXYGEN WINDOW CONTROL THE SIZE OF PRE-EXISTING MICRO-BUBBLES?

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**Introduction:** The question was raised after the review of publications on divers preconditioning. They showed that bubbles in decompression relate to an initial population of micro-bubbles present in the diver's tissues prior to the dive. It remains explaining how these pre-existing bubbles form and balance their inner and outer pressure to last. We postulated that:

- the tissue "oxygen window" (OW) is the mechanism driving their formation.
- Non-extensive thermodynamics can provide an expression for their interfacial energy and stabilization.

**Method:** We first designed a mathematical model for the calculation of the tissue OW. We used it to define the pressure difference between the inner and outer pressure. We then referred to a derivation established with non-extensive thermodynamics for calculating the stabilization energy. Finally, we established a new condition for the micro-bubbles stability. Contrary to other bubble models, the equation refers neither to surface tension nor specific geometrical configurations but only to gas volumes.

**Results:** We confronted this equation to divers' pre-conditioning using oxygen breathing at surface. Obviously, oxygen breathing has a direct effect on the OW and reduces the size of pre-existing gas pockets.
We then studied experiments with pre-conditioning based on exercise. Our OW model states that exercise has little effect on the OW. We deducted that gas pockets were stored in non-perfused area and that exercise re-perfuse capillaries and wash them away before decompression. We found this explanation also consistent with pre-conditioning based on sauna.

**Discussion / Conclusion:** Our equation accounts for the existence and survival of pre-existing micro-bubbles. Experiments indicate that they should be vascular and located in non-perfused capillaries. They appear as an intermediate step in between gas nuclei and decompression bubbles. Variation of the OW or re-perfusion of the capillaries could be the mechanisms reducing the initial micro-bubble population and protecting the diver in further decompression.

**Keywords:** Oxygen window, micro-bubbles, preconditioning, decompression bubbles

## O-17 EFFECT OF A SINGLE, OPEN-SEA, AIR SCUBA DIVE ON HUMAN MICRO- AND MACRO-VASCULAR FUNCTION

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**Introduction:** Previous studies have shown bubble-induced endothelial damage on conduit arteries.[1] We aimed to evaluate the effect of diving on microvascular and macrovascular function.

**Methods:** Nine divers took part in a scuba dive to 30 msw (400 kPa) for 30 min bottom time. Pre- and post-dive, they underwent an assessment of endothelial-dependent (acetylcholine) and endothelial-independent (sodium nitroprusside) microvascular function (Laser Doppler Flowmetry), as well as endothelial-dependent (flow-mediated dilation) and endothelial-independent (nitroglycerin-mediated dilation) function. Bubble grades were monitored with Doppler according to the Spencer grade.

**Results:** Mean +/- SD KISS bubble score ranged from 21.1 ±4.7 at rest to 55.0 ± 8.8 after knee flexion. The increase in cutaneous blood flow elicited by either acetylcholine (2,427 ± 705% to 724 ± 122%, $P = 0.03$) or sodium nitroprusside (3,260 ± 821% to 735 ± 192%, $P = 0.02$) was significantly reduced after diving. Similarly, both flow-mediated dilatation (10.8 ± 4% to 5 ± 1%, $P = 0.002$) and nitroglycerin-mediated dilatation (15 ± 2% to 6 ± 2%, $P = 0.001$) were also significantly decreased. There were no correlations between vascular parameters and bubble formation.

**Conclusion:** There appears to be a reduction in endothelium-dependent and -independent, macro- and microvascular function. Our results suggest that in the process of vascular dysfunction during diving, functional changes in the vessel wall may not be limited to the endothelium and may be mediated by alterations in vascular smooth muscle.

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**Keywords:** Decompression, endothelium, microcirculation, laser-Doppler flowmetry

## O-18 HEART RATE VARIABILITY DURING HYPERBARIC EXPOSURE – A PHYSIOLOGICAL MODEL WITH APPLICATION IN THE STUDY OF AUTONOMIC FUNCTION DURING DIVING

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**Introduction:** Exposure to the subaquatic environment produces a complex cardiovascular response, related to the acute changes in barometric pressure, temperature, physical exercise and different patterns of submersion. The physiological evaluation of heart rate variability (HRV) during rapid pressure changes in a controlled environment, as verified in a hyperbaric chamber, can be useful in the characterization of the autonomic function attributed to barometric changes during a normal diving response.

**Methods:** We evaluated 12 healthy subjects (5 females), age 36 ± 9.8, during a hyperbaric chamber session at 253 kPa, in stable temperature of $22^0$C. Heart rate was continuously registered in sitting position (sampling rate 1000Hz), 10 minutes (min) before pressurization (Phase 1), during pressurization (10 min; $\Delta P$= 15 kPa $min^{-1}$ –Phase 2) and during 10 min at 253 kPa (Phase 3). The following variables were evaluated: RR interval (iRR), the time domain measures SDNN and RMSSD, and pNN50, triangular index (TrIn) and "Lorenz plot" (SD1, SD2) autoregression of the low (LF) and high frequency (HF) specter normalized units (nu) and LF/HF.[1,2] We compared the different phases with paired sample analysis.

**Results:** We obtained in Phases 2 and 3 a significant increase in RRi and a tendency of variation for the generality of markers of parasympathetic modulation, reaching statistical significance for SDNN, RMSSD and SD1. For the generality of variables the variation was most pronounced during pressurization (Phase2) – Table1.

| | iRR | SDNN | RMSSD | pNN50 | InTr | SD1 | SD2 | LFnu | HFnu | LF/HF |
|---|---|---|---|---|---|---|---|---|---|---|
| Phase1 | 755±114 | 60,8±31,0 | 40,6±45,1 | 5,0±5,1 | 12,1±1,5 | 28,7±31,5 | 79,2±35,2 | 77,0±20,67 | 22,8±20,5 | 5,4±2,8 |
| Phase2 | 671±101* | 85,3±49,9* | 89,5±73,4* | 7,0±11,9 | 11,7±2,4 | 61,1±15,4* | 96,1±52,5 | 63,9±18,8 | 35,8±18,7 | 3,2±3,8 |
| Phase3 | 733±142Ψ | 62,3±19,4(1) | 38,1±28,5Ψ! | 9,0±10,3 | 12,9±4,2 | 25,6±5,4Ψ! | 83,0±25,0 | 63,1±26,2 | 29,9±17,4 | 3,9±3,5 |

p<0,05 : * F1vs F2; ! F1 vs F3; Ψ F2 vs F3
(1) F2vs F3: p=0,058

Table-1

**Conclusions:** Exposure of healthy volunteers to a hyperbaric air environment at 253 kPa is associated with an increase in vagal activity, particularly during the pressurization phase. The evaluation of HRV, in the controlled conditions of a hyperbaric chamber, can be a useful tool for further research in the characterization the autonomic response and associated cardiovacular effects during different patterns of diving.

**References:**

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2. Bilchick KC, Berger RD. Heart Rate Variability. *J Cardiovasc Electrophysiol* 2006; 17(6):691-694.

**Keywords:** Heart rate variability, autonomic function, diving physiology

## O-19 PILOT STUDY: UNDERWATER OSCILLOMETRIC BLOOD PRESSURE MONITORING WITH A $H_2O$-INFLATED CUFF

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**Introduction:** Assessment of blood pressure underwater on divers requires special instrumentation that is water- as well as pressure-proof. Previously we have developed three prototype blood pressure meters, all of which were based on inflation of a cuff with air from a scuba tank [1,2,3]. The method works well, but requires a static depth during measurement, as any depth changes of the subject will lead to a pressure change in the cuff. This makes BPM measurements on moving divers without depth reference practically impossible. Electronic compensation of the pressure-induced error is possible, but only with small depth changes. As an alternative, one could inflate the cuff with water.

**Methods:** While pressure control in a standard cuff (flexible but not elastic) is simple because of the compressibility of gas, it is difficult when water is used as inflation medium, because small volume changes lead to large pressure changes. A reasonable compliance is usually required for precise pressure control. In an experiment three different cuffs were inflated with water utilizing an electric micro pump and the differential pressure was measured with 24 bit digital pressure sensors.

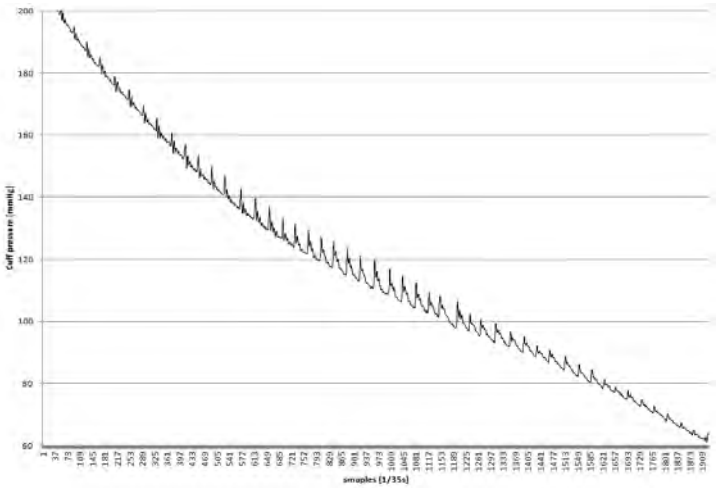


Figure 1: Pressure signal from a water inflated cuff

**Results:** In all 3 cuffs, oscillations were reliably detected. The best signal-to-noise ratio was achieved with an inflexible cuff tightly placed on the upper arm. At MABP oscillations of up to 6-8 mmHg were measured.

**Conclusion:** Blood pressure assessment with a water-inflated cuff is possible, but precise pressure control is more difficult. The pressure sensor has to be located directly on the cuff without a long hose, as small hose movements will lead to a high noise level typically larger than the oscillations themselves.

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3. A. Schuster, M. Ljubkovic, Z. Dujic, A. Sieber. Novel oszillometric method for underwater blood pressure monitoring. EUBS 2012 Conference, Belgrade, September 2012

**Keywords:** Blood pressure measurement, cuff, underwater

**Acknowledgment:** The work was co-funded by the EU-MC-ITN PHYPODE and relates to the US Navy Grant N62909-11-1-7044 (Office for Naval Research Global).

## O-20 WIRELESS MONITORING OF DIVERS IN A WET CHAMBER DURING LONG TIME EXPOSURE EXPERIMENTS

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**Introduction:** Continuous monitoring of core and skin temperature and one channel ECG was required for a 10 h exposure experiment in a wet chamber at 20 m depth and 17°C water temperature. For the safety of the diver a wireless data transmission was requested.

**Methods:** ECG is typically recorded with a sampling rate of at least 200 Hz with 10-24-bit resolution. 200 Hz and 24-bit resolution results in a data stream of approximately 5 kBit $s^{-1}$. Temperature data from 9 sensors are collected with a VitalSense recorder in 30 s intervals, which adds additional 24 bit $s^{-1}$ (9 sensors x 80 bits per data transmission per 30 s) to the necessary transmission bandwidth.

State of the art underwater wireless data transmission is usually based on acoustic technology, but in a closed space like a pressure chamber many reflections occur which cause disturbances. Wireless transmission technology used in diving computers use an electromagnetic carrier between 5 and 8 kHz, which allows data rates of only a few Hz. Bluetooth and WLAN are based on 2.5 GHz wireless technology and penetrate water only a few cm.

An alternative device was developed, which transmits data optically through a window of the pressure chamber. A waterproof box houses the temperature logger and the ECG circuit. A microcontroller combines the data streams from the temperature logger and the ECG and transmits them via USART at 19200 BAUD to a second microcontroller. This one transmits the data optically at 820 nm with a 450 kHz carrier.


*Figure 1: Prototype of the system*

**Results:** A first prototype (figure1) was assembled and used during three 10 h experiments. ECG and temperature data transmission worked flawlessly. The data were visualized and recorded with a GUI developed in National Instruments LabWindows™.

**Conclusion:** Optical data transmission of ECG and temperature data is an economic, safe and reliable alternative to wired technology.

**Keywords:** Wireless, ECG monitoring, temperature monitoring, wet chamber

**Acknowledgment:** The work was co-funded by the Marie Curie ITN PHYPODE and the French Navy (IRBA).

## O-21 HOW MUCH OF A RISK MIGHT BE INVOLVED IN PERFORMING A YO-YO DIVE? A COMPARISON OF TWO DIFFERENT ANIMAL MODELS

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**Background:** We evaluated the risk of decompression sickness (DCI) following 'yo-yo' dives in, rat and pig models.

**Methods – rats**: Seventy-six rats were dived to 1.013 MPa (10 ATA) for 32 min bottom time. Group A breathed air and Group B a 3% $O_2$ / 97% $N_2$ gas mixture. Each group was divided into three subgroups for 'peeps', with an ascent/descent rate of 10 m $min^{-1}$: A1& B1– control (no 'peeps'; n = 16 and12 respectively); A2 & B2 – 2 'peeps'; A3 & B3 – 3 'peeps' (n = 12 for each group). A 'peep' was defined as a single ascent for 1 min at surface, and then descent back to the bottom pressure.

**Methods – pigs**: Twenty-eight pigs were divided into four groups. Group A: control, no 'peeps'; Group B: 2 'peeps'; Group C: 4 'peep' (n = 7, 8 and 8 respectively); and Group D: sham pigs that weren't expose to any dive (n = 5). The dive was conducted on air to 507 kPa (5 ATA) for 30 min. Before and for 90 min after the dive, echocardiography was performed to detect gas bubbles in the heart.

In both models, motor performance was observed after the dive. The significance of differences between groups was tested using Chi Square test where $P < 0.05$ was accepted as significant.

**Results – rats**: Thirteen of the 16 rats in group A1 and all the rats in B1 developed symptoms of DCI. In groups A2 and B2, 6 of 12 and 9 of 12 rats respectively, and 2 of 12 in both groups A3 and B3 developed symptoms of DCI. The differences between the A subgroups were significant ($P < 0.001$). Significant differences were also found between the B subgroups ($P < 0.01$).

**Results – pigs**: DCI symptoms were observed in 4 of 7 pigs of group A (one died); 2 of 8 pigs in group B (one died); and 3 of 8 pigs in group C (one died). In the majority of the animals, gas bubbles were detected in the right ventricle. However, we found gas bubbles in the left ventricle of 3 animals of group C, a finding not observed in the other groups. Chi Square analysis revealed insignificant differences probably because the experimental groups were too small.

**Conclusions**: In both rats and pigs, 'peeps' significantly reduced the risk of DCI. 'Peeps' increased the mortality of DCI in rats though not in pigs. Following four "peeps" in pigs, we observed an estimated higher number of bubbles in the right ventricle and increased number of bubbles in the left ventricle.

**Key words**: Decompression illness, pug, rat, yo-yo dive

## O-22 FIELD VALIDATION OF YO-YO DIVING SCHEDULES USING DOPPLER ANALYSIS (DA) OF DECOMPRESSION STRESS IN AQUACULTURE DIVERS

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**Introduction:** Tasmania's aquaculture industry produces over 30,000 Tonnes of fish annually (value > AUD300 million). Past high levels of decompression illness (DCI) may have resulted from "yo-yo" diving, and empirical dive tables were actioned in the 1990's to mitigate risk.

**Aim:** To assess working divers, using Doppler analysis (DA) to determine if yo-yo diving was an independent risk factor for DCI, and to determine safe dive profiles.

**Methods:** Field data were collected during bounce diving at marine farms near Hobart, Tasmania. Ascent rates were < 18 m $min^{-1}$ with routine safety stop (3 min @ 3 msw) after the last bounce. After diving, DA was performed at 20-minute intervals (previously validated DR&D methods). The Kisman-Masurel scale (bubble grades 0 to 4) was used to grade decompression stress. KM scales post-diving were compared to known DR&D tolerances; rejected as unsafe if >50% of scores were over grade 2. Divers completed pre- and post-dive health questionnaires reporting up to 24 hours.

**Results:** Between 2001-2007, bounce-diving Doppler field data was collected from a total of 150 divers. DA was consistent with low stress dive profiles. DA grades did not correlate with the number of bounces, hence the original hypothesis was rejected. Three bounce profiles were validated; all with DA median grade zero: 13-15 m, 10 bounces inside 75 minutes, 16-18 m, 6 bounces inside 50 minutes and 19-21 m, 4 bounces inside 35 minutes. Further evaluation has validated combined bounce and square profile dives and two successive series of bounces. Field DA has improved industry productivity, compared to empirically derived tables with good safety record maintained through 2012.

**Conclusions:** These data suggest that bounce diving was not a major factor causing DCI in aquaculture divers. Data are now available to provide guidance for bounce diving at depths up to 21 m, combination bounces with square profiles and successive bounce profiles.

**Keywords:** Yo-yo diving, bounce diving, decompression illness, aquaculture diving, Doppler analysis, dive tables

## Wednesday, September 25th, 2013

**Tricontinental Scientific Meeting, Special Session "Asthma and Diving"**
*Location: "Nacre" room, Tamarun*

| | | | | |
|---|---|---|---|---|
| | **Special Session: "Asthma and Diving"** | | **Chair: Phil Bryson** | |
| 14:30 | | WAJ Meintjes | Introduction – what are the issues with asthma and diving? | |
| 14:50 | | WAJ Meintjes | Asthma and diving in Southern Africa | |
| 15:10 | | C Edge | Asthma and diving in the UK | |
| 15:30 | | M Bennett | Asthma and diving in Australasia – the SPUMS diving medical appendix | |
| 15:50 | | P Denoble (tbc) | Asthma and diving from a US perspective | |
| 16:10 | | Chair-led discussion | Defining the key differences. Where do we go from here – the research agenda | |
| 16:30 | **Adjourn** | | | |

**Tricontinental Scientific Meeting, Special Keynote**
*Location "Aquarium of Reunion Island", St.Gilles-les-Bains Marina*

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| 17:00 | **Exclusive visit to the Aquarium of La Reunion** | | |
| 19:00 | | **Keynote Lecture:** | |
| | | R Fitzpatrick | DANGEROUS MARINE ANIMALS AND THE MEDIA: FACT OR FICTION ? |
| 20:00 | **Drinks and snacks @ Aquarium, offered by ARESUB** | | |

## Thursday, September 26th, 2013

**Tricontinental Scientific Meeting, Day Three**

*Location: "Le Voilier" room, Tamarun*

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| 09:00 | | **Keynote Lecture:** | | |
| | | J Kot | CONTROVERSIES ON TECHNICAL AND PROCEDURAL ASPECTS OF HBO THERAPY | |
| | **Special Session "Controversies in Hyperbaric Medicine"** **Chair: Peter Germonpré** | | | |
| 09:40 | | S Mitchell | WHAT IS AN 'UNAPPROVED' INDICATION ? – A ROSE BY ANY OTHER NAME | |
| 10:20 | | M Bennett | OK – LET'S GET THIS STRAIGHT! ABOUT PLACEBO, NOCEBO, HAWTHORNE AND PARTICIPATION EFFECTS IN HBO | |
| 10:40 | **Coffee / Tea** | | | |
| 11:00 | | I Millar | WE WILL NEVER AGREE ON HBOT FOR CARBON MONOXIDE POISONING. HOW DID WE GET HERE? | |
| 11:20 | | M Bennett | SUDDEN HEARING LOSS – WHY ARE WE DEAF TO THE EVIDENCE? | |
| 12:00 | **Lunch** | | | |

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| 13:00 | | **Keynote Lecture:** | | |
| | | R Fitzpatrick | FILMING MARINE LIFE FOR TV | |
| | **Session 5: Diving Medicine** **Chair: Karen Richardson** | | | |
| 13:40 | O-23 | P Adolfsson | IN TYPE 1 DIABETES NEW TECHNOLOGY CREATES OPPORTUNITIES TO DIVE WITH INCREASED SAFETY | |
| 14:00 | O-24 | BN Andrews | THE VALIDITY OF SPIROMETRY PERFORMED ON NAVY DIVERS AND SUBMARINERS FROM 1 JULY 2010 - 1 JULY 2012 | |
| 14:20 | O-25 | M Mattiuzzo | INFLUENCE OF HYPERBARIC PRESSURE ON THE PERFORMANCE OF TWO TYPES OF SALBUTAMOL METERED DOSE INHALERS | |
| 14:40 | O-26 | M Pieri | ANALYSIS OF ACCIDENTS IN DAN EUROPE DSL ( DIVING SAFETY LABORATORY) DATABASE AND GRADIENT FACTOR EVALUATIONS | |
| 15:00 | O-27 | DM Fothergill | THE POTENTIAL OF PORTABLE OXYGEN CONCENTRATORS TO PROVIDE OXYGEN FOR REMOTE DIVING CASUALTIES | |
| 15:20 | O-28 | JE Blatteau | PREVENTION AND TREATMENT OF DECOMPRESSION ILLNESS USING IN-WATER RECOMPRESSION: RELEVANCE OF A TRAINING PROGRAM FOR FISHERMAN DIVERS IN VIETNAM | |
| 15:40 | O-29 | A Sieber | REDUNDANT $PO_2$ AND $PCO_2$ AND GAS MONITORING SYSTEM FOR REBREATHERS COMPRISING 2 GALVANIC $PO_2$ SENSORS, ONE OPTICAL $PO_2$ SENSOR AND ONE DUAL WAVELENGTH $PCO_2$ SENSOR | |
| 16:00 | **Coffee / Tea** | | | |
| | **Session 6: Hyperbaric Oxygen Therapy** **Chair: Mike Bennett** | | | |
| 16:20 | O-30 | T Wunderlich | AGE-DEPENDENT EFFECTS OF HYPERBARIC HYPEROXIA ON HUMAN PERIPHERAL VASCULAR FUNCTION | |
| 16:40 | O-31 | T Wunderlich | AGE-DEPENDENT EFFECTS OF HYPERBARIC HYPEROXIA ON DIASTOLIC MYOCARDIAL FUNCTION | |
| 17:00 | O-32 | RK Peleg | EFFECT OF HYPERBARIC OXYGEN THERAPY ON BLOOD GLUCOSE LEVELS IN PATIENTS WITH DIABETES MELLITUS, BRAIN INJURY AND HEALTHY VOLUNTEERS | |
| 17:20 | O-33 | M Nolting | HYPERBARIC OXYGEN THERAPY INCREASES INSULIN SENSITIVITY IN OVERWEIGHT MEN WITH AND WITHOUT TYPE-2 DIABETES | |

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| 17:40 | O-34 | A Cakkalkurt | THE EFFECTS OF HYPERBARIC OXYGEN ON CULTURED HUMAN BREAST CANCER CELLS | |
| 18:00 | O-35 | B Aviner | LOW HYPERBARIC PRESSURE MODULATES $Ca^{2+}$ INFLUX VIA VOLTAGE DEPENDENT $Ca^{2+}$ CHANNELS IN CULTURED CORTICAL NEURONS | |
| 18:20 | O-36 | F Tillmans | EFFECTS OF NORMOBARIC HYPEROXIA ON THE ACTIVITIES OF COMPLEX I AND II OF THE MITOCHONDRIAL RESPIRATORY CHAIN IN DIFFERENT LEUKEMIC CELL LINES | |
| 18:40 | O-37 | W Hemelryck | EFFECT OF NORMOBARIC OXYGEN BREATHING ON HUMAN BRAIN COGNITIVE FUNCTIONS | |
| 19:00 | **Happy Hour @ Tamarun** | | | |

## O-23 IN TYPE 1 DIABETES NEW TECHNOLOGY CREATES OPPORTUNITIES TO DIVE WITH INCREASED SAFETY

P Adolfsson[1,2], J Jendle[3,4], and H Ornhagen[5]

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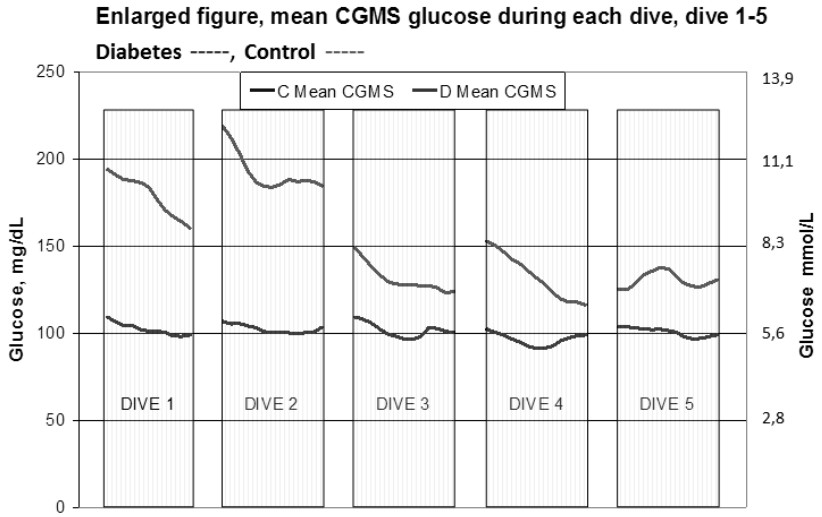
**Introduction:** In type-1 diabetes mellitus, glucose control has been assessed by self-monitoring of blood glucose. Continuous glucose monitoring (CGM) offers an alternative or complementary method. Studies have been conducted where CGM have been used during scuba diving and in a pressure chamber, providing a basis for the updated Swedish recommendations on recreational diving and diabetes mellitus.[1]

**Methods:** CGM was used during five repetitive dives over three days using a dry suit.[2] In another study CGM was used within a pressurized aluminum container.[3] Later, CGM was evaluated in a pressure chamber, including both an *in vitro* [4] and an *in vivo* [5] study. Sensors attached to two different CGM systems were immersed in three different glucose concentrations and exposed to scheduled pressure changes.[4] The performance of the sensors was also evaluated attached in one healthy individual who was exposed to the same scheduled pressure changes.[5]

**Results:** Used beneath a dry suit, CGM recordings were available during all dives (n = 117), while within a pressurized aluminum container, for 56% (27/48) of the monitored dives. Mean absolute relative difference between plasma and interstitial glucose was 14.4 ± 6% and 13.1 ± 5.4%, whereas the coefficients of correlation (r) were 0.93 ± 0.04 and 0.95 ± 0.02 respectively. Hypoglycemia was detected in both studies, interestingly without symptoms. All sensors worked in the pressure chamber. No significant differences in sensor signal were noticed depending on applied pressure conditions, glucose concentration, pre-wetted sensor or sensor insertion site in these studies.




**Conclusion:** CGM offers a potential advantage revealing hypoglycaemia unawareness before diving. In close relation to dive, CGM provides a useful tool on which to base the decision whether a dive should be conducted or not. Therefore CGM could increase the safety of diving with type 1 diabetes.

**References:**

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5. Adolfsson P, Ornhagen H, Eriksson BM, et al. Continuous glucose monitoring – a study of the Enlite sensor during hypo- and hyperbaric conditions. *Diabetes Technol Ther* 2012; 14(6):527-32.

**Keywords:** Glucose, continuous glucose monitoring, diabetes mellitus, diving, barometric pressure

## O-24 THE VALIDITY OF SPIROMETRY PERFORMED ON NAVY DIVERS AND SUBMARINERS FROM 01 JULY 2010 – 01 JULY 2012

BN Andrews[1] and WAJ Meintjes[1]

[1]Stellenbosch University, Faculty of Health Sciences

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**Introduction:** We investigated the acceptability and reproducibility of lung function tests performed on a population of navy divers and submariners for the period 01 July 2010–01 July 2012. Spirometry forms an important component of the Fitness to Work Assessment. The outcome of this test may have long term implications on an individual's career.

**Methods:** A cross sectional study design was used. All lung function tests performed on a sample population of naval divers and submariners for the study were stored electronically for analysis. The variables captured included participant age, height, weight, gender, smoking habits, mustering (diver or submariner), test operator, test date, calibration date and the acceptability and repeatability of each lung function test.

**Results:** A total ofof 550 lung function tests were analysed : 42.7% (n = 235) of the tests recorded three or more acceptable curves. Of the 235 acceptable curves, 91.9% (n = 216) were reproducible. The acceptability of the tests was statistically associated with individual operators, but no association was found between acceptability and the other variables.

**Conclusions:** The training and performance of operators is an important component in the overall validity of lung function test results.

**References**:

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**Keywords**: Spirometry, divers

## O-25 INFLUENCE OF HYPERBARIC PRESSURE ON THE PERFORMANCE OF TWO TYPES OF SALBUTAMOL METERED DOSE INHALERS

M Mattiuzzo[1], O François[1], C Combescure[2], P Bonnabry[1,4], C Fonzo-Christe[1], and R Pignel[3]

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**Background:** Hyperbaric oxygenotherapy (HBOT) is used for various clinical conditions. Elective patients with asthma might need bronchodilator treatment inside the hyperbaric chamber. Salbutamol metered dose inhalers (MDIs) may deliver a lower quantity of aerosol under hyperbaric pressures [1]. We evaluated the performance of two salbutamol MDIs (Ventolin® aerosol-nebuliser, GlaxoSmithKline; Salamol® Autohaler, Teva Pharma).

**Methods:** The performance of two MDIs was evaluated by determination of the delivered quantity of aerosol. Each device was weighed before and after 15 actuations at 101.3, 253 and 405 kPa and with a full, half-empty or near-empty canister. Tests were performed in triplicate for each condition. Results are expressed as mean of weight difference in mg (+/-SD) and compared using Kruskal-Wallis test.

**Results:** Ventolin: the delivered quantity of aerosol was significantly lower as pressure increased ($P = 0.03$).
Salamol: the delivered quantity of aerosol was not significantly influenced by the pressure increase ($P > 0.05$)
Differences observed for the different filling conditions at a given pressure were not significant for both MDIs.

**Conclusion:** Increased pressure reduced by 20% the quantity of aerosol delivered by Ventolin at 405 kPa. Reasons for the differences observed between the two devices may be explained by differences in the triggering mechanism. The clinical consequences of these observations on the management of an asthma crisis under hyperbaric conditions are unknown and should be investigated. Performance of devices in hypobaric conditions should also be investigated.

**References:**

1. Johnson GA et al. Albuterol metered dose inhaler performance under hyperbaric pressures. *Undersea Hyperb Med* 2009; 36:55-63.

**Keywords:** Hyperbaric oxygen therapy, salbutamol, asthma, aerosol

## O-26 ANALYSIS OF ACCIDENTS IN THE DAN EUROPE DIVING SAFETY LABORATORY DATA BASE AND GRADIENT FACTOR EVALUATIONS

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**Introduction:** Diving Decompression accidents are luckily not very frequent, but at times very serious, furthermore they often occur after 'innocent' dives according to the current decompression models (60% of cases in the DAN Europe past accident and claims data base). For this reason we started an in-depth epidemiological analysis on the DAN Diving Safety Laboratory Data Base (DSLDB). Our scope is to identify possible inherent risk factors such as biometrical data, medical history, as well as the relationship between tissue supersaturation expressed as gradient factor (GF) and DCS.

**Materials and Methods**: 181 DCS cases from the (DSLDB) were analysed and the resulting data were compared with tissue saturation values according to the Buhlmann ZH16 Model. Possible correlation between DCS cases and anthropometric data (sex, age, weight, height, BMI) equipment malfunctions and medical history were also evaluated. Original software was developed for the analysis of GF values.

**Results:** Of the 181 DCS cases recorded, only 53 (30%) showed a GF > 0.85, of which only 1 implied a GF > 1. i.e., only one of all these cases would have been "predicted" by the ZH16 model. The majority of cases (128/181) occurred in a 'grey zone' between 0.72 and 0.84 GF values according to the ZH16 model ($P < 0.001$). Usually the highest GF value on surfacing for the 38.2 minute half-time compartment of the ZH16 model.No other significant correlations between DCS and anthropometric data (sex, age, weight, height, BMI), equipment malfunctions or medical history) were found.

**Conclusion:** Our data show that the majority of DCS cases in the DSLDB occurred when the GF was less than 0.85 according to Buhlmann ZH16 model, that is after dives which are considered 'safe' according to that model. The reliability of current algorithms shows 'grey areas' as to their ability to predict DCS that need further research and, most likely, a more physiological approach to decompression.

**Keywords:** Gradient factor, compartmental model, diving, ascent speed, M-value

## O-27 THE POTENTIAL OF PORTABLE OXYGEN CONCENTRATORS TO PROVIDE OXYGEN FOR REMOTE DIVING CASUALTIES

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**Introduction:** Recent advancements in $O_2$ concentrator technology have resulted in portable, low-power oxygen ($O_2$) concentrators that can convert ambient air into 90-95% $O_2$ for normobaric $O_2$ treatment. This presentation describes ongoing work conducted at the Naval Submarine Medical Research Laboratory (NSMRL) that assesses the potential of commercially available portable $O_2$ systems to provide emergency oxygen for a diving casualty.

**Methods:** The U.S Food and Drug Administration 510(k) data base was searched to determine the number of approved portable medical oxygen generators available in the US market. Products receiving 510(k) approval within the 5-year period 2006-2011 were further investigated for type of $O_2$ concentrator technology, maximum $O_2$ flow and $O_2$ fraction delivered.

**Results:** In the last 5 years, 42 products from 26 companies received 510(k) clearance to market their portable $O_2$ concentrators in the U.S. Thirty-five of these products use molecular sieve pressure swing adsorption (PSA) technology to generate >90% $O_2$ from ambient air. Only 6 products could output >10 l $min^{-1}$ $O_2$ and these products require a 120 volt AC power supply. The majority of light weight portable battery/DC powered $O_2$ concentrators deliver $\leq$ 5 l $min^{-1}$ of oxygen.

**Conclusions:** When using PSA technology to provide portable medical grade $O_2$ (United States Pharmacopeia (USP) oxygen 93%), there is a compromise between $O_2$ output flow rate and the size, weight and power requirements of the system. Currently available $O_2$ concentrators that provide high $O_2$ flows are not practical for remote dive operations due to their large weight and power requirements. Potential solutions for using smaller lower power $O_2$ concentrators that will support resting minute ventilation levels and provide an $FiO_2 \geq 90\%$ include coupling one or more units to a hi flow oxygen mask or to a closed-circuit $O_2$ rebreather circuit. The effectiveness of these solutions is currently being investigated at the NSMRL.

**Keywords:** Oxygen concentrator, pressure swing adsorption, medical oxygen, diving.

(Supported by grants from NAVSEA and the Defence Medical Research and Development Program)

# O-28 PREVENTION AND TREATMENT OF DECOMPRESSION ILLNESS USING IN-WATER RECOMPRESSION: RELEVANCE OF A TRAINING PROGRAM FOR FISHERMAN DIVERS IN VIETNAM.

JE Blatteau[1,5], JM Pontier[4,5], V Mui Nguyen[2,5], V Than Nguyen [3,5], P Cavenel[5], and J Ruffez[5]

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**Background:** Most of fisherman divers in Vietnam use surface-supplied compressed air and suffer from decompression illness (DCI), responsible for joint pain, severe neurological deficits or even death. Access to a hyperbaric center is unfortunately limited. The objective of this study was to evaluate the relevance of a training program to prevent and treat DCI using in-water recompression (IWR).

**Methods:** Two sites in central Vietnam were selected, including about 1250 fisherman divers, with a fishing activity in remote areas. There were 63 divers interviewed and trained over a period of 3 years from 2009. 51% of all trained divers were re-interviewed in 2011-2012 for investigating results in mortality and morbidity as well as changes in diving practices.

**Results:** Since 2009, most of the fisherman divers have changed their diving practices, limiting bottom time or depth. They also took into account dangers associated with their equipment to avoid broken hoses or compressor failures. Mortality was significantly reduced from prior to 2009, and the incidence rate of severe neurological DCI fell by three-quarters. A total of 24 DCI were reported, all were treated by IWR, with $O_2$ breathing (N = 8, depths ≤ 10 msw, times ≤ 90 min), or air breathing (N = 16, depths ≤ 10 msw, times from 2 to 6 h). No adverse effects were recorded on all IWR conducted. 10 ep[isodes of joint pain were treated with IWR using air, resulting in 100% immediate relief. Among 10 neurological DCI, 4/4 recovered completely after IWR with $O_2$, whereas only 2/6 subjects obtained immediate recovery after IWR with air.

**Conclusion:** The organization of a training program dedicated to the issue of DCI may reduce mortality and morbidity in precarious communities of fisherman divers. Our results suggest that potential risks due to IWR can be taught and controlled and that IWR with $O_2$ should be used for severe neurological DCI.

**References:**

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**Keywords:** Diving, fisherman divers, decompression sickness, bubbles, hyperbaric oxygen, in-water recompression

## O-29 REDUNDANT $PO_2$ AND $PCO_2$ AND GAS MONITORING SYSTEM FOR REBREATHERS COMPRISING TWO GALVANIC $PO_2$ SENSORS, ONE OPTICAL $PO_2$ SENSOR AND ONE DUAL WAVELENGTH $PCO_2$ SENSOR

A Sieber[1,2,3], A Schuster[1,2], S Reif[1], and P Enoksson[3]

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**Introduction:** Critical parts in closed circuit rebreathers are the gas sensors, as their failure may lead to a non-life-sustaining breathing gas mixture in the loop. Traditional galvanic $O_2$ sensors are unreliable, thus different methods were developed to increase the safety of rebreathers, e.g. voting algorithms and true sensor validation.[1] Scrubber failure may lead to $CO_2$ poisoning, therefore, $CO_2$ monitoring is also of high interest; however; $PCO_2$ sensors for rebreathers are still immature and expensive. Some new technologies are promising, but at an academic level far from commercialization.[2]

**Methods:** A novel sensor system is proposed consisting of two traditional galvanic $PO_2$ sensors and one optical $O_2$ sensor. A separate temperature sensor is used for digital temperature compensation. The galvanic $PO_2$ sensor signals are validated every 2 minutes with a voltametric method [3], which calculates the cell resistance. The optical sensor is based on fluorescing colour pigments. A NDIR $CO_2$ sensor was developed with two optical windows, one at 4 um and one at 4.25 um.

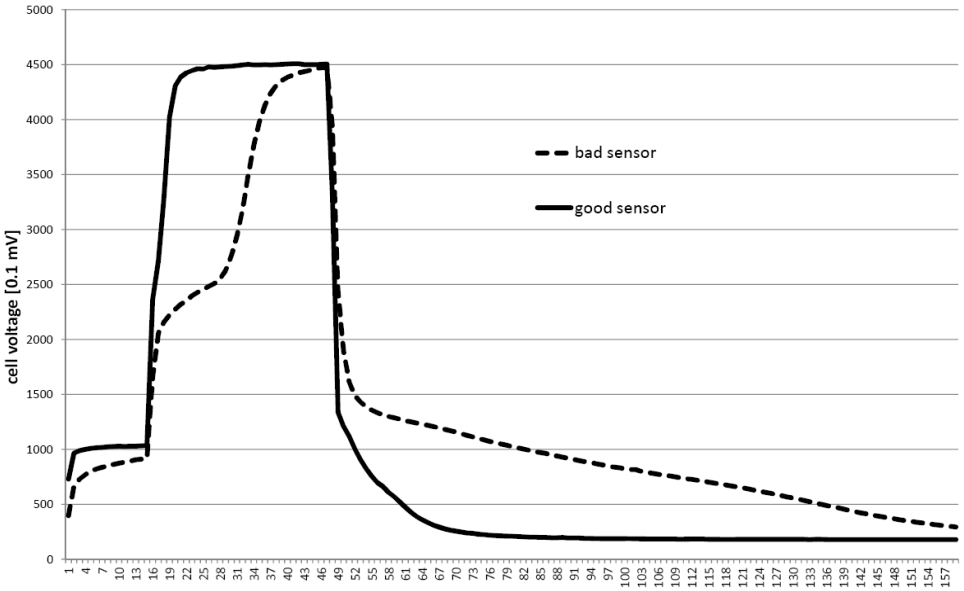


***Figure 1: "Electronic fingerprint" one good and one faulty gaslvanic O2 sensor.***

**Results:** Twoprototypes of the sensor electronics were manufactured. Voltametry allows a characteristic 'fingerprint' of a galvanic sensor to be created. Failure modes of cells result in changes in this fingerprint. Voltametric validation is also able to detect sensor failures that cannot be seen during normal calibration or pressure-pot testing. The optical $PO_2$ sensor was successfully characterized from 0.2 to 1.6 bar $PO_2$. An accuracy of 0.05 bar was achieved even at high $PO_2$s between 1.3 and 1.6 bar. The dual wavelength $PO_2$ sensor is moisture or humidity insensitive.

**Conclusion:** Optical $O_2$ and $CO_2$ sensors are suitable for rebreathers. Electronic voltammetric validation of galvanic sensors is an economic alternative to true validation with gases [1] and voting logic.

**References**

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**Acknowledgment:** The work was co-funded by the EU-MC-ITN PHYPODE, the Austrian Wirtschaftservice and the Center of Applied Technology in Leoben, Austria.

## O-30 AGE-DEPENDENT EFFECTS OF HYPERBARIC HYPEROXIA ON HUMAN PERIPHERAL VASCULAR FUNCTION

T Wunderlich[1], P Radermacher[2], B Winkler[2], W Kähler[1], J Witte[1], and A Koch[1]

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**Objective:** It is known that simulated chamber diving results in acute arterial endothelial dysfunction[1], induced by a decreased availability of endothelial nitric oxide (NO) because of $O_2^-$-production. A common method to evaluate the vasomotor activity is the flow mediated dilatation (FMD) by high-resolution ultrasound of the brachial artery[2]. Little is known about age-dependent effects of hyperbaric hyperoxia on FMD and therefore on endothelial function under hyperoxia.

**Methods:** Twenty-five subjects, separated into two groups, participated in this study. The first group consisted of 15 young, healthy men, (mean age 23 ± 3 years); in the second group, we enrolled 10 patients older than 40 years (mean age: 56 ± 10 years). We assessed the FMD by ultrasound directly prior to and after hyperoxic exposure (Group 1: oxygen tolerance tests, 30 min at 284 kPa oxygen; Group 2: HBOT, 90min at 240kPa oxygen). Continuous measurement of vessel diameter using duplex ultrasound was performed, the peak artery diameter was assessed on a single frame at 30 s and 60 s after deflation of cuff and time to peak diameter as a potential simple marker was recorded also.

**Results:** After hyperoxic exposure the systolic and diastolic FMD decreased non-significantly in Group 1 ($FMD_{sys}$: 13.15 ± 5.26% to 10.2 ± 7.21%, $P$ = 0.169; $FMD_{diast}$: 16.28 ± 6.34% to 13.28 ± 9.43%, $P$ = 0.188) but decreased statistically significantly in Group 2 ($FMD_{sys}$: 12.71 ± 6.35% to 7.33 ± 3.34%, $P$ = 0.004; $FMD_{diast}$: 14.42 ± 8.4% to 8.18 ± 4.87%, $P$ = 0.027). The FMD 30s and 60s after deflation of the cuff remained unchanged in both groups. The time to peak decreased slightly in both groups, but this was not statistically significant.

**Conclusion:** Hyperoxia induces a reduction in brachial artery endothelium function in both young and elderly participants, but FMD decreases more severely in older persons. The reason could be a pre-existing reduced vascular compliance in the elderly.

**References:**

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## O-31 AGE-DEPENDENT EFFECTS OF HYPERBARIC HYPEROXIA ON DIASTOLIC MYOCARDIAL FUNCTION

T Wunderlich[1], W Kähler[1], P Radermacher[2], BE Winkler[2], J Witte[1], and A Koch[1]

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**Objective:** Hyperoxia is known to influence cardiovascular function[1,2], but it is unknown whether there are differences between younger and older persons. The aim of this study was to monitor changes in myocardial diastolic function in younger and elderly volunteers, before and after exposures to hyperbaric hyperoxia.

**Methods:** 22 young, healthy men, (mean age: 24 ± 3 years) and 11 persons older than 40 years (mean age: 56 ± 9 years) were enrolled. The younger (Group 1) were tested for individual oxygen tolerance prior to closed-circuit diving (30 min, 280 kPa oxygen), the older (Group 2) got standard HBO-protocol (90 min, 240 kPa oxygen). We performed echocardiographic examination directly prior and after exposures with focus on diastolic function: E/A-Ratio, e'/a'-Ratio, E/e'-Ratio, deceleration time (DT) and isovolumetric relaxation time (IVRT).

**Results:** After hyperoxic exposures DT significantly increased in Group 1 from 198 ± 25 ms to 219 ± 46 ms ($P$ = 0.048), and in Group 2 from 208 ± 44 ms to 252 ± 44 ms; $P$ = 0.002). E/A-Ratio increased in Group 1 from 1.83 ± 0.49 to 1.91 ± 0.42 ($P$ = 0.256) and remained unchanged in Group 2 (E/A-Ratio: 0.87 ± 0.24 to 0.86 ± 0.21; $P$ = 0.70). E/e'-Ratio slightly decreased in Group 1 (E/e'-Ratio: 3.89 ± 0.94 to 3.65 ± 0.7; $P$ = 0.119) and also in-Group 2 (E/e'-Ratio: 4.53 ± 1.93 to 4.16 ± 1.07; $P$ = 0.83). e'/a'-Ratio, a parameter for cardiac tissue compliance, changed positively in Group 1 from 2.18 ± 0.64 to 2.74 ± 0.75 ($P$ < 0.0001), but only slightly increased in Group 2 from 0.85 ± 0.24 to 0.9 ± 0.39 ($P$ = 0.48). IVRT remained unchanged in both groups.

**Conclusion:** Hyperoxia seems to influence myocardial diastolic function differently in younger and elderly: DT increases more severely in the older person, whereas e´/a´-ratio changes significantly towards better compliance only in the younger. A pre-existing impaired diastolic function in the elderly might be causative.

**References:**

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2. Mak S, Azevedo ER, Liu PP, Newton GE. Effect of hyperoxia on left ventricular function and filling pressures in patients with and without congestive heart failure. *Chest* 2001; 120:467-473

**Keywords:** Hyperoxia, diastolic dysfunction, echocardiography

## O-32 EFFECT OF HYPERBARIC OXYGEN THERAPY ON BLOOD GLUCOSE LEVELS IN PATIENTS WITH DIABETES MELLITUS, BRAIN INJURY AND HEALTHY VOLUNTEERS

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**Introduction**: Hypoglycemia during hyperbaric oxygen therapy (HBOT) is a well known phenomenon. HBOT may increase glucose utilization by skeletal muscle and brain tissue in various animal models [1-3]. However, since data on blood glucose levels (BGL) in patients treated by the common HBOT protocols is limited [4], it is unclear whether HBOT itself induces the decrease in BGL or whether it is due to food deprivation during the treatment.

The aim of this study was to evaluate changes in BGL during HBOT in different subgroups of patients with Type 2 diabetes mellitus (DM), traumatic brain injury (TBI) or post stroke and healthy volunteers in a prospective, randomized cross-over study.

**Methods**: 39 participants were included: 13 patients with DM, 13 patients post stroke or TBI, and 13 healthy volunteers. Measurements of body temperature, blood pressure (BP) and heart rate (HR) were taken in addition to BGL and carbon dioxide levels.
Evaluation was made before HBOT, after one hour, after the completion of 90 minutes exposure to 100% at 203 kPa and at a control session at normobaric environment, breathing room air.

**Results**: A similar decrease ($P$ = 0.59) in BGL was found in all three groups during HBOT (from 131 ± 66 mg/dL to 121 ± 70 mg $dL^{-1}$ ($P$ = 0.037) and at normobaria (from 134 ± 63 mg $dL^{-1}$ to 121 ± 68 mg $dL^{-1}$ ($P$ = 0.004). HR also decreased to a similar extent in both conditions ($P$ = 0.68), from 74 ± 11 bpm to 68 ± 10 bpm,( $P = 6.6*10^{0.07}$) during HBOT and from 72 ± 11 bpm to 69 ± 11 bpm ($P$ = 0.01) during the control session. Diastolic BP decreased (from 74 ± 10 mmHg to 69 ± 11 mmHg $P$ = 0.002) only during the control session and not during HBOT (from 71 ± 9 mmHg to 74 ± 10 mmHg, $P$ = 0.21).

**Conclusions**: BGL may decrease during HBOT and accordingly it should be monitored before entering the chamber. However this decrease in BGL should not necessarily be attributed to the hyperbaric environment.

**References:**

1. Yasuda K, Adachi T, Gu N et al. Effects of hyperbaric exposure with high oxygen concentration on glucose and insulin levels and skeletal muscle-fiber properties in diabetic rats. *Muscle Nerve* 2007; 35:337-43.
2. Badr AE, Yin W, Mychaskiw G et al. Effect of hyperbaric oxygen on striatal metabolites: a microdialysis study in awake freely moving rats after MCA occlusion. *Brain Res*. 2001; 916(1-2):85-90.
1. Lou M, Zhang H, Wang J et al. Hyperbaric oxygen treatment attenuated the decrease in regional glucose metabolism of rats subjected to focal cerebral ischemia: a high resolution positron emission tomography study. *Neuroscience*. 2007; 146(2):555-61.
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**Keywords:** Hyperbaric oxygen, blood glucose, prospective control trial, blood pressure, heart rate

## O-33 HYPERBARIC OXYGEN THERAPY INCREASES INSULIN SENSITIVITY IN OVERWEIGHT MEN WITH AND WITHOUT TYPE 2 DIABETES.

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**Introduction:** In patients who have type 2 diabetes, the clinical use of hyperbaric oxygen therapy (HBOT) increases insulin sensitivity. However, whether this occurs in a non-patient population with and without type 2 diabetes is unknown, along with the mechanism of this effect.

**Methods:** Insulin sensitivity was assessed by hyperinsulinemic euglycemic clamp in overweight and obese men without (n = 11) or with type 2 diabetes (n = 8) at baseline and during the third HBOT. Fasting serum and adipose tissue samples were collected at baseline and after 4 HBOT.

**Results:** In response to HBOT, insulin sensitivity was significantly increased by 29 ± 32% in those without, and by 57±66% in those with diabetes. This increase was maintained for at least 30-minutes after exit from the hyperbaric chamber. Reductions in serum inflammatory markers MCP-1 and TNF-α were observed after 4 days of HBOT, while IL-6 and IL-1ra were increased. The increase in IL-6 correlated with the increase in insulin sensitivity ($r^2$ = 0.36, $P$ = 0.02).

**Conclusion:** Insulin sensitivity was increased by HBOT in individuals with and without type 2 diabetes and this effect was maintained for at least 30-minutes after exit from the hyperbaric chamber. Changes in inflammatory cytokines may partly explain this effect.

**Keywords:** Hyperbaric oxygenation, insulin resistance, adipose tissue, inflammation.

## O-34 THE EFFECTS OF HYPERBARIC OXYGEN ON CULTURED HUMAN BREAST CANCER CELLS

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**Introduction**: The effects of hyperbaric oxygen (HBO) on tumour cells have been controversial (1). The incidence of breast cancers is higher than the other malignancies in women. Radiotherapy and chemotherapy are conventional approaches to cancer treatment. HBO is used as an adjunctive treatment of radionecrosis (2) and extravasation of certain chemotherapeutics. In this study, the effects of HBO on cultured human breast cancer cells were investigated.

**Materials and methods:** Cultured Michigan Cancer Foundation-7 (MCF-7) cells were divided into five groups and named as; G-INC: The group kept in incubator; G-NBA: The group treated with normobaric air; G-HBA: The group treated with hyperbaric air; G-NBO: The group treated with normobaric oxygen and G-HBO: The group treated with hyperbaric oxygen. In the first step of the experiment, by using trypan blue staining the numbers of the cells and vitality analysis in each group were compared. In the second step proliferation indices by deoxyribonucleic acid synthesis were evaluated by working with bromodeoxyuridine. Hyperbaric sessions were done at 253 kPa.

**Results:** The number of cells in group G-HBO was significantly lower when compared to the groups G-INC and G-HBA ($P < 0.001$). In the vitality analysis, the number of viable cells was significantly lower in group G-HBO compared to the group of G-HBA ($P = 0.041$). The cell proliferation index in group G-HBO was smaller than all the other groups.

**Conclusion:** In this in vitro experimental study HBO decreased the number of cells in MCF-7 cell culture and suppressed deoxyribonucleic acid synthesis.

**References**:

1. Feldmeier J, Carl U, Hartmann K, Sminia P. Hyperbaric oxygen: does it promote growth or recurrence of malignancy? *Undersea Hyperb Med*. 2003; 30(1):1-18.
2. Hampson NB, Holm JR, Wreford-Brown CE, Feldmeier J. Prospective assessment of outcomes in 411 patients treated with hyperbaric oxygen for chronic radiation tissue injury. *Cancer* 2012; 118(15):3860-8

**Keywords:** Hyperbaric oxygen, breast cancer, vitality analysis, proliferation index, cell culture

## O-35 LOW HYPERBARIC PRESSURE MODULATES $Ca^{2+}$ INFLUX VIA VOLTAGE DEPENDENT $Ca^{2+}$ CHANNELS IN CULTURED CORTICAL NEURONS

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**Introduction**: The phenomenon of sensory, motor, and cognitive impairments when diving to depths greater than 100 msw (1.1 MPa) is known as the high pressure neurological syndrome (HPNS). An increased transfer function between synaptic inputs and somatic spike generation was found in rat brain slices at high pressure (HP). Modulations of the $Ca^{2+}$ influx through voltage dependent $Ca^{2+}$ channels (VDCCs) at HP could potentially lead to some of the HPNS signs and symptoms.[1]

**Methods**: Primary rat cortical neurons were cultured for 19-30 day and then incubated for 1 hour in CSF with 10 μM Fura2-AM. A dual 340/380 nm excitation procedure was performed with a Nikon microscope and an electrical stimulation was delivered to the culture while acquiring ratiometric measurements by a cool-snap camera. Compression by helium was performed at 0.04 MPa $min^{-1}$. Measurements were taken at 22 ± 0.5°C.

**Results**: The immediate maximal rise in $[Ca^{2+}]_i$ following an electrical stimulation was augmented already at 0.3 MPa (20 msw) by 25 ± 5% in somata, and by 22 ± 5% in dendrites (n = 9, $P < 0.01$). This augmentation subsided after a few minutes in 7 of 9 cultures and reached a steady state of 7-10% at 20-25 minutes post compression. The baseline $[Ca^{2+}]_i$ seems to be elevated at HP, and did not recover upon decompression. More cells reacted to the stimulus under HP conditions.

**Discussion**: HP transiently increased the $Ca^{2+}$ influx. A similar transient increase of action potential $Na^+$ current was described in lobster bifurcating axon.[2] The overall HP effect shown here is most probably a combination of HP modulations of several VDCC types expressed in the culture, the prominent one likely to be L-type $Ca_V1.2$, which was suggested to be augmented by HP.[1]

**Conclusion:** These results suggest unexpectedly high sensitivity of cortical neurons to relatively low HP exposure.

**References:**

1. Aviner B, Mor M, et al. Hyperbaric pressure facilitates barium currents in L-type Ca2+ channel. In: Proceedings European Undersea Baromedical Society, 34th Annual meeting, Graz, Austria. 2008. p. 30-33.
2. Grossman, Y, Kendig JJ. Pressure and temperature modulation of conduction in a bifurcating axon. *Undersea Biomed Res* 1986; 13(1):45-61.

**Keywords:** High pressure neurological syndrome; voltage dependent calcium channels, $Ca^{2+}$ imaging, cortical neuronal culture

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## O-36 EFFECTS OF NORMOBARIC HYPEROXIA ON THE ACTIVITIES OF COMPLEX I AND II OF THE MITOCHONDRIAL RESPIRATORY CHAIN IN DIFFERENT LEUKEMIC CELL LINES

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**Background:** Recent work suggests that the main mechanism behind the reduced mitochondrial respiratory capacity after exposure to normobaric hyperoxia is a defect of cytochrome c reductase (complex III of the mitochondrial respiratory chain).[1] In that case, one would expect that the activity of complexes I and II, which are situated proximal to complex III, should be reduced to a similar degree regardless of the cell type studied. Therefore, we investigated the mitochondrial respiratory activity of two different leukemic cell lines (Jurkat and CCRF-SB) after exposure to normobaric hyperoxia.

**Methods:**The respiratory activity of both cell types was measured after exposure to air + 5% $CO_2$ at 37°C ; 18 h hyperoxia (60% $O_2$, 5% $CO_2$, 37°C) or 18 h hyperoxia + 6 h normoxic conditions using an $O_2$k-Oxygraph (OroborosInstruments, AT). Mitochondrial respiration was then quantified in pmol $O_2$/sec*$10^6$ cells in the uncoupled state after permeabilisation of the cells and addition of malate (5 mM) and glutamate (10 mM) for complex I. Addition of succinate achieved a combined complex I and II activity state. Selective complex II activity was determined by blocking complex I by addition of rotenone (0.5 µM).

**Results:** Our results show a significant reduction in separate and combined complex I+II activity (60% of control value) directly after hyperoxia, independent of the cell line. The mitochondrial activity decreases further in Jurkat cells to 28% after the 6-h normoxic interval. While complex II activity stays impaired in CCRF-SB, complex I starts recovering, leading to a return to 76% of control value in combined complex I and II activity after 6 h of recovery.

**Conclusion:** Since the two cell lines under stimulation of selective complex I and/or II do not respond identically with respect to their recovery period after normobaric hyperoxia exposure, reduction of complex III activity cannot be the only explanation for the impaired mitochondrial respiration.

**References:**

1. Tatarkova Z, Engler I, Calkovska A et al Effect of normobaric oxygen treatment on oxidative stress and enzyme activities in guinea pig heart. *Gen Physiol Biophys* 2012; 31:179-84.

**Keywords:** Leukemia, normobaric hyperoxia, mitochondrial respiratory chain

This study is part of the Phypode Project, financed by the European Union under a Marie Curie Initial Training Network programme.

## O-37 EFFECT OF NORMOBARIC OXYGEN BREATHING ON HUMAN COGNITIVE FUNCTION

W Hemelryck[1,2,3], C Balestra[2,3], V Papadopoulou[2,4], M Rozloznik[2], P Germonpré[1,2,3], and P Lafère[1,2,3]

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**Introduction:** Brain executive functions are important for successful adaptation and performance in real-life situations. Since environment can be unpredictable, executive functions are vital for humans to recognize the significance of unexpected situations and to make alternative plans quickly. However, an objective measurement of neurophysiological parameters remains challenging. Critical flicker fusion frequency (CFFF) and the Psychology Experiment Building Language (PEBL) battery tests provide valid and versatile research tools for measuring brain function and allow the detection of modifications of brain performance and, as such, can be used in the field of diving experimentation.

**Methods:** Twenty, male, non-smoking sports students (mean age 25 ± 6.6 years), body composition (BMI 22.8 ± 2.0) were tested with the CFFF and three tests selected from the Psychology Experiment Building Language (PEBL) battery to track deterioration in visual-perceptual organization, visual-motor coordination and integration, and visual memory. The experiment consisted of two randomly assigned runs, one with air (21% $O_2$) and the other after 10 minutes of 100% $O_2$ breathing.

**Results:** Following the breathing of oxygen, the number of errors made by each participant was significantly reduced in all three PEBL tests: Perceptual vigilance task ($P$ = 0.03); trail-making task ($P$ = 0.04); Math-processing task ($P$ = 0.03). An increase in CFFF of 21.46 ± 15.31% was also statistically significant.

**Conclusion:** We hypothesize that the main reason for these effects depends on exposure to higher $O_2$ partial pressure, but precise mechanisms are still debated. Further studies, including wet and dry dives, are needed to understand the $O_2$ effect on brain function in humans at pressure.

**Keywords:** CFFF, neuropsychometric, oxygen, normobaric, brain

This study is part of the Phypode Project, financed by the European Union under a Marie Curie Initial Training Network Program

## Friday, September 27th, 2013

**Tricontinental Scientific Meeting, Special Session "Young Investigators – PHYPODE"**
*Location: "Coquillage" room, Tamarun*

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|---|---|---|---|
| | **Special Session: Young Investigators – PHYPODE – "Treatment of DCI"**<br>**Chair: Costantino Balestra** | | |
| 14:00 | | Clinical decision algorithm for treatment of DCI cases<br>*Martin Sayer - Colin Wilson* | |
| 14:40 | | Use of different gas mixes for the treatment of DCI<br>*Jacek Kot* | |
| 15:00 | | Use of adjunctive medication in the treatment of DCI<br>*Mike Bennett* | |
| 15:40 | **Coffee / Tea** | | |
| 16:00 | | DCI management in remote geographical areas - compromise between cost/efficiency (including IWR strategy or guidelines)<br>*Jack Meintjes* | |
| 16:40 | | Evidence base for the use of different recompression profiles used in DCI treatment<br>*Jorg Schmutz – Marco Gelsomino* | |
| 17:20 | **Adjourn** | | |

**18:30 Tricontinental Scientific Meeting - Conference Dinner**
*Location: Hotel LUX* Les Villas du Lagon*

## Saturday, September 28th, 2013

**Scientific Societies (EUBS, SPUMS, SAUHMA) Annual General Meetings**
*Location: "Le Voilier", "Nacre", "Coquillage" rooms, Tamarun*

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| 09:00 | **AGMs of EUBS, SPUMS, SAUHMA** | |

**Tricontinental Scientific Meeting, Day Four**
*Location: "Le Voilier" room, Tamarun*

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| 10:00 | | **Keynote Lecture:** | | |
| | | J Kot | BIOCHEMICAL MARKERS OF PULMONARY OXYGEN TOXICITY | |
| | **Session 7: Hyperbaric Oxygen Therapy** | | **Chair: Jonathan Rosenthal** | |
| 10:20 | O-38 | M Davis | HYPERBARIC OXYGEN THERAPY FOR ACUTE CENTRAL RETINAL ARTERY OCCLUSION: A CASE SERIES | |
| 10:40 | O-39 | D Smart | TREATMENT OF LATE SOFT TISSUE RADIATION INJURY (LSTRI) – A SYSTEMATIC REVIEW OF THE LITERATURE INCLUDING HYPERBARIC OXYGEN TREATMENT (HBOT) | |
| 11:00 | O-40 | R Boussi-Gross | HYPERBARIC OXYGEN THERAPY FOR CHRONIC COGNITIVE IMPAIRMENT DUE TO TRAUMATIC BRAIN INJURY- RANDOMIZED PROSPECTIVE TRIAL | |
| 11:20 | O-41 | D Smart | MISUSE OF EVIDENCE BASED MEDICINE TO JUSTIFY REMOVAL OF HEALTHCARE FUNDING. AUSTRALIAN EXPERIENCE WITH HEALTH TECHNOLOGY ASSESSMENT AND HBOT | |
| 11:40 | O-42 | S Efrati | HYPERBARIC OXYGEN AMELIORATES FIBROMYALGIA SYMPTOMS AND FUNCTIONAL IMPAIRMENT – RANDOMIZED PROSPECTIVE TRIAL | |
| 12:00 | O-43 | D De Bels | HYPEROXIA ALTERS THE ULTRASTRUCTURE OF LEUKEMIA CELL LINES AND INDUCES APOPTOSIS | |
| 12:20 | **Lunch** | | | |

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|---|---|---|---|---|
| | **Session 8: Diving Medicine** | | **Chair: Alf Brubakk** | |
| 13:40 | O-44 | I Eftedal | APPLYING GENOMICS TOOLS IN STUDIES OF BAROPHYSIOLOGY | |
| 14:00 | O-45 | K Hansen | AN MRI/CT/PET-COMPATIBLE PRESSURE CHAMBER FOR BAROMETRIC RESEARCH – DEVELOPMENT AND VALIDATION | |
| 14:20 | O-46 | A Bliznyuk | HYPERBARIC PRESSURE EFFECTS ON NMDA RECEPTOR VARIANTS | |
| 14:40 | O-47 | D Cialoni | GENETIC PREDISPOSITION TO ACUTE RESPIRATORY SYMPTOMS IN BREATH-HOLD DIVERS | |
| 15:00 | **REUNION2013 Closing Ceremony** | | | |
| 15:20 | **Happy Hour @ Tamarun** | | | |

## O-38 HYPERBARIC OXYGEN THERAPY FOR ACUTE CENTRAL RETINAL ARTERY OCCLUSION: A CASE SERIES

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**Introduction:** Central retinal artery occlusion (CRAO) is a major cause of sudden, painless visual loss, often leaving no useful vision in the affected eye. Its incidence is cited at 0.85 per 100,000 persons per year but may be higher because of under-reporting.[1] The natural history is difficult to study, but a spontaneous resolution rate of <1–8% for acute, non-arteritic CRAO has been cited.[1] Occurrence in an only eye is devastating for the patient. There is currently no consensus regarding management of CRAO and little evidence to support any treatment modality. Despite only limited case series, hyperbaric oxygen therapy (HBOT) has been recommended for CRAO recently by the Undersea and Hyperbaric Medical Society (UHMS).[2]

**Methods:** Between early 2003 and December 2012, all CRAO patients presenting to Christchurch Hospital were referred for consideration of HBOT. These 31 consecutive patients' medical records were reviewed retrospectively. The time delay from onset of visual loss to commencing HBOT; the presenting visual acuity; various demographic data; the HBOT administered and the outcome visual acuity were documented.

**Results:** All 31 patients underwent at least one HBOT (median 4, range 1–7), either at 203 or 243 kPa for 1.5 to 2.0 h. One patient's treatment was terminated after 60 min at their request; another declined further HBOT and one suffered middle ear barotrauma. Thirteen patients also received anticoagulants at the discretion of the referring ophthalmologist. Twenty-three patients had improved vision with the first HBOT but this was only transient in 14. Seven patients had good, permanent visual recovery (6/18 or better; Snellen chart); and two had only modest improvement (6/60). Patients appeared more likely to improve if treated within nine hours of symptom onset.

**Conclusions:** Where available, HBOT is indicated for CRAO. Our protocol may not have been aggressive enough and the UHMS protocol is recommended. A multi-centre, randomised controlled trial is warranted.

**References:**

1. Fraser SG, Adams W. Interventions for acute non-arteritic central retinal artery occlusion. *Cochrane Database of Systematic Reviews* 2009, Issue 1. Art. No.: CD001989. DOI: 10.1002/14651858.CD001989.pub2
2. Murphy-Lavoie H, Butler F, Hagan C. Central retinal artery occlusion treated with oxygen: A literature review and treatment algorithm. *Undersea Hyperb Med* 2012; 39(5):943-953.

**Keywords:** Acute retinal artery occlusion, hyperbaric oxygenation, case series

## O-39 TREATMENT OF LATE SOFT TISSUE RADIATION INJURY (LSTRI) – A SYSTEMATIC REVIEW OF THE LITERATURE INCLUDING HYPERBARIC OXYGEN TREATMENT (HBOT)

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**Introduction:** Late soft tissue radiation injury (LSTRI) is a distressing complication experienced by a minority of individuals who successfully recover from radiation treatment for cancer.

**Aim:** To identify the various surgical and non-surgical treatments (including HBOT) for LSTRI and evaluate evidence for their efficacy, using a hierarchy as described by the NH&MRC.

**Methods:** The search strategy aimed to identify all papers documenting treatment of LSTRI (April 1984 - April 2009). The search identified randomized and pseudo-randomized controlled trials (RCT's), and comparative trials comparing the effect of LSTRI treatment, with other treatments including placebo. LSTRI was defined as "*all adverse effects of radiation affecting tissues other than bone, commencing greater than one month after completion of radiotherapy*". Databases searched were: CENTRAL (Cochrane April 2009), MEDLINE, EMBASE, CINAHL, the DORCTHIM (Bennett 2004) and Google Scholar. Relevant controlled trials in hyperbaric literature sources and hyperbaric textbooks published since 1984 were also searched.

**Results:** The review identified 28 RCT's assessing treatment of LSTRI (including surgical intervention), that produced positive outcomes for patients. Treatments included acupuncture, aspiration, fenestration, myringotomy and grommets, resistance training, mechanical massage, acupuncture, gel products, fluoride and pilocarpine for xerostomia, surgical treatment of carotid blowout syndrome, intravesical placental extract, flavoxate and formalin, sucralfate, hydrocortisone, heater probes, argon plasma coagulation, short chain fatty acids, metronidazole and anti-inflammatories, alpha tocopherol, surgical dorsi flaps, and HBOT. HBOT showed positive benefit for tooth socket healing, surgical flap repairs in the head and neck, xerostomia, chest wall injury, head and neck radiation injury, pelvic radiation injury, radiation proctitis and cystitis.

**Conclusions:** The paucity of published research indicates the rarity of LSTRI and the difficulty of undertaking quality research. Treatments were anatomically specific and not comparable. HBOT was the only intervention that allowed meta-analysis. A consistent finding for HBOT was a positive influence on wound and mucosal healing, and a positive benefit when used in support of other treatments, such as surgical intervention.

**Keywords:** Systematic review, randomized controlled trial, late soft tissue radiation injury, treatment, hyperbaric oxygen treatment

## O-40 HYPERBARIC OXYGEN THERAPY FOR CHRONIC COGNITIVE IMPAIRMENTS DUE TO TRAUMATIC BRAIN INJURY- RANDOMIZED PROSPECTIVE TRIAL

R Boussi-Gross[1], H Golan[3]. G Fishlev[1], Y Bechor[1], O Volkov[3], J Bergan[1], M Friedman[1], E Ben-Jacob[2,4,5] & S Efrati [1,2]

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**Introduction**: A common sequela of mild traumatic brain injury (mTBI) is the so called postconcussion syndrome (PCS), a complex of symptoms that includes neuropsychiatric symptoms, and cognitive impairment. Even though the majority of patients will recover, 9-25% will have persistent symptoms.[1-3] In these patients hypoxia in the damaged brain tissue plays a major role in the impaired regeneration/healing processes.[4] Recently, we reported that hyperbaric oxygen therapy (HBOT) can induced neuroplasticity in the chronic phase of post-stroke patients.[5] The aim of this study was to evaluate the effect of HBOT on cognitive impairments and brain metabolism in chronic mTBI patients in a prospective, controlled, randomized, cross-over study.

**Methods:** The study included 90 patients who suffered from mTBI, 1-6 years prior to inclusion, and had complaints regarding their cognitive function. Patients were randomized into two groups: a treated group and a cross group. The patients in the treated group were evaluated twice: baseline and after HBOT. Patients in the cross group were evaluated three times: baseline, after control period of no treatment, and after HBOT. The HBOT protocol was: 40 sessions, 5 days/week, 90 minutes, 100% oxygen at 1.5ATA.
The primary end points included neuropsychological function (Mindstreams testing battery), and brain metabolism, evaluated by SPECT. Secondary end point included quality of life evaluation. Evaluations were made by medical and neuropsychological blinded to patients' group.

**Results:** Following HBOT a significant improvement in all cognitive measures (memory, executive function, attention and information processing speed) as well as quality of life was observed in both groups after HBOT ($p<0.005$ for all). No improvement was noticed in the crossed group during the control period.
Concomitantly, a significant improvement in brain metabolism was also demonstrated in the brain SPECT evaluation.

**Conclusion**: HBOT may induce significant neuroplasticisty and improve cognitive function in patients with mTBI even years after the acute injury.

**References**

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**Keywords**: Hyperbaric oxygen, traumatic brain injury, prospective randomized control trial.

## O-41 MISUSE OF EVIDENCE BASED MEDICINE TO JUSTIFY REMOVAL OF HEALTHCARE FUNDING. AUSTRALIAN EXPERIENCE WITH HEALTH TECHNOLOGY ASSESSMENT AND HBOT

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**Introduction:** Health Technology Assessments (HTAs) are promoted as an appropriate methodology to evaluate health technology and allocate healthcare funding. In Australia, this role is performed by the Medical Services Advisory Committee (MSAC), who advise the Federal Minister for Health and Ageing on whether a new medical service should be publicly funded.

**Objective:** To outline the Australian experience where evidence based medicine was misused with the specific intent of controlling health funding.

**Results and Discussion:** HBOT has been funded since the Australian Medicare Benefits Schedule (MBS) started in the 1980's, and treatment prescription was at the discretion of the specialists who worked in the field. In 1999, a monoplace hyperbaric chamber manufacturer applied to MSAC for funding. MSAC launched a succession of reviews of HBOT, contrary to its brief that it was set up to review new technologies NOT existing funded technology. The Australian Federal Government withdrew funding from HBOT for all but seven medical conditions. After MSAC's third review in 2012, funding was withdrawn for HBOT of non-diabetic problem wounds (NDW). MSAC's HBOT reviews cost AUD1 million, exceeding the national annual HBO budget for NDW!

**Conclusion:** MSAC had no process for evaluating funded treatments. Biased methodology allowed MSAC to isolate a single treatment for focused critique without evaluating the evidence for all treatments available to the relevant patient population. MSAC provided no evidence that any alternative treatment was more effective than HBOT and ignored poor outcome data for standard care of NDW. Key flaws in processes (including alteration of report wording without consent) were not addressed by MSAC; there is evidence that MSAC intends to generalize their methodology to disinvest in the MBS. Of major concern is that the same flawed processes will be taken up by other countries for HTAs.

**Key words**: Health technology assessments, problem wounds, hyperbaric oxygen treatment, health funding

## O-42 HYPERBARIC OXYGEN AMELIORATES FIBROMYALGIA SYMPTOMS AND FUNCTIONAL IMPAIRMENT – RANDOMIZED PROSPECTIVE TRIAL

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**Introduction:** Fibromyalgia syndrome (FMS), considered to represent a prototype of central nervous system sensitization, is a common condition characterized by chronic widespread pain and diffuse tenderness, along with symptoms of fatigue.[1,2] Hyperbaric oxygen (HBOT) has the capacity to induce neuroplasticity in different chronic brain pathologies.[3] The current study evaluated the utility of HBOT for influencing central sensitization as a therapeutic modality for FMS.

**Methods:** A prospective, randomized, controlled trial was conducted of 60 female patients, aged 21-67 years, diagnosed with FMS for at least 2 years. Patients were randomized into two groups: a treated group and a cross (control) group. The patients in the treated group were evaluated twice: at baseline and after HBOT. Patients in the cross group were evaluated three times: baseline, after control period of no treatment, and after HBOT. The following HBOT protocol was practiced: 40 sessions, 5 days a week, 90 minutes of 100% oxygen breathing at 203 kPa. At each time point, level of pain was evaluated by physical examination including tender point count and dolorimetry, as well as extensive evaluation of parameters relating to quality of life, presence of widespread pain, fatigue, physical and social dysfunction, and symptoms related to anxiety, depression, and somatization.

**Results:** A significant reduction of all FMS symptoms such as pain (threshold and number of tender points) and fatigue was apparent following the HBOT sessions as well as significant improvement of distress symptoms. Moreover, the quality of life and functional capabilities of all patients were significantly improved following the HBOT sessions. No improvement in any of the parameters was found during the control period of the patients in the cross group.

**Conclusions:** The results indicated that HBOT can lead to significant improvement in all FMS symptoms as well as significant improvement in patients' quality of life.

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**Keywords:** Hyperbaric oxygen, fibromyalgia, prospective randomized control trial.

## O-43 HYPEROXIA ALTERS THE ULTRASTRUCTURE OF LEUKEMIA CELL LINES AND INDUCES APOPTOSIS

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**Introduction:** Cancer cells are hypermetabolic cells with large oxygen requirements. Oxygenation conditions are also crucial for growth and tumoUr progression. Recent data suggested a decrease in cancer cell proliferation secondary to normobaric hyperoxia both in vitro and in vivo as well as fractal dimension change after hyperoxia in breast cancer cells. The purpose of this research WAs to study the impact of hyperoxia on apoptosis and morphologic changes of leukemia cell lines.

**Material & methods:** Three hematopoietic cancer cell lines (one myeloid, U937 and two lymphoid, Jurkat and SUP-T1) were tested under conditions of normobaric hyperoxia (> 60% $O_2$, ± 18h vs 21% $O_2$, ± 18h) and compared to their normoxic control. We tested apoptosis using an Annexin V binding assay and then a Caspase-3 immunoblot. We examined cell morphology by cytospin, microphotography after coloration and analysis by a fractal software.

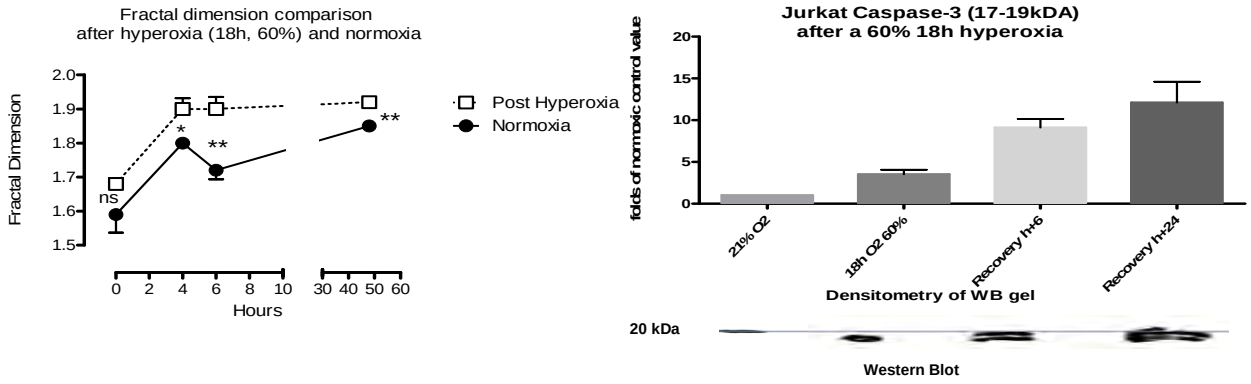


**Figure 1.** A: Variation of JURKAT cells fractal dimensions after an 18-hour hyperoxic exposure as compared to normoxia. Cells are in the same culture medium and are followed during 48 hours. B. Variation of Caspase-3 expression of JURKAT cells after an 18-hour hyperoxic exposure as compared to normoxia.

**Results:** Exposure of cell cultures to transient normobaric hyperoxia induced early apoptosis (elevated caspase-3) as well as a significant and precocious modification in cell shape, as demonstrated by Fractal dimension (FD) increase. Their behavior was correlated to the change in shape.

**Conclusions:** Such morphological alterations could be due to several molecular mechanisms and rearrangements in the cancer cell leading to cell cycle inhibition and very early apoptosis as shown by caspase-3 activity.

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**Keywords:** Hyperoxia, apoptosis, leukemia, fractal dimension

This study is part of the Phypode Project, financed by the European Union under a Marie Curie Initial Training Network Program, the Belgian Lottery and the Wallonie-Bruxelles Federation.

## O-44 APPLYING GENOMICS TOOLS IN STUDIES OF BAROPHYSIOLOGY

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**Background:** High throughput molecular analysis and bioinformatics are valuable tools for investigating the biological basis of physiological traits.When applied in studies of barophysiology, these tools may provide helpful information for prevention and treatment of conditions such as decompression illness (DCI).

**Methods:** We have used gene expression profiling, protein detection techniques and in-silico prediction of transcriptional pathways and biological processes to characterize responses to diving in the rat aorta and human blood. Vascular gas bubbles[1] and cardiovascular parameters[2,3] were measures of physiological stress.

**Results:** Gene expression profiling of the rat aorta after diving revealed increased expression of genes involved in acute responses to oxidative stress. Rats with no detectable bubbles and rats with high bubble loads had similar gene expression patterns, indicating that elevated oxygen tensions ($PO_2$) rather than bubbles instigated the responses. Expression of the plasminogen activator inhibitor protein (PAI1) was highly increased after diving. PAI1 is a major physiological inhibitor of fibrinolysis in blood and a biomarker for cardiovascular disease, and increased PAI1 levels could signify procoagulant development[3]. Results from gene expression profiling of blood from human divers supported the involvement of elevated $PO_2$ over that of vascular bubbles in acute genetic responses to diving. The human results also point to possible long-term changes in processes of coagulation and inflammation (data not published) but in the absence of clinical DCI symptoms any harmful effects of the observed gene expression changes remain uncertain.

**Conclusion:** Through gene expression profiling verified by protein analyses, we have shown that a high $PO_2$ during diving specifically triggers oxidative stress-responsive genes. Putative involvement of biological processes was predicted by bioinformatic techniques. The identified genes and biological processes may provide targets for further studies of prevention and treatment of diving-related pathology.

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**Keywords**: Biological processes, coagulation, diving, gene expression profiling, inflammation

## O-45 AN MRI/CT/PET-COMPATIBLE PRESSURE CHAMBER FOR BAROMETRIC RESEARCH – DEVELOPMENT AND VALIDATION

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**Background:** Barometrical research carries considerable experimental challenges because the model animal is located within a pressure chamber. Conventional pressure chambers are constructed from tough materials to sustain the inner pressure. Magnetic resonance imaging (MRI), computed tomography (CT) and positron emission tomography (PET) are valuable diagnostic tools in the clinic because they non-invasively visualize hard- and soft tissue anatomy and provide several important physiological parameters. The purpose of this study was to develop and validate a preclinical rodent-size imaging-compatible pressure chamber system.

**Methods:** A pressure chamber (range 0.1 – 11 ATA) was constructed from nonmagnetic materials (Fig. 1). The effect of hyperbaric conditions on the scanning signal was investigated using phantoms: for MRI, vials of $MnCl_2$ (0-3.2 mM) in 10 mM HCl; for CT, different materials (cylinders, 5 x 2 cm); and for PET, two 35 mL vials containing demineralized water enriched with fluorine isotope ($^{18}$F-FDG). Phantoms were then scanned at 1, 3, 6, 8 and 11 ATA with either a 3.0 T Siemens MRI system or General Electric PET/CT system. $T_1$- and $T_2$-weighted MRI sequences and standard clinical CT/PET protocols were used.

**Results:** Measurements of the signal intensity, standard deviation of the mean signal, and the distributed signal profile (histogram) in respective phantoms revealed that hyperbaric conditions *per se* do not alter the MRI, CT or PET- signal. Also, we aim to present preliminary data from pressurized rodents.

**Discussions:** The underlying physiology during and following hypo/hyperbaric conditions is poorly understood. The developed system allows for uncompromised MRI/CT/PET acquisition during hypo/hyperbaric exposure, and we believe that this system can become valuable for barometric research.

**Key words:** MRI, CT, PET, scanning, pressure chamber

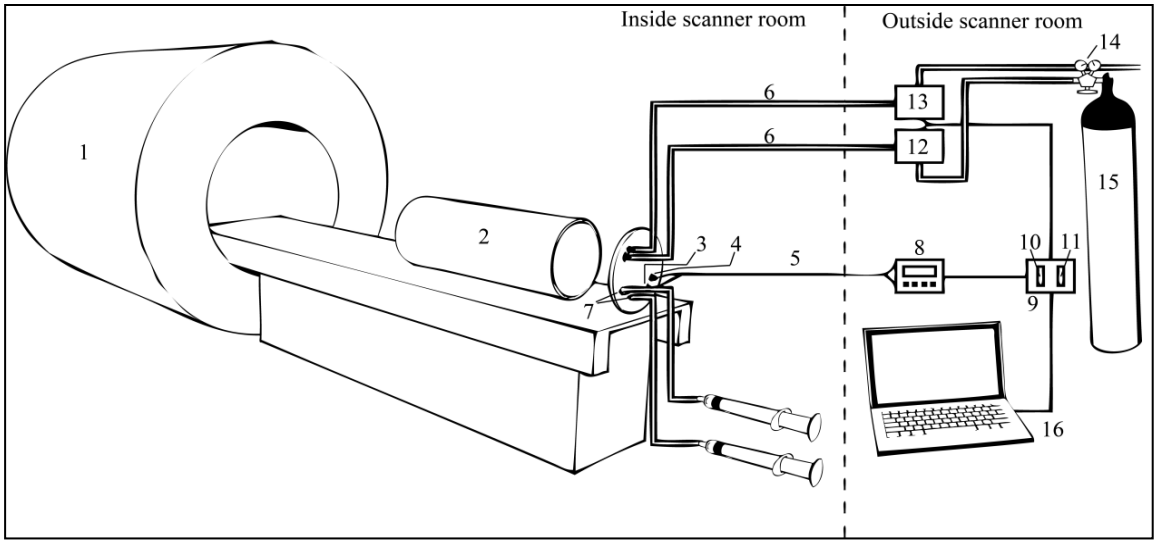


**Figure**. **1.** Imaging system (MRI, CT or PET), **2**. Pressure chamber (0.1-11 ATA), **3**. Fiber optical pressure sensor, **4**. Fiber optical temperature sensor (deep body-core temperature is used in a feedback temperature regulation loop), **5**. Fiber optical cables (7 m), **6**. High-pressure gas tubes (7 m), **7**. Cable- and catheter pressure-ports, **8**. and **9.** Signal converter, **10**. Data acquisition interface, **11**. Valve-control interface, **12**. and **13.** Proportional solenoid valves for gas-in- and outlet, **14**. Pressure reducing valve, **15**. Gas tank, **16**. Computer system (pressure profile execution and data acquisition).

## O-46 HYPERBARIC PRESSURE EFFECTS ON NMDA RECEPTOR VARIANTS

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**Introduction:** Professional divers may suffer from direct high pressure (HP) effects. Various animals and humans exposed to ambient pressure above 1.1 MPa develop the high pressure neurological syndrome (HPNS). The glutamate N-methyl-D-aspartate receptor (NMDAR) has been implicated with CNS hyperexcitability as part of the HPNS. Furthermore, NMDAR subunit GluN1 subtypes were implicated with selective pressure effects (Mor et al. 2012). In order to explore this possibility we studied HP effects directly on four specific alternatively spliced GluN1 subtypes.

**Methods:** Newly synthesized GluN1(1a,1b,2a or 2b) were co-expressed with GluN2A subunits in *Xenopus laevis* oocytes. Ionic currents ($Ca^{2+}$ was substituted for by $Ba^{2+}$) were measured by two-electrode voltage clamp, in response to bath application of the co-agonists Glutamate (100 μM) and Glycine (10 μM) at helium pressures of 0.1, 5.0 or 10.1 MPa.

**Results:** All the subtypes' ionic currents were increased by HP (table 1); GluN1-1b+GluN2A currents were increased in contrast to our previous report (above).

| Subunit composition | Amplitude (nA) 0.1MPa mean ± SEM | Amplitude (nA) 5.0 or 10.1MPa mean ± SEM | Amplitude(%Δ) 10.1/0.1MPa mean ± SEM | n | *P*-value |
|---|---|---|---|---|---|
| GluN1-1a + GluN2A | 2,186.39±638.14 | 4,362.45±1164.22 | 101.11±5.45 | 2 | 0.151 |
| GluN1-1b + GluN2A | 2,226.88±256.11 | 3,424.04±410.31 | 52.51±9.07 | 9 | < 0.001 |
| GluN1-2a + GluN2A | 2,103.28±313.58 | 2,899.94±390.69 | 39.37±6.63 | 5 | 0.003 |
| GluN1-2b + GluN2A | 2,943.40±560.81 | 3,809.22±489.74 | 32.49±8.52 | 3 | 0.027 |

**Table 1.** Statistical analysis of NMDAR currents at HP. Maximal current amplitude under control (0.1MPa) and HP conditions (5.0 or 10.1 MPa), and mean % change ± SE of amplitude (calculated for each pair of measurements and averaged), n, number of experiments (oocytes), p, degree of statistical significance: Statistical tests: paired t-test (0.1 MPa vs.5.0 or 10.1 MPa).

**Conclusion:** We are currently exploring possible reasons for this finding including $Ca^{2+}$/ $Ba^{2+}$ substitution, time dependence of HP effects, compression protocol, or spontaneous error in cRNAs sequence. Four additional subtypes will be studied in order to verify (or reject) selective HP effects on the GluN1 subtypes.

**Key words:** High pressure neurological syndrome (HPNS), NMDA receptors, *Xenopus* oocytes.

## O-47 GENETIC PREDISPOSITION TO ACUTE RESPIRATORY SYMPTOMS IN BREATH-HOLD DIVERS

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**Introduction:** Non-cardiogenic acute pulmonary edema is a syndrome that has been occasionally observed in healthy subjects engaged in swimming, scuba diving or, more rarely, in other strenuous physical activity. In contrast, acute respiratory symptoms (ARS) have been reported in a considerable proportion (24.6%) of breath-hold divers (BHD).[1]
The aim of this study was to investigate possible inherent risk factors such as genetic predisposition, as already demonstrated in high-altitude pulmonary oedema; (HAPE).[2]

**Materials and Methods**: 64 male and female experienced healthy BHD were studied, 26 of them reported at least one episode of ARS while the remaining had no clinical history of ARS. We investigated the association of angiotensin-converting enzyme (ACE) gene insertion/deletion polymorphism with high-altitude pulmonary oedema. We also investigated two variants of the endothelial nitric oxide synthase gene (eNOS) the G894T polymorphism, implicated in vasodilatation regulation and regulating blood flow and pressure, and polymorphism T786c implicated in the pathogenesis of cardiovascular deseases.[3] Additionally we investigated: Interleukin-1 beta (IL1B rs16944), interleukin 1 receptor antagonist (IL1RN rs419598), glutathione S-transferase Mu 1 (GSTM1), glutathione S-transferase theta-1(GSTT1), superoxide dismutase 2 (SOD2 rs4880) implicated in inflammatory and the oxidative stess response respectively.

**Results:** We found interesting associations between ACE insertion/deletion polymorphism and ARS ($P$ = 0.006), as well as associations between polymorphism of endothelial nitric oxide synthase gene (eNOS) and ARS especially if different combinations of polimorphism are considered ($P$ = 0.0055). We did not find significant correlation with the other genes.

**Conclusions:** Our results show a possible genetic predisposition to pulmonary oedema in BHD in agreement with similar studies in HAPE. A simple genetic test could help to predict predisposed subjects and improve breath-hold diving safety through better individual awareness of possible risk factors.

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**Keywords:** Acute respiratory distress syndrome, breath-hold diving, diving, haemoptysis

## REUNION2013
## Scientific Posters on Diving and Hyperbaric Medicine

| **Hyperbaric Oxygen Therapy Posters** | | | |
|---|---|---|---|
| P-01 | X Chan | HYPERBARIC OXYGEN AND MYOCUTANEOUS FLAP REPAIR | |
| P-02 | D Linnarson | FORTY YEARS OF HYPERBARIC RESEARCH IN EUROPE: HIGHLIGHTS FROM THE FIRST EUROPEAN UNDERSEA BAROMEDICAL SOCIETY MEETING 1973. | |
| P-03 | P Brkic | HYPERBARIC OXYGENATION ATTENUATES NEUROINFLAMATION IN A RAT MODEL OF BRAIN INJURY | |
| P-04 | T Jovanovic | THE EFFECT OF HYPERBARIC OXYGENATION ON 7,12-DIMETHYLBENZ(a) ANTHRACENE-INDUCED (DMBA) CARCINOGENESIS IN THE WISTAR RAT | |
| P-05 | P Bothma | CEREBRAL GAS EMBOLISM: PARADOXICAL OR RETROGRADE ACCESS? | |
| P-06 | | *(withdrawn)* | |
| P-07 | H Renner | HOW TO SETUP A STUDENT TEACHING MODULE ON HBOT AND DIVING MEDICINE FOR THE MEDICAL STUDY PROGRAM – GOING THE LINE FROM DAILY ROUTINE TO SCIENCE | |
| P-08 | V Campanaro | THE EUROPEAN BAROMEDICAL ASSOCIATION FOR NURSES, OPERATORS AND TECHNICIANS (EBASS) CONSIDERATIONS ABOUT A HYPERBARIC OXYGEN FACILITY | |
| P-09 | V Campanaro | WHAT REPRESENTS EBASS (EUROPEAN BAROMEDICAL ASSOCIATION FOR NURSES, OPERATORS AND TECHNICIANS) FOR HYPERBARIC FACILITIES IN EUROPE | |
| P-10 | Y Tkachenko | MEASUREMENT OF ISOPROSTANES IN EXHALED BREATH CONDENSATE (EBC) AFTER HYPERBARIC HYPEROXIA | |
| P-11 | M Clamer | EFFECT OF OXYGEN AND PRESSURE ON DROSOPHILA MELANOGASTER (FRUIT FLY): OXIDATIVE STRESS, MITOCHONDRIAL ACTIVITY AND LIFE SPAN | |
| P-12 | P Vera Cruz | EFFECT OF HYPERBARIC OXYGEN THERAPY ON GLUCOSE HOMEOSTASIS IN TYPE 2 DIABETIC PATIENTS | |
| P-13 | F Guerreiro | EVALUATION OF CLINICAL AND FUNCTIONAL PULMONARY EFFECTS OF HYPERBARIC OXYGEN THERAPY | |
| P-14 | N Subbotina | TREATMENT OF ACUTE HEPATITIS B WITH HBO. A CASE STUDY | |

## P-01 HYPERBARIC OXYGEN AND MYOCUTANEOUS FLAP REPAIR FOR UNHEALED PERINEAL WOUNDS AFTER PROCTECTOMY FOR INFLAMMATORY BOWEL DISEASE

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**Objectives:** Persistent perineal sinus (PPS) following proctectomy for inflammatory bowel disease affects up to 50% of patients and in 10% is refractory to conventional drainage, nutritional support and time. Myocutaneous flap repair is then an option, but fails in up to 10%. Anecdotal reports of hyperbaric oxygen therapy (HBOT) for chronic wounds and Crohn's perianal disease led us to explore pre- and post-operative HBOT combined with myocutaneous flap repair in a highly selected group of patients with PPS who had failed all other interventions.

**Methods:** Patients with PPS for >2 years received pre-operative HBOT (up 90-minute sessions at 223-243 kPa, 5 times per week for 5-6 weeks), before abdominoperineal PPS excision and perineal reconstruction with vertical or transverse rectus abdominis myocutaneous (RAM) flap within 2-4 weeks of completing HBOT. Post-operative HBOT (ten further 90-minute sessions) was administered within 2 weeks where practicable.

**Results:** Between 2007 and 2011, four patients with PPS underwent adjunctive HBOT and myocutaneous flap repair. Median duration of PPS prior to HBOT was 88.5 months (23-156 months). All patients had previously failed multiple (5 to >35) surgical procedures. Complete healing occurred in all 4 patients at a median follow-up of 2.5 months (range 2-3 months). There were no further hospital admissions for PPS at a median follow-up of 35 months (8-64 months).

**Conclusions:** HBOT in conjunction with PPS excision and perineal reconstruction with a RAM flap led to complete perineal healing in four patients with PPS refractory to all other interventions and appears to be a viable therapeutic strategy.

**Keywords:** Inflammatory bowel disease, proctectomy, persistent perineal sinus, wound healing, hyperbaric oxygen

## P-02 FORTY YEARS OF HYPERBARIC RESEARCH IN EUROPE: HIGHLIGHTS FROM THE FIRST EUROPEAN UNDERSEA BIO-MEDICAL SOCIETY MEETING 1973.

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The first meeting of the European Undersea and Bio-medical Society in Stockholm 1973 had 164 participants from 16 European countries, US, and Australia. The seven separate sessions dealt with; Current research activities in the host country; oxygen, hydrogen and inert gases; ventilation, circulation and gas exchange; workshop on ethical considerations in hyperbaric research; workshop on vestibular disturbances in diving; performance and heat exchange and hydrostatic pressure, inert gas exchange and decompression.

The meeting and its 530 page proceedings provided a broad review of the state-of-the-art in hyperbaric physiology and medicine. From today's perspective, it is fair to state that much of the work presented 40 years ago formed the basis for further rapid developments in several fields, especially in the physiology of the exercising diver, use of hydrogen in deep diving, the mechanisms for hydrostatic pressure effects and inert gas narcosis. The workshop on ethical considerations was the first of its kind in the hyperbaric research community, and defined several issues that are still debated, such as how to ascertain sufficient expertise in review boards for research ethics. Likewise, the workshop on vestibular disturbances was the first international review in the field and presented several novel concepts, such as alternobaric vertigo which is not uncommon in recreational diving, that have expanded over the subsequent four decades. Over the same period, research on extremely deep diving and the use of exotic gases like hydrogen first expanded and later almost disappeared.

This first meeting formed the basis for a series of meetings that became the natural annual meeting point for scientists in the field of diving medicine on this side of the Atlantic. Later, HBO activities were included and in 1993 the name of the Society was changed to the European Underwater and Baromedical Society.

**Reference**

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## P-03 HYPERBARIC OXYGENATION ATTENUATES NEUROINFLAMATION IN A RAT MODEL OF BRAIN INJURY

P Brkic[2], I Lavrnja[3], A Parabucki[3], I Bjelobaba[3], D Stojkov[3], S Dacic[4], I Bozic[3], S Pekovic[3], M Stojiljkovic[3] and T Jovanovic[1]

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**Objective**: Finding effective therapies for traumatic brain injury (TBI) is the ultimate goal of experimental TBI research. Such therapies must be focused, in particular, on blocking the secondary inflammatory response or promoting regeneration and repair mechanisms. We aimed to evaluate the effects of hyperbaric oxygen therapy (HBOT) on inflammation and astroglial scarring in an animal model of TBI.

**Methods**: Rats were divided into five groups(n = 8 per group): Control animals (C), Control + HBO (CHBO), Sham controls (S), Sham control + HBO (SHBO), Lesion group (L), subjected to stab injury of the left sensorimotor cortex, Lesion + HBO (LHBO). HBOT protocol: pressure 253 kPa, for 60 minutes, daily for 10 consecutive days Animals were sacrificed two hours after the 10th HBO exposure. The posttraumatic processes were evaluated on the tissue preparations using an immunohistochemical method (antibodies: Glial fibrillary acidic protein (GFAP) and Vimentin), immunofluorescence (antibodies: CD40 and CD154), followed by Western blot and Real time (RT)-PCR analysis, for: GFAP, vimentin, CD40, CD154 and for Intercellular adhesion molecule 1(ICAM1).

**Results**: Reduced GFAP gene, protein and tissue expression occurred in the LHBO in comparison to the L group ($P < 0.005$). Reduced expression of vimentin gene was registered in LHBO in comparison to the L group ($P < 0.005$), but there were no differences in protein expression ($P > 0.005$). Weaker vimentin staining and formation of fibrous astrocytes in the LHBO group indicate a reduction of reactive astrogliosis and the prevention of glial scar formation. Inflammatory infiltration was seen around lesion in L and LHBO group.ICAM1 mRNA expression and CD154 protein expression were lower in LHBO in comparison to the L group ($P < 0.005$), indicating that HBOT is decreasing leucocytes adhesion. There were no differences in CD40 protein expression ($P > 0.005$).

**Conclusions**: These data suggest that HBOT attenuates the inflammatory response to TBI and creates a permissive environment for neuronal regeneration.

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**Keywords**: Brain injury, research, astrogliosis, immune response

## P-04 THE EFFECT OF HYPERBARIC OXYGENATION ON 7,12-DIMETHYLBENZ(a) ANTHRACENE-INDUCED (DMBA) CARCINOGENESIS IN THE WISTAR RATS

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**Objectives**: Breast cancer is the second most common type of cancer, and the fifth most frequent cause of death from cancer. The objective of this study was to examine the protective potential of molecular oxygen under hyperbaric conditions (HBO) in rats with DMBA-induced breast carcinogenesis.

**Methods**: HBO effects were evaluated by monitoring the tumour incidence and analysing biochemical and molecular markers during DMBA-induced breast carcinogenesis. The carcinogenesis was induced by a single intramammary injection of linseed oil and saline emulsion containing 25 mg of DMBA. Wistar rats were divided into four groups (n = 10 per group): G1 – DMBA+HBO three days after DMBA administration and daily for the following 4 weeks, treated with 253 kPa for 90 minutes; G2 – DMBA only; G3 – intramammary 1ml of saline and same HBO protocol and G4 – intramammary 1ml of saline.

**Results**: Ten weeks after the beginning of the experiment, HBO statistically significantly prevented (G1 vs. G2, $P < 0.05$) the formation of tumour in G1 group (80% of the rats had no malignant changes) in comparison to the G2 animals which, in which 80% of the rats had developed malignant breast disease with metastatic changes. Only one malignant change at the level of application without metastasis was recorded in one animal of the G3 and with less local tumorous change in one animal of the G4. HBO showed a significant protective effect from DMBA-induced carcinogenesis in rats, which is documented not only by the number of tumorous changes but also by significantly lesser deviation in biochemical and molecular abnormalities (G1 vs. G2, $p < 0.05$).

**Conclusions**: These findings confirm a possibility to consider molecular oxygen application under hyperbaric conditions in terms of its antigenotoxicity with antioxidative potential and modulating effects in some of cascade phases of detoxification and malignant alterations.

**References**

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**Keywords**: carcinogenesis, research, hyperbaric oxygen

## P-05 CEREBRAL GAS EMBOLISM: PARADOXICAL OR RETROGRADE ACCESS?

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**Introduction**: Iatrogenic cerebral arterial gas embolism (CAGE) is a potential devastating complication of many invasive medical procedures. The incidence in most health care systems is unknown. Recently, it was shown in Paris that 2.69 cases with CAGE per 100,000 hospital admissions were treated in the hyperbaric unit.[1]
Cerebral gas embolism can result from:

a) direct entry of gas into the arterial system;
b) the venous system paradoxically through an intracardiac right-to-left shunt. Cases where intracardiac shunt is excluded have been postulated to be due to AV malformations in the lungs or overwhelming of the pulmonary capillary filter mechanism.[2]
c) Another lesser known source is retrograde cerebral venous gas embolism (RCVGE).[3]

**Methods**: Finding cerebral venous air in the case reported[3] lead to the following:

1. A MEDLINE search was done to gain more information about cerebral venous air embolism as opposed to cerebral arterial air embolism. To get guidance as far as management strategies, expected outcome, as well as possible explanations for the gas entry in this case.[3]
2. Studying existing review articles on air embolism, as well as hyperbaric, critical care and emergency medicine textbooks for discussion of cerebral venous air embolism.

**Results**: The MEDLINE search found one case report in a critical care journal and did not find any references in hyperbaric and emergency medicine journals. A handful of case reports were found in forensic medicine[4,5], radiology and neurology journals. No useful information was available in the textbooks traditionally teaching emergency medicine and critical illness.

**Conclusion:** RCVGE is certainly not innocuous.[5] Flow dynamics allowing retrograde venous embolisation has been demonstrated experimentally.[4,5] A number of published reports of CAGE were most likely mistaken RCVGE (4). An International Registry is in progress. To participate, contact pabothma@gmail.com

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**Key words:** Gas embolism, iatrogenic, cerebral, retrograde

## P-07 HOW TO SETUP A STUDENT TEACHING MODULE ON HBOT AND DIVING MEDICINE FOR THE MEDICAL STUDY PROGRAM – GOING THE LINE FROM DAILY ROUTINE TO SCIENCE

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**Introduction**: We report on the design, introduction and daily life of students in the university setup of our hyperbaric chamber. This module is fully integrated into the medical study program with a final exam.

**Methods**: We evaluated the preliminary needs and limits of teaching students in HBOT and diving medicine in the only high-dependency hyperbaric chamber in Austria at university level. The daily routine with more then 1,600 compressions a year was reviewed with reference to the extra workload students might cause. Then, the organizational structures and workflow needed to be transparent. Getting all the peaces together involved various disciplines who were brought together for a brainstorming session; for instance, to identify topics like how to give students an insight into what is involved in treating critical ill intubated patients in a hyperbaric chamber. Finally, the scienific perspective should have enough space.

**Results**: The student module running during summer time is designed for a maximum of 18 students. As our chamber fits 10 outpatients or two intensive care patients at the same time, it was necessary to split the students into small groups for practical work. To foster practical aspects, the standard lectures were reduced to a minimum and replaced mainly by group work. Each student is responsible for “his/her” patient together with their medical lecturer. Lectures in the virtual medical campus completed the required basic knowledge. “Hip” aspects and an unusual final exam were integrated into the program to make this special module on “Hyperbaric and Diving Medicine” even more attractive.

**Conclusions**: Although the practical and technical requirements of a hyperbaric chamber makes it difficult for teaching it can be done successfully.

**Keywords:** Education, student work, university

## P-08 THE EUROPEAN BAROMEDICAL ASSOCIATION FOR NURSES, OPERATORS AND TECHNICIANS (EBASS) CONSIDERATIONS ABOUT A HYPERBARIC OXYGEN FACILITY

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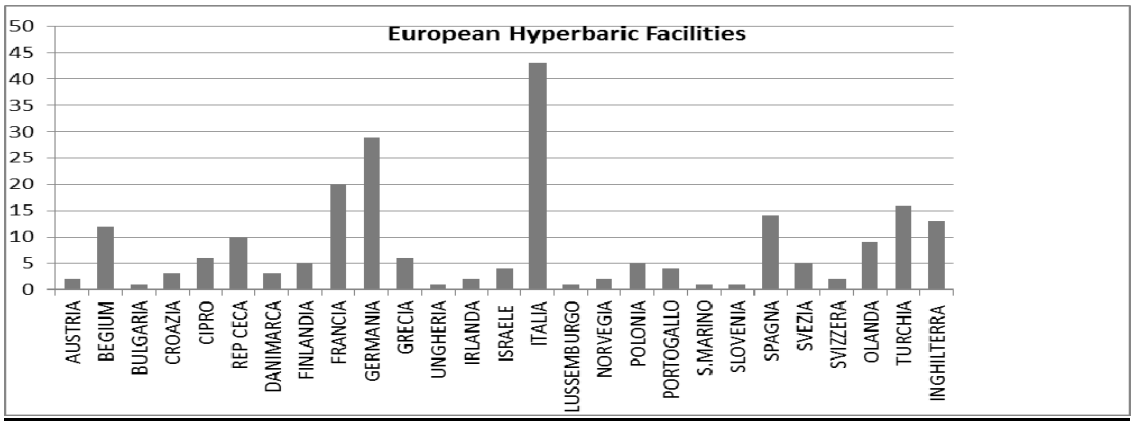
**Introduction:** At ECHM Consensus Conference in Belgrade 2012 discussion took place regarding the steps needed to establish a new hyperbaric baromedical facility. A checklist to be used when creating a new facility was developed. EBAss participated, bringing hands-on experience to this event from professionals working in hyperbaric facilities throughout Europe, most with many years of working to ensure quality and safety in their own centres. We thought it would be useful to present EBAss's views on this subject at this meeting. .

**Methods:** We gathered information from a selection of European hyperbaric facilities (in Belgium, Greece, Germany, Italy, Netherlands, Sweden, Switzerland and the United Kingdom), bearing in mind that it was a small sample compared to the approximately 220 centres in Europe. We then compared various parameters such as: environment of the facility (tertiary or other hospital, stand-alone, etc.); intensive care capability; ability to treat diving emergencies, etc. Any deficiencies: Ergonomics (environment); personnel (e.g., insufficient numbers); education (recognition by national norms), certification and accreditation of courses, etc., were documented.




**Results:** The facilities sampled are advanced as regards technological and safety aspects. Each facility uses detailed work procedures. The majority of hyperbaric centres provide care for both emergency and elective cases. There are variations in staffing levels which tend to be justified on the basis of the high costs to manage and in some cases because of the lack of national regulations. There is no European code to plan medical supervision, so each facility has developed their own protocols.

**Conclusion:** Accreditation of individual staff is now possible under the EBAss/ECB scheme (1) (2). Hyperbaric training centres can also now be accredited if they show their training is carried out to the agreed standards. This ensures that staff trained under this scheme are well prepared for the tasks undertaken at an HBO centre.

**References:**

1. A European Code of Good Practice for a HBO (by Working Group "Safety" of the Cost Action B14 "Hyperbaric Oxygen Therapy" May 2004)
2. Resources Manual of EBAss (Education of nurses, operators and technicians in hyperbaric facilities in Europe Resources manual: a document of EBAss/ECHM, Version 2008)

## P-09 WHAT REPRESENTS EBASS (EUROPEAN BAROMEDICAL ASSOCIATION FOR NURSES, OPERATORS AND TECHNICIANS) FOR HYPERBARIC FACILITIES IN EUROPE

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**Introduction:** EBAss was formed 10 years ago; aims were to clarify identities and roles of staff working in hyperbaric facilities across Europe. It was unthinkable that staff should work without definition of their roles. It is essential for all levels of our staff, to understand their roles, allowing them to work within specified parameters both technically and clinically.

**Methods:** All BD members together with Robert Houman, the first President of EBAss, have worked with ECHM/ECB to promulgate the first Pan-European training scheme for hyperbaric staff culminating in their accreditation (EBAss/ECB).

**Results:** We now have formulated very clear reference "Manual of Resources" that is recognized by ECHM and describes the individual modules of reference for nurses and chamber operators.[1] We are now working to establish the role of 'Safety Manager' and are conducting a review of the Manual, which will be completed during 2013. As part of this review there will be inclusion of modules for those who work with hyperbaric monoplace chambers where individual member states' national legislation for HBO allows their use.

**Conclusion:** In many countries there are no specific recognized specialties for HBO personnel; the EBAss certification demonstrates commitment and competence of hyperbaric personnel. EBAss/ECHM have become acknowledged European reference bodies that centres can use for reference. Accreditation of staff under the EBAss/ECB scheme is now possible as training schools can achieve accreditation showing their training satisfies the EBAss/ECB requirements, in accordance with agreed standards as defined in our RM[2] This ensures staff trained and accredited under this scheme will be thoroughly prepared for the tasks required. It would be useful to allow members of staff, operators and nurses, to attend at least one workshop or congress at national or European level per year.

**References:**

1. Resources Manual of EBAss (Education of nurses, operators and technicians in hyperbaric facilities in Europe Resources manual: a document of EBAss/ECHM Version 2008).
2. By News 2013 site www.ebass.org

## P-10 MEASUREMENT OF ISOPROSTANES IN EXHALED BREATH CONDENSATE (EBC) AFTER HYPERBARIC HYPEROXIA

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**Introduction:** Lipid peroxidation of arachidonic acid by free radicals creates isoprostanes. Metabolites of 8-iso-prostaglandin-F2alpha (8-iso-PGF2alpha) are reliable markers of oxidative stress when measured in plasma.[1] The aim of this pilot study was to investigate whether hyperbaric hyperoxia increases the levels of 8-iso-prostaglandin-F2alpha (8-iso-PGF2alpha) both in exhaled breath condensate (EBC) and in blood plasma of humans.

**Methods:** Eight young (age from 21 to 35 years), healthy soldiers were subjected to hyperbaric hyperoxia during the Oxygen Tolerance Tests (OTT) performed exclusively for military purposes. The research was approved by the local Ethics Committee. The EBC and blood samples were collected immediately before and after the exposure to 280 kPa oxygen for 30 minutes in a multiplace hyperbaric chamber. Breath condensate was collected over about 10 minutes breathing tidal volume by exhaling air through a tube system immersed in an ice bath (-10$^{O}$C). Then EBC and plasma samples were immediately frozen and stored at minus 70$^{O}$C until analysis.[2] High-performance liquid chromatography/mass spectrometry (HPLC-MS) was used for chemical analysis of 8-iso-PGF2alpha levels in both the EBC and blood plasma.

**Results:** The 8-iso-PGF2alpha level in the EBC increased significantly after oxygen exposure when compared to initial levels (median 5.25 [range 2,5-11,3] pg/ml vs median 2.22 [range 0-4,95] pg/ml; $P < 0.05$). At the same time there was no statistically significant change in blood plasma levels of 8-iso-PGF2alpha (median 41.75 [range 0-98] pg/ml vs median 31.3 [range 0-45,9] pg/ml; $P = 0.26$).

**Conclusions:** From this pilot study it seems that the measurement of exhaled isoprostanes provides a noninvasive method to quantify local (i.e. pulmonary) oxidant stress during hyperbaric hyperoxia. Moreover, these results provide further evidence that even short exposure to hyperbaric oxygen induces lipid peroxidation in lungs of humans.

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**Keywords:** Isoprostanes, exhaled breath condensate, hyperbaric hyperoxia

The paper was prepared with the support of the PHYPODE Marie Curie Initial Training Networks (FP7-PEOPLE-2010-ITN).

## P-11 EFFECT OF OXYGEN AND PRESSURE ON *DROSOPHILA MELANOGASTER* (FRUIT FLY): OXIDATIVE STRESS, MITOCHONDRIAL ACTIVITY AND LIFE SPAN.

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**Introduction: A**daptation to different atmospheric conditions is one of the major factors involved in the evolution of species; the relationship existing among oxidative stress, oxygen levels and pathological state of the individuals is complex. The aim of this study was to analyze the effects of oxygen and pressure in *Drosophila melanogaster*.

**Methods:** For this study we used two wild-type strains of *Drosophila melanogaster*: Canton-S and W1118. The flies were exposed to treatments different for duration (90 min and 3 hours), $O_2$ concentration (100% and 2%) and pressure (252, 151 and 121.5 kPa) in order to study the hypothesized different effects on life span of both adults and larvae and citrate synthase of adult male and female flies.

**Results:** The spectrophotometric mitochondrial analysis revealed specific, sharp alterations in citrate synthase activity: hyperoxia led to a significant increase of the enzymatic activity while, on the other hand, hypoxia and hyperbaric conditions caused the biggest reduction of the mitochondrial enzymatic activity. We observed a significant reduction of life span especially compromised after ten days from the exposure (and decreased from the usual 70-80 days to a 20-30 days survivorship after treatments). The most relevant alteration can be observed under hypoxic conditions with an even smaller effect in larvae when under concomitant hyperbaric conditions.

**Conclusion:** These results provide evidence that the alteration of environmental conditions can alter both mass and enzymatic activity of mitochondria. Moreover, there is an interesting relationship between mitochondrial alterations and lifespan, which needs further study.

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**Keywords:** Oxygen, pressure, *Drosophila melanogaster*

## P-12 EFFECT OF HYPERBARIC OXYGEN THERAPY ON GLUCOSE HOMEOSTASIS IN TYPE 2 DIABETIC PATIENTS

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**Introduction:** The carotid bodies (CB) are arterial chemoreceptors that sense changes in arterial blood $O_2$, $CO_2$ and pH levels. When activated, the CBs respond to induce a range of cardio-respiratory reflex responses, aimed to normalize the altered blood gases[1] and to regulate blood pressure and cardiac performance via sympathetic nervous system activation.[2] Recently we have demonstrated that the CB is involved in the development of insulin resistance and hypertension in overfed animals through sympathoadrenal over-activation.[3] Knowing that CB activity is abolished by hyperoxia (100% oxygen) we have tested the effects of hyperbaric oxygen therapy (HOT) on glucose homeostasis in type 2 diabetic (T2D) patients.

**Methods:** Volunteers with an indication for hyperbaric oxygen therapy (HBOT) were recruited at the Teaching and Research Unit of the Subaquatic and Hyperbaric Medical Centre of the Portuguese Navy. Written informed consent was obtained from all individuals. Volunteers were divided in two groups: T2D patients and controls. Inclusion criteria for the T2D are those defined by the American Diabetes Association in 2010. All groups were submitted to 20 sessions of hyperbaric oxygen therapy. Oral glucose tolerance tests were done before the 1$^{st}$ and in the last HBOT session.

**Results:** Six male T2D patients were included (Age 66 ± 3.89 years; BMI 25.54 ± 1.08 $kgm^{-2}$). Blood glucose levels before the 1st HBOT session were 134.67 ± 10.83 mg $dl^{-1}$ fasting and 290.50 ± 32.11 mg $dl^{-1}$ 2 hours post-oral glucose tolerance test (OGTT). After 20 HBOT sessions, glycemia values were 120.83 ± 7.70 mg $dl^{-1}$ fasting and 203.833 ± 30.72 mg $dl^{-1}$ 2 hours post-OGTT. In control group HBOT did not modify fasted glycemia but also decreased glycemia post-OGTT.

**Discussion:** Our preliminary results showed that HBOT improves glucose tolerance in type 2 diabetic patients and suggest that HBOT could be used as a therapeutic intervention for the treatment of T2D.

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**Keywords:** Glucose homeostasis, type 2 diabetes, hyperbaric oxygen therapy, carotid body

## P-13 EVALUATION OF CLINICAL AND FUNCTIONAL PULMONARY EFFECTS OF HYPERBARIC OXYGEN THERAPY

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**Introduction:** Previous studies reported changes in pulmonary function tests in patients submitted to hyperbaric oxygen therapy (HBOT). This study aimed to quantify both the short-term and cumulative effects of our standard HBOT protocol on pulmonary function, as assessed by serial standard pulmonary function testing.

**Methods:** A prospective, observational study included patients treated between December 2012 and April 2013. Patients with lung disease, clinical respiratory findings, abnormal chest X-rays, or previous irradiation of the head, neck or thorax, were excluded. Former were also exclusion criteria. Patients received daily HBOT, 5 days per week at 252 kPa for two 35-minute periods of oxygen breathing, separated by 5 minutes air breathing. Patients answered a symptoms questionnaire and performed serial lung function tests before and after the 1st, 10th and 20th sessions. We compared different phases with paired sample analysis.

**Results:** We evaluated 21 patients (9 females), aged 53±15.1 years. Two patients reported nonspecific chest complaints. Multiple respiratory functional parameters - forced vital capacity (FVC), forced expiratory volume in one second ($FEV_1$), peak-flow (PEF), residual volume (RV) and single-breath carbon-monoxide diffusing capacity ($DL_{CO}$) were assessed. Pre-treatment parameters were within normal reference values, as shown for FVC (102.93 ± 18.5%), $FEV_1$ (103.05 ± 18.20%) and $DL_{CO}$ (81.17 ± 15.86%). FVC, $FEV_1$, and PEF declined both in short-term evaluation (before and immediately after HBOT sessions) and before the 1st, 10th and 20th sessions), in some cases with significant differences. RV increased and $DL_{CO}$ declined across the treatment course, but this was not statistically significant.

**Discussion:** Intermittent exposure to HBOT seems to have short-term effects on pulmonary function. As patients were evaluated approximately 22 hours after the previous HBOT session we cannot be sure about single responsibility of cumulative effects. More studies are needed, namely recreating other HBOT protocols in order to assess this problem.

**Keywords:** Pulmonary function, hyperbaric oxygen therapy, oxygen toxicity, short-term oxygen exposure, cumulative oxygen exposure

## P-14 TREATMENT OF ACUTE HEPATITIS B WITH HBO. A CASE STUDY.

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**Introduction:** Hepatitis B (HB) continues to be a significant public health issue. An estimated 2 billion people have been infected with the hepatitis B virus worldwide, and some 300 million are chronically infected and become carriers of the virus.[1] Except for aggressive and/or chronic forms, treatment is limited to rest and diet. Typically, it takes several months to recover from symptoms, and some patients progress to chronic liver disease. Hyperbaric oxygen therapy (HBOT) has been reported to be of clinic benefit in liver disease.

**Materials and Methods:** A previously healthy 39-year-old, male patient presented with general malaise, anorexia, nausea, vomiting, jaundice and fever persisting for several weeks. He was positive for HBsAg, anti-HBc IgM, HBeAg, negative for anti-CMV Ig M, HIV1/HIV2. His hepatic enzymes were altered (SGOT > 1200 U/mL; GPT > 2800 U/mL, GGT > 220 U/mL), total bilirubin 13.9 mg/dL. Two weeks after diagnosis, HBOT was added to his standard HB treatment, and he received 20 HBO sessions at 223 kPa, 60 min of duration, over 60 days.

**Results:** Soon after starting HBOT, the patient experienced improvement, with normalization of clinical and laboratory parameters within less than 2 months after the initiation of HBOT. Seroconversion for HBeAg was observed (loss of HBeAg and appearance of anti-HBe).

**Conclusion:** In a case of acute HB HBOT was applied with a favorable and rapid improvement in the patient's condition, normalization of hepatic enzymes and seroconversion for HBeAg, which occurred 2 months from the start of HBOT. Generally this phenomenon occurs in 5-6 months and precedes seroconversion for HBsAg.

**Reference:**

1. http://health.usnews.com/health-conditions/infectious-diseases/hepatitis-b

**Keywords:** Hepatitis B, HBOT, seroconversion for HBeAg

| **Diving Medicine Posters** | | | |
|---|---|---|---|
| P-15 | J Jendle | NEED FOR INTERNATIONAL GUIDELINES ON DIABETES MELLITUS AND RECREATIONAL SCUBA DIVING | |
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## P-15 NEED FOR INTERNATIONAL GUIDELINES ON DIABETES MELLITUS AND RECREATIONAL SCUBA DIVING

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The ban for subjects with diabetes to dive is still under debate. The diving fatalities were evaluated using the DAN fatality database 1992-2004. Among a total of 975 fatalities, 37 divers were registered as having diabetes (3.8%). Eleven of these were on insulin treatment (1.1%) and 26 were on oral antidiabetic treatment (2.7%). To evaluate the risk of hypoglycaemia during diving and the possible link between fatalities and hypoglycaemia has been difficult. A problem about diving with diabetes is that this is accepted in some, but not all countries. The nationality of the diver is of less interest since recreational diving is performed worldwide. A person could be tempted to avoid mentioning his/her illness in order to be able to participate in a dive or dive training.

DAN and UHMS conducted a workshop in 2005: *Diabetes and recreational diving: guidelines for the future*.[1] Topics were selection and surveillance, scope of diving and glucose management on the day of diving. The old Swedish recommendations on diabetes and recreational diving have recently been updated and are now harmonized with the DAN recommendations.[2]

The number of divers is growing annually and, with the epidemic increase of diabetes, more divers will be prone to diabetes. What can be done to further improve safety for recreational divers with diabetes? New treatment options such as the use of new glucose-lowering drugs (DPP-4 inhibitors and GLP-1 analogues) and the new insulin formulations with a more physiological profile have been shown to reduce the risk of hypoglycaemia. The use of continuous glucose monitoring and self-monitoring blood glucose have been shown to identify individuals with unstable glucose control and elevated risks of hypoglycaemia.

**Conclusion:** There is a need for international guidelines in order to further improve the safety of subjects with diabetes during recreational scuba diving

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**Keywords:** Diabetes, hypoglycaemia, guidelines, fitness-to-dive

## P-16 IS THE TRANSITION FROM DAY TO NIGHT ACTIVITY A RISK FACTOR FOR THE DEVELOPMENT OF CNS OXYGEN TOXICITY DURING HYPERBARIC OXYGEN EXPOSURE?

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**Background:** The greatest risk involved in diving with closed-circuit oxygen apparatus is the development of CNS oxygen toxicity (CSN-OT). Several cases of CNS-OT have occurred recently, with one fatality. It seems that all of these incidents occurred at a time when the divers were active during the night.

**Objectives**: 1. To investigate whether a switch from day to night activity is a risk factor for the development of CNS-OT, by searching for a possible correlation between any reduction in latency to CNS-OT after the transition to night activity and a concomitant change in levels of melatonin. **Methods**: The study was conducted on the male fat sand rat (*Psammomys obesus*), which is a diurnal animal. A preliminary exposure to hyperbaric oxygen established the pressure that would enable us to compare latency between the experimental and control groups. The animals were kept in light for 12 hours in daytime and in darkness for 12 hours at night. Baseline measurements of latency to CNS-OT were carried out at 5 atmospheres absolute during the daytime hours, when melatonin levels were lowest according to measurements of urinary 6-sulphatoxymelatonin (6-SMT) excretion. During the experimental phase, the animals were kept in light for 12 hours in daytime and in an illuminated environment for most of the night hours. Melatonin levels were again determined by measuring 6-SMT in the urine.

**Results:** Latency to CNS-OT was significantly reduced after the transition from day to night activity (19.26 ± 4.00 and 8.53 ± 0.68 min, respectively). This was associated with alterations in the levels of melatonin.

**Conclusions:** Activity during the night probably resulted in changes of the melatonin level. This was associated with shortening of the latency to CNS-OT. A phase of nocturnal activity is probably an additional risk factor for the development of CNS-OT.

**Keywords:** CNS-oxygen toxicity, melatonin, sand fat rat

## P-17 CAN DEPRESSION OF CRITICAL FUSION FREQUENCY BE MODIFIED BY ACCELERATED DECOMPRESSION WITH EAN50 DURING EXTREME DEEP COLD WATER DECOMPRESSION DIVES? A PILOT STUDY

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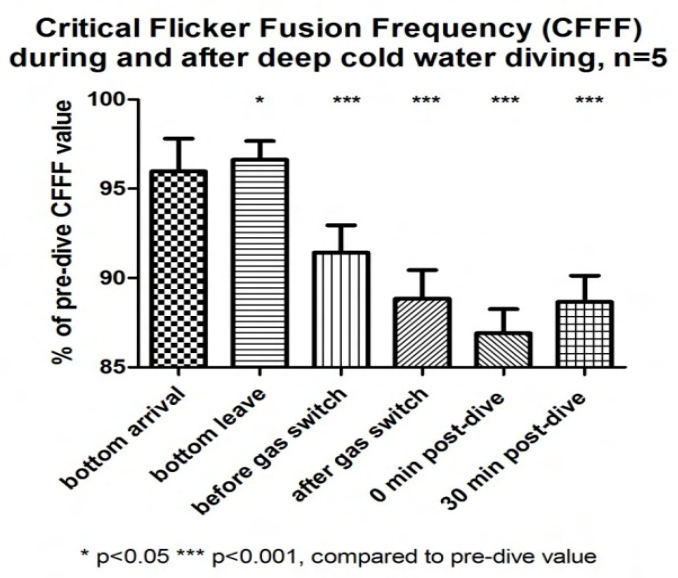
**Introduction:** Technical recreational diving is usually associated with dives outside the standard recreational range. This includes extreme cold exposure, depth, prolonged dive times and switch of breathing gas to accelerate decompression procedure. Understanding of brain function during these dives is limited. In the present study, the effect of cold, deep-water decompression dives on cognitive brain function was evaluated.

**Methods:** Five well-trained, adequately equipped divers participated in the study and performed an open water air dive to 37 msw with bottom time of 40 min and decompression with EAN50 from 21 msw to the surface. Water temperature during the dive was 2–3ºC. Cognitive brain function was tested with Critical Flicker Fusion Frequency (CFFF) and modified Psychology Experiment Building Language battery tests (PEBL): modified perceptual vigilance task, modified Timewall and math processing task. The PEBL tests were performed once 15-30 min before and 30 min after the dive. CFFF was measured 15-30 min before the dive, at arriving to bottom, 5 min before leaving bottom, before the gas switch at 21msw, 5 min after gas switch at 21 msw, immediately after surfacing and 30 min after surfacing.

**Results:** There were no significant differences in the modified Timewall or math processing tasks before and after the dive. Simple reaction time in the modified perceptual vigilance task was non-significantly prolonged after the dive compared to pre-dive (305.6 ± 35.26 ms vs. 323.8 ± 30.24 ms; P = 0.31) The CFFF decreased when divers arrived at depth and remained attenuated during the bottom phase. No recovery of the CFFF depression was observed during breathing EAN50. The CFFF remained significantly decreased after re-surfacing and 30 min post-dive compared to pre-dive values (see Fig.1).




**Conclusion:** Deep, cold-water decompression dives significantly decreased CFFF throughout and for some time after the dive, suggesting an impaired arousal state.

**Keywords:** CFFF, neuropsychometric, arousal, deep diving

This study is part of the PHYPODE project, financed by the European Union under a Marie Curie Initial Training Network Program.

## P-18 COMPARATIVE ANALYSIS OF EXPRESSION AND PLASTICITY OF THE DIVING BRADYCARDIA TRIGGERED BY REPETITIVE FACE IMMERSIONS IN INSPIRATION AND EXPIRATION

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This study was partially financed by Phypode project, a Marie Curie Initial Training Network Program.

**Introduction:** The diving response is characterized by apnea, diving bradycardia and peripheral vasoconstriction. These autonomic adaptations can decrease oxygen consumption and delay the rapid progression of asphyxia during breath-hold diving. Humans usually perform dives in inspiration, while many aquatic mammals dive in expiration. In animals, the expiratory position is essential to trigger the pronounced diving bradycardia that allows their outstanding dive performances [1]; however, data on the expression of the diving bradycardia in expiration in humans are sparse.

**Methods:** In the present study we performed a comparative analysis of variations in the expression and plasticity of the diving bradycardia triggered by face immersion (FI) in cold water whilst breath-holding (BH) in comfortable end-inspiration or expiration. The experiments were conducted in three groups with different BH diving experience: non-divers (control subjects, n = 15), underwater-rugby players (UW, n = 6) and free divers (n= 7).

**Results:** In freedivers, a single FI in comfortable expiration immediately triggered more pronounced diving bradycardia than FI in comfortable end-inspiration. No short-term training effects were observed in this group for repetitive FI in either the inspiratory or expiratory position. In the control group and UW-rugby players, breath-hold duration progressively increased during repetitive FI in both expiration and inspiration. Moreover, in both these groups a progressive increase in the strength of the diving bradycardia evoked by FI was observed, whereas repetitive FI in inspiration revealed a progressive attenuation in the strength of the diving bradycardia.

**Conclusion:** We showed that, in humans, FI in expiration was most favorable for full expression and training of the diving response.

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**Keywords:** Diving response, breath-hold, apnea, training, free-diving

## P-19 VIAGRA PRE-TREATMENT PROMOTES DECOMPRESSION SICKNESS IN RATS

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**Background:** Vascular bubble formation after decompression contributes to endothelial injuries which form the basis for the development of decompression sickness (DCS). Nitric oxide (NO) is a powerful vasodilator that contributes to vessel homeostasis. It has been shown that NO-releasing agent may reduce bubble formation and prevent serious DCS. The use of sildenafil (Viagra®), a well-known phosphodiesterase-5 blocker, which acts by potentiating vasodilation, has never been studied in DCS. The purpose of the present study was to evaluate the clinical effects of sildenafil pre-treatment on DCS in a rat model.

**Methods:** 67 rats were subjected to a simulated dive at 90 msw for 45 min before staged decompression. The experimental group received 10 mg/kg of sildenafil one hour before exposure (n = 35) while controls were not treated (n = 32). Clinical assessment took place over a period of 30 min after surfacing. At the end, blood samples were collected for blood cell counts and the level of circulating bubbles in the right-sided cavities was quantified.

**Results:** There were significantly more manifestations of DCS in the sildenafil group than in the controls (34.3% vs 6.25% respectively, $P = 0.012$). Platelet count was more reduced in treated rats than in controls (-21.7% vs -7%, respectively, $P = 0.029$), whereas bubble grades did not differ between the two groups.

**Conclusion:** Pre-treatment with sildenafil promotes the onset and severity of neurological DCS. This effect is likely due to the increase of cerebral blood flow with accumulation of inert gas responsible for bubble formation and DCS during decompression.

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**Keywords:** Diving, decompression sickness, bubble, nitric oxide, sildenafil, rat

## P-20 A DIVING AND MEDICAL PROFILE OF OCCUPATIONAL DIVERS IN AUSTRALIA

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**Introduction:** This presentation will profile occupational divers in Australia to ascertain the prevalence of pathological pulmonary changes in occupational divers. Divers may be at risk of developing lung disease.[1,2] Findings indicate that increased diving experience correlates with changes in pulmonary function, indicating pathological changes of potential clinical significance.[3] The results are more controversial for professional divers because of the different degrees of exposure and training. Although the prevalence of pulmonary oedema in divers is unknown, Slade et al [4] argue that over 1% of scuba divers have experienced it, though this is probably under-reported. Given that not all divers develop pulmonary oedema, even those who have all the potential risk factors, Snyder et al [5] suggest that genetic variation in the regulatory proteins important in lung fluid balance may influence susceptibility to pulmonary oedema.

**Methods:** Prospective and retrospective approaches gather data from occupational divers across Australia. As part of their annual occupational diving medical assessment, participants complete spirometry and a health questionnaire requesting information on allergies, smoking profile, respiratory illnesses, experience of pulmonary oedema, medication use, regular exercise undertaken, frequency of diving and diving experience. A venous blood sample is collected to obtain DNA.

**Results:** Data collection is due for completion mid-year. Preliminary findings from 240 participants show that 72.9% are male; mean age 36 years, BMI 26; 28.5% currently smoke, 93.6% drink alcohol. On average, participants have dived for over 15 years and completed up to 8,500 dives. Ear/sinus squeeze, decompression illness, headaches, extreme tiredness, haemoptysis, persistent cough, S.O.B., bronchitis/pneumonia and vomiting blood are among conditions experienced. Lung function measures indicate a wide distribution in $FEV_1$ [58.7-95.0; (16% <75)] and FVC (2.61-8.93)

**Conclusion:** This presentation will provide a detailed health and diving profile of occupational divers across Australia.

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**Keywords:** Diving medicine, occupational divers, pulmonary oedema

## P-21 IN VITRO AIR DIVING SIMULATION OF ENDOTHELIAL CELLS: MITOCHONDRIAL ACTIVITY, REACTIVE OXYGEN SPECIES AND APOPTOSIS.

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**Introduction:** Function of vascular endothelium during diving plays a key role in diving-related injuries, e.g. decompression sickness (DCS). In this study mitochondrial activity, cellular morphology and viability of arterial endothelial cell were examined in real time during simulated 45 min air dive to 810 kPa in a 1cc microchamber. [1]

**Methods:** Endothelial cells isolated from calf aorta were loaded into the *in vitro* system before diving simulation. Fluorescent dyes DASPMI, rhodamine 123, MitoSOX Red, propidium iodide (PI) and calcein-AM, were used to analyze mitochondrial activity and membrane potential (MMP), superoxide, apoptosis, cell death and viability of diving endothelial cells respectively. N-acetylcysteine (NAC), a reactive oxygen species (ROS) scavenger, was used examine the impact of ROS during dive. Results are compared with a non-diving control group.

**Results:** During the 7 min compression to 810 kPa, ROS and mitochondrial superoxide were increased; MMP decreased and cells lost their innate morphology becoming spherical. During the 45 min-air dive ROS content decreased and became undetectable after 45 min at 810 kPa, MMP became undetectable gradually and more cells became spherical. Cell death and apoptosis showed time-dependent increases during 45 min-diving simulation. NAC decreased cell death in diving endothelial cells.

**Conclusion:** During air compression and following diving superoxide, ROS species and MMP are abnormally modified suggesting that the mitochondrial activity of arterial endothelial cells may be affected. Cytoskeleton is irreversibly reorganized and viability of the endothelial cell is affected via apoptosis induction in which the injured mitochondria may be involved.

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**Keywords:** Diving, ROS, mitochondria, cytoskeleton, apoptosis

## P-22 PHYSIOLOGICAL EFFECTS OF LONG-DURATION DIVING (8-10H) IN PROFESSIONAL DIVERS

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**Introduction:** Physiological adaptations induced by long duration diving are still unclear[1,2].

**Objectives:** We assessed physiological variables during and after 8-10h immersion, reproducing the constraints actually incurred by professional divers, to assess whether this type of diving presented risks to the health of divers.

**Methods:** We studied a series of 12 experimental dives (duration 8-10h; water temperature 18°C; wearing 7.5 mm wet suit). Divers used rebreather apparatus, alternating dynamic (finning; 100% $O_2$/7msw) and rest periods (50% Nitrox/10-20msw). Oral hydration was given regularly (200 ml/h).To drink, divers had to stay "head-out of the water" for a few seconds. Urine volumes and blood samples were collected. Hemoglobin (Hb), hematocrit (Hct), plasma sodium [$Na^+$] and potassium [$K^+$] concentrations, core and mean skin temperature were measured. Ingested pills were used to assess core temperature. Echocardiography was performed before and immediately after each immersion.

**Results:** Core temperature was maintained during diving (36.1 ± 0.5°C) thanks to an increase in the intensity and duration of shivering during diving, and the finning periods. Despite this, two divers' core temperature dropped below 35.5 °C ('danger' threshold).
Although divers hydrated regularly, body mass fell (2.4 kg) with a 15% loss in plasma volume. Water intake limited the divers' water deficit, but did not improve their overall hydration status, due mainly to increased urination. Plasma [$Na^+$] remained stable throughout the duration of immersion, while [$K^+$] fell close to the lower limit of normal.
After the 8-10 h dives, left and right ventricular preloading and stroke volume fell, and these variables remained abnormally low 15h after the end of the immersion.
Respiratory function after the dives showed no anomalies, but an ophthalmological examination revealed a slight reduction in visual field.

**Conclusions:** Our results show large differences in adaptation between divers. Body temperature and hydro-electrolyte status should be closely monitored during extended dives.

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**Keywords**: Long term immersion, rebreather, thermo physiology, hydration status, oxygen toxicity.

## P-23 DEVELOPMENT OF A PHYSIOLOGICALLY BASED COMPUTATIONAL MODEL TO DESCRIBE THE KINETICS OF GAS TRANSFER AND BUBBLE FORMATION DURING DIVES.

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**Introduction:** A physiologically-based model was developed to analyze the kinetics of gas transfer and of gas bubble formation during the compression and decompression phases of scuba dives.

**Methods:** Saturation / desaturation kinetics during air dives were evaluated through the continuous measurement of oxygen saturation in a simplified physical model. A computational model of this simplified system considered diffusion and convection between compartments. Bubbles were included, a pre-existing population of stabilized micronuclei was defined and pressures within bubbles were calculated from ambient pressure and surface tension pressure. Gas transfer between bubbles during the liquid transport phases was calculated considering diffusion laws. The next step was to transfer this approach to a physiological model of mammal: the rat. A simple model of gas transfer taking into account the steps of gas exchange and metabolic oxygen consumption has been built. The model was established using bibliographic data on basal physiological state.

**Results:** The computational model of the simplified physical model allowed reproduction of the oxygen partial pressure kinetics during compression and decompression. It also allowed calculation of the total volume of bubbles formed during and after the decompression. The mammal model allowed basal oxygen partial pressures in various biological compartments to be reproduced.

**Conclusion:** The first validation of the model has been made measuring oxygen partial pressure in different tissues during normobaric hyperoxia. The results are under analysis. The next step will be to measure gas transfer kinetics in the rat during air dives. This will identify the parameters of bubbles in each compartment.

**Keywords:** Modeling, oxygen, gas kinetics, rat

## P-24 BLEEDING COMPLICATION RATES DURING SCUBA DIVING BY DIVERS USING ANTIPLATELET OR ANTICOAGULANT DRUGS: PRELIMINARY RESULTS FROM THE DIDIH (DIVING WITH DISORDERS IN HEMOSTASIS) STUDY AND CALL FOR COOPERATION

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**Background:** Recreational scuba diving by divers with comorbid disease and use of medication is common practice and includes divers using antiplatelet or anticoagulant drugs. Although some experimental data suggest a protective effect of these drugs on the sequelae of decompression sickness, their use might also exacerbate bleeding complications from diving. There is a paucity of safety data regarding diving with drugs that increase bleeding tendency. **Methods:** We initiated a web-based observational cohort study. Participants filled out an online questionnaire on www.divingresearch.org to retrospectively document personal medical details, diving experience and diving incidents with a particular focus on bleeding complications.

**Preliminary results:** Between February and April 2013, 580 divers completed the questionnaire. Thirty-five divers indicated the use of antiplatelet (n = 26; aspirin, dipyridamole and/or clopidogrel) or anticoagulant (n = 9; acenocoumarol or low-molecular-weight-heparin) medication during 24,729 dives. The incident rates during these dives were compared to those of the remaining 545 divers who reported on 299,917 dives. DCS rates were similar (0.81 vs. 0.73 / 10,000 dives), as was the incidence of ENT barotrauma (3.24 vs. 3.90 / 10,000 dives), although this more frequently involved nose bleeds in divers using antiplatelet drugs. One case of minor hemoptysis was reported by a diver taking aspirin. No other serious bleeding complications were reported.

**Discussion / conclusion:** Our preliminary results do not indicate a clear increase in diving-related (bleeding) complications in recreational divers using **antiplatelet or anticoagulant** drugs, besides a higher incidence of nose bleeds. This supports the notion that scuba diving can be safely performed by divers using these drugs. Inclusion using the Dutch-language questionnaire version is ongoing and will be followed-up by a prospective study. An English (and possibly French) variant of the questionnaire will become available online. To further facilitate international diver participation, the authors aspire to find collaborators at the Reunion 2013 meeting.

**Keywords:** Recreational scuba diving, diving safety, bleeding complications, antiplatelet drugs, anticoagulant drugs

## P-25 KINETICS OF CHANGES IN PLASMA CONCENTRATION OF CARDIAC NATRIURETIC PEPTIDES ACCORDING TO DIVE FEATURES

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**Introduction:** Stretch and distortion of atrial myocytes cause atrial natriuretic peptide (ANP) release. Contractility of ventricular myocytes triggers brain natriuretic peptide (BNP) synthesis. Diving increases cardiac preload and breathing work, whilst cold exposure increases cardiac afterload. High plasma levels of ANP and BNP are found after immersion in pulmonary oedema cases.[1,2]

**Methods:** Kinetics of plasma Nt-proANP, Nt-proBNP and GMPc were assessed during experimental and sea dives. Water temperatures ranged from cold (10°C) to thermoneutral (34°C).[3] In one study, active ANP and BNP were assessed together with stoichiometric propeptides. Some studies were at rest, and others had intermittent or sustained exercise.[4] Subjects were fit, trained military divers.

**Results:** Compared with pre-immersion values proANP increased 1.7 times during 1-3 h rest at 1m depth. Using a rebreathing device led to a slightly larger increase (x 1.9). Sustained fining (4 times larger $VO_2$ than rest) led to a 2.2 increase after 1 and 2 hours. Resting in cold water caused a 2.3 increase in plasma ANP. After 3 h at 10 m depth in rebreathing condition, plasma proANP reached 3.4 times pre-immersion values. Plasma proANP decreased after 1-3 h dive. BNP and proBNP changes were delayed beyond ANP. During quiet dives, plasma proBNP had doubled after 6 h in thermoneutral water, and was tripled after 3 h in cold water. ProBNP plasma levels remained high for more than 2 h after immersion. The largest ANP and BNP levels during the dives remained lower than averages in heart failure patients, and cases of diving pulmonary oedema. Plasma GMPc changes paralleled ANP kinetics.

**Conclusions:** In fit young adults, diving breathing work drives graded ANP release, beyond the effect of immersion-enlarged cardiac preload. Cold-increased cardiac afterload forcefully bolsters BNP release. Due to their longer half-lives, plasma levels of cardiac propeptides better reflect the overall cardiac release than values for biologically active peptides.

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**Keywords**: Diving immersion, atrial natriuretic peptide, brain natriuretic peptide, cardiac preload, cardiac afterload, breathing work, cold

## P-26 THE EFFECT OF COLD FRESH WATER ON CORE TEMPERATURE IN DIVERS AFTER SPINAL CORD INJURY

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**Objectives:** Thermoregulatory responses to cold in divers after spinal cord injury (SCI) are altered. This study evaluated the decrease in core temperature in divers after SCI and in able-bodied divers.

**Methods:** Ten scuba divers, five able-bodied and five with SCI (thoracic level), performed a shallow dive (maximum depth 5.3m, duration 30–36min) in cold water (temperature range 6–9°C) in a wet suit (two-piece 5mm, Kanoko® superstrech/ Pile® thermal inside, zipper to face). Core temperature was measured with an ingestible radio-pill (CoreTemp® Temperature Sensor 262K15VSOHCO38075) and detected by an external receiver/recorder (CoreTemp® Data Recorder 262K w/HR HT 130042, HQI Calibration) prior to, directly after and 5, 10, 15, 30 and 120 min subsequent to the dive.

**Results:** The core temperature in divers after SCI decreased more than in the control group, but the difference was not statistically significant directly after and 5, 10, 15 min post-dive. There was a significant difference between the groups 30 min (SCI mean 36.03°C, SD 0.904; able-bodied mean 37.76°C, SD 0.493; $P = 0.037$) and 120min (SCI mean 36.62 °C, SD 0.135; able-bodied mean 37.44°C, SD 0.569; $P = 0.050$) after the exposure. Time for core temperature to recover post-dive was longer in the SCI group than the able-bodied group.

**Conclusion:** Divers after SCI are capable of diving in cold water but should have a shorter exposure time than able-bodied divers. Because of the slower recovery of core temperature post-dive, active rewarming must be available for SCI divers.

**Keywords:** Cold water, spinal cord injury, SCI, core temperature, diving

## P-27 DECOMPRESSION INDUCED BUBBLE GROWTH ON TISSUE SURFACES FROM GAS SATURATED SOLUTIONS

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**Introduction:** The study of bubble formation and growth during hyperbaric decompression can improve decompression models used in SCUBA diving. In particular, we wish to develop an experimental set-up for studying how bubbles induced through degassing of gas saturated solutions depends on the liquid properties and composition, decompression profile and nucleation site (tissue).

**Method:** A new experimental set-up that allows the accurate and sensitive optical recording of bubble growth on tissues *during* hyperbaric decompression was developed. Fresh fat and muscle tissues were obtained from rabbits. Each tissue was then put into a specially designed cell in a small decompression chamber and covered by nitrogen saturated liquid, then decompressed. Two decompression profiles were tested, 3 bar gauge pressure to 0 bar gauge, with and without a 15 min decompression stop at 1bar gauge. Bubble growth rates and densities over surface area were measured, recording the radii of bubbles every 5 seconds from the beginning of the decompression.

**Results:** The set-up was shown to allow the observation of the growth rate of selected bubble, but also bubble density per unit surface area, comparing how these vary for different tissue surfaces, decompression profiles and gas saturated liquid composition. The bubble growth optical set-up provided a field of view of 6 x 4mm$^2$, with a resolution of 1.75μm.

**Keywords:** Degassing, hydrophobicity

## P-28 EVALUATING COUNTING OF VENOUS GAS EMBOLI ON POST-SCUBA DIVE ECHOCARDIOGRAPHS

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**Introduction:** An automated counting of the venous gas emboli (VGE) observed on post-dive B-mode ultrasound can permit a more sensitive evaluation of dive-induced stress. This information could then be used to develop improved decompression algorithms which are currently evaluated on the severity grade attributed by trained experts on such recordings.

**Method:** An automated bubble recognition algorithm was developed in MatLab™. Firstly the right and left heart cavities are segmented using intensity thresholds, top-hat filtering and morphological openings. Secondly the cardiac cycle information is used to extract the frames where the valves are fully open. Finally bubbles are labelled and counted after an ultrasound speckle removal procedure. The automated method was initially evaluated in vitro with known concentrations of microbubbles in a laboratory setup. It was then evaluated in vivo compared to manual assessment from ultrasound B-mode sequences acquired post dive both pre- and post- knee flexion from 10 volunteers.

**Results:** The developed automated method is able to segment the B-mode data of the heart into two regions of interest consisting of the right and left chambers. The in vitro validation shows that the automated counting is able to successfully reflect a doubling in microbubble concentration. Initial results from the in vivo evaluation show good agreement and suggest this method to be a powerful tool for validating decompression algorithms quantitatively.

**Keywords: B**ubble, imaging

## P-30 FLYING AFTER DIVING: IN-FLIGHT ECHOCARDIOGRAPHIC STUDY AFTER A WEEK OF INTENSIVE RECREATIONAL DIVING

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**Introduction:** Flying after diving may increase the risk of decompression sickness (DCS), but strong evidence indicating minimum pre-flight surface intervals (PFSI) is missing. We performed Echocardiography on divers flying back from a seven-day diving holiday, with a 24 hour PFSI.

**Methods:** 22 healthy subjects were studied by in-flight Doppler echocardiography during the return flights after two different diving weeks in the Maldives. Doppler echocardiography was done in four different conditions: 1: outgoing flight, no previous dive; 2: during the diving week; 3: before the return flight, after a 24 hour PFSI; 4: during the return flight.

**Results:** We did not find any bubble in the right heart in any of the subjects tested during the outgoing flight. Echocardiograms during the diving week showed that three subjects consistently showed bubbles after every dive. No bubbles were observed prior to the return flight (pre-flight control with previous diving). During the return, flight bubbles were only observed in the same three subjects who demonstrated bubbling during the diving week. Thirty minutes after reaching cruising altitude (condition 4), these subjects showed bubbles scores of 3, 1 and 2 respectively, according to the Eftedal and Brubak scoring system ($P = 0.0237$).

**Conclusions:** If a 24-hours pre-flight interval is respected, the majority of subjects did not develop bubbles during altitude exposure. However, it is intriguing to note that only the same subjects who developed significant amounts of bubbles after every dive, showed equally significant bubble grades during in-flight echocardiography notwithstanding the 24-h PFSI.

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**Keywords: P**re-flight interval, echocardiography, bubbles

## P-31 FLYING AFTER DIVING: TECHNICAL HURDLES AND PROCEDURES FOR IN-FLIGHT ECHOCARDIOGRAPHIC MONITORING OF CIRCULATING GAS EMBOLI IN DIVERS.

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**Introduction:** Investigation of decompression-related problems occurring when flying after recreational diving requires not only simulated altitude studies, but real-time clinical investigation, ideally by in-flight Doppler echocardiography. However, the safety requirements for the use of electronic devices on board commercial aircraft are very stringent and so far forbid this investigation.

Scope of this work was to implement an airline-approved protocol to use such devices during commercial flights.

**Methods:** Specific tests were agreed with the Airline (NEOS) to assure that in-flight use of a Doppler-echocardiograph (MyLab 5, Esaote SPA, Florence, Italy) does not generate any interference with the aircraft instrumentation. As per request by the Airline, in-flight avionics conditions and configurations of the aircraft were replicated, during repeated tests on board, to rule out any possible interference. The Echocardiograph was then classified according to avionics safety procedures as not detrimental to native aircraft instrumentation.

To complete the study we also developed an original instrument to constantly monitor the aircraft's cabin pressure (adapted dive computer I Dive Pro, Dive System, Valpiana) thus allowing for independent measuring and comparison with the aircraft's native altimeter data.

**Results:** No electromagnetic interference with the aircraft's native instruments could be identified and our Device was classified as a Group 1 Portable Electronic Device (PED) (non-intentional Transmitting - Leaflet 29, JAA guide - EASA). All following tests were successful and clearance for in-flight use of the echocardiograph was granted (see EASA OPS 1). Our original cabin pressure monitor also proved effective and showed remarkably consistent values with the aircraft's native altimeter ($P = 0.9874$).

**Conclusion:** A Doppler-echocardiograph can be used in-flight. This innovation offers the opportunity to better investigate the safety of altitude exposure after diving.

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**Keywords:** Cabin pressure, measurements, electromagnetic interference, echocardiograph

## P-32 DECOMPRESSION ALGORITHM CONSIDERATIONS FOR THE BIONIC DIVER

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**Introduction:** The implementation of a physiological parameter-based algorithm adaptation is presented which interprets measurements of physiological parameters pre-, during and post-dive in order to provide adaptation of the decompression schedule which is not only tailored to the physiology of the diver but more specifically to the physiology of the diver in real time, thus accounting for hydration, fatigue and a variety of other conditions which affect how the human body responds to a dive stress.

**Methods:** The base algorithm is a Bühlmann-based 10 tissue model, with half times ranging between 2.5 and 240 minutes. Adaptation is achieved via on-the-fly modification of tissue half times, thereby artificially increasing or reducing ongassing and/or offgassing. Modifications are based on analysis and interpretation of measurements on physiological parameters such as heart rate, blood pressure, skin temperature, oxygen saturation, coupled with activity measurements such as strain gauges in the fins. Data collection comprises pre dive and post dive measurements such as blood chemistry and VGE. Data interpretation is performed in a modified Mares Icon dive computer, which has a powerful enough processor to handle the volume of calculations required, in addition to a wireless bidirectional communication which allows data collection without impeding diver mobility with wires.

**Conclusion:** Analysis of data and consequent adaptation of ascent profiles will lead to a better understanding of influential factors in human physiology and eventually to a more comprehensive decompression algorithm.

**Keywords:** Bionic diver, decompression algorithm

## P-33 ISOLATED GIANT BULLAE – NON-SURGICAL PROCEDURE TO GET THESE RELATIVELY YOUNG PATIENTS FIT TO DIVE

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**Introduction**: A bulla is defined as an air space in the lung measuring more than one cm in diameter in the distended state; the term giant bulla is used for bullae that occupy at least 30% of a hemithorax. A single giant bulla may be surrounded by normal lung tissue but is an absolute contraindication for diving due to the possibility of air trapping. Patients are diagnosed either occasionally at young age or come up symptomatically at the age around 50.

**Methods**: Because of a wish to go scuba diving by otherwise well, relatively young patients having a single giant bulla we were forced to rethink the options. New advances in interventional bronchoscopy provide the possibility of completely collapse a bulla with an endobronchial valve. This valve lets air and mucus out but no air in. It can be placed at segmental level. After examination and measurement, the involved segment is identified and confirmation of an isolated bulla with no other air inlet is gained. The valve is then placed endoscopically as part of the same procedure.

**Results**: Bullectomy involves the surgical removal of one or more giant bullae to improve symptoms and respiratory function in patients with bullous emphysema, which leads to a prohibition of diving for a minimum of a further three years. Body plethysmography showed that the patient recovers to levels conformable with scuba diving. After endoscopic endobronchial valve insertion, young patients can develop complete collapse of the bulla in the absence of pleural effusion due to the high expandability and elasticity of the healthy part of the lungs. Due to local adhesions, the bulla is securely sealed after 6 months.

**Conclusions**: The placement of an endobronchial valve seems to be effective to allow these patients to dive six month after the procedure.

**Keywords:** Endobronchial valve, lung giant bulla

## P-34 MEDICAL ASSISTANCE ON DIVING WORKSITE: THE "DIVINGDOC" EXPERIENCE

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Clients are progressively lowering the risk threshold, in the Hazard Identification (HAZID), for which Medical assistance on diving worksites is required. In PubMed no publication can be found about this topic.[1,2] "Divingdoc" Service was born in August 2009 to provide this service to eight Diving Contractors which employ 189 commercial divers full time and 476 freelance divers. 47 doctors and nurses cooperate in this team. Between November 2009 and April 2013 the team has covered 999 days (23.976 hours) of medical assistance, on nine worksites around the world: seven by saturation, two by Surface Supplied diving (*Table 1*). One is the "Costa Concordia wreck removal Project" (http://www.theparbucklingproject.com/) in the waters off the Giglio island (I) where, at the date of April 28th 2013, 7255 dives have been accomplished using three different decompression procedures: USN Rev 5 in water decompression using Oxygen Enriched Air; USN Rev. 6 with Surface Decompression and French tables "Travaux en Mileu Hyperbare" (June 1992). 430 personnel units are involved (180 divers) and 11 hyperbaric chambers.

The medical team includes a diving physician (3 - Level II D ECHM/EDTC med) supported by a nurse and/or Paramedic all trained for emergency management (especially for traumatic injuries and medical causes such as Anaphylaxis, AMI, etc.) as well as primary health care (colds, flus, stitches, wounds etc.) which is 90% of the medical team's activity. With regards to the diving medicine (the other 10% of activities), medical staff gives advice to the diving Supervisor and his Diving Medical Technicians (DMT's). Should a diver get seriously injured, medical personnel is responsible for his stabilization prior to his entrance into the hyperbaric chamber. The Authors analyzed the cases of health interventions (on average 130 per month for each site).

The appropriate training of health personnel in service aboard on diving worksites is recommended.

Table 1: Medical assistance on diving worksites (number of days)

| **Date** | **Company** | **Location** | **days of work (medical assistance on board)** |
|---|---|---|---|
| November 2009 | Micoperi | Croatia | 60 |
| March 2010 | Micoperi | Egypt | 43 |
| July 2010 | Micoperi | Italy | 126 |
| August 2010 | Micoperi | Israel | 226 |
| August 2012 up to now | Titan – Micoperi (Project "Costa Concordia) | Italy | 270 |
| November 2012 | Micoperi | Ghana | 93 |
| November 2012 | RANA | Congo | 20 |
| February 2013 | CNS | Angola | 45 |
| February 2013 | RANA | Lybia | 35 |
| March 2013 | CNS | Italy | 26 |
| March 2013 | Micoperi / CNS | Spain | 30 |
| April 2013 | RANA | Italy | 25 |

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**Keywords:** Diving medicine, medical assistance on diving worksite, working diving

## P-35 COMPARISON BETWEEN DIFFERENT DECOMPRESSION PROFILES OF TEK DIVES: A RESEARCH MODEL FOR ASSESSING INFLAMMATORY CHANGES AFTER DIVING.

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**Aim**: To share with the experts gathered at the Tricontinental Scientific Meeting (Reunion2013) the protocol of our study on the relationship between inflammation and diving profiles.

**Objectives of the study**: To evaluate circulating cytokines and chemokines following different kinds of technical (tech) and recreational diving profiles and to compare different tech decompression models and to correlate inflammation with Doppler bubble grading.

**Methods**: Three groups of 20 tech divers made a similar dive (50 metres, 25 min bottom time) using trimix 18/45/37; OEA 50% and oxygen 100%, but different decompression models and profiles: the compartimental model (Z-Plan), the Ratio Deco model and the Mnemonic Deco System UTRtek model. In addition, two further groups of 20 tech divers each, breathing air, were studied: one group swam on the surface for 30 min, the other made a recreational dive with minimum decompression (30 metres, 25 min bottom time; 3 minutes at 3 meters safety stop).Nurses were responsible for blood sampling at the diving site, and were also prepared to manage DCI (as actually occurred).

**Pre-post inflammatory profile**: 60 min before diving and 90 min after surfacing, venous blood (5 ml) was sampled from each diver. Cytokines and chemokines plasma levels were determined by using the multiplex Luminex© technology. Plasma determinations were made in triplicate, by using a 27 plex panel of analytes.

**Echocardiogram**: was recorded in each diver 60 min before and 90 min after diving, by a commercially available instrument (MyLab 30, ESAOTE-Italy). Bubble grading was determined off-line, on 16" loop (according to previously validated score) and compared with the inflammatory profile in each diver.

**Preliminary findings**: the following cytokines and chemokines changed after diving: IL-1b, IL-6, IL-8, MIP-1b, Rantes, IP-10 and MCP-1.

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**Keywords:** Ddiving medicine, decompression profiles, technical diving, stress response to diving, inflammation and diving, bubble grading

## P-36 EXCEPTIONAL DIVING EXPOSURE WITH DEEP STOPS AND DCS (CASE STUDY)

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**Background:** A healthy, 33- year-old male with no previous DCS dived to 110 m for seven minutes in 4°C water, followed by 147 minutes of decompression, including 40 minutes in a dry habitat at 4m. The water, reached via 406 stairs, was in a former mine at an altitude of 195 m above sea level. While exiting, two hours after the 170 minutes dive, the diver collapsed on the stairs, was lifted to the surface and evacuated on oxygen to the nearest recompression facility. A diagnosis of neurological DCS was made and treatment commenced 6-7 hours after surfacing. The diver received three recompressions, made a full recovery and returned to diving.

**Methods:** Using the R package SCUBA, stepwise tissue pressures in 17 Buhlmann compartments (ZH-L16A)[1] were estimated from the profile recorded by the dive computer. The RGBM dive plan generated by his software was similarly interrogated, as was a third profile without deep stops generated for comparison using the VPM-B/E model.

**Results:** Tissue pressure estimates are presented in Table 1.

Table 1: Estimated compartment pressure differences between dissolved inert gas and ambient (bar), with respective half-times for $N_2$ and **He**. [a]Estimated with R package "SCUBA", [b]Planned with Suunto Dive Manager 4, [c]Planned with V-planner VPM-B/E

| Compartment number | | 1 | 1a | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Half-time $N_2$ (mins)<br>**He** | | 4.0<br>**1.5** | 5.0<br>**1.9** | 8.0<br>**3.0** | 12.5<br>**4.7** | 18.5<br>**7.0** | 27.0<br>**10.2** | 38.3<br>**14.5** | 54.3<br>**20.5** | 77.0<br>**29.1** | 109<br>**41.1** | 146<br>**55.1** | 187<br>**70.6** | 239<br>**90.2** | 305<br>**115.1** | 390<br>**147.2** | 498<br>**187.9** | 635<br>**239.6** |
| **Max Delta P[a]** | Actual dive | -0.06 | -0.05 | 0.15 | 0.43 | 0.55 | 0.64 | 0.69 | 0.63 | 0.52 | 0.57 | 0.59 | 0.57 | 0.52 | 0.46 | 0.38 | 0.30 | 0.22 |
| | Dive plan[b] | -0.11 | -0.03 | 0.30 | 0.55 | 0.63 | 0.65 | 0.70 | 0.62 | 0.61 | 0.68 | 0.68 | 0.64 | 0.57 | 0.49 | 0.40 | 0.31 | 0.23 |
| | Without deep stops[c] | -0.09 | -0.09 | 0.01 | 0.29 | 0.49 | 0.61 | 0.68 | 0.67 | 0.71 | 0.71 | 0.70 | 0.65 | 0.58 | 0.49 | 0.40 | 0.31 | 0.21 |

**Discussion:** During the dive, compartments 5-7 appeared to experience the highest level of supersaturation above ambient pressure, compared with 6-10 if the plan had been followed, or VPM-B/E without deep stops. Compartments 1-5 had lower total inert gas supersaturation than was predicted by the RGBM but were higher than predicted by the VPM-B/E. In this dive the combination of gas switches (n = 5) and deep stops from 76 m depth appear to move decompression stress forward to faster compartments than a VPM-B/E plan with no deep stops, but not as far forward as the RGBM plan would have allowed. The effect of deep stops during exceptional exposure diving remains undetermined.

**Reference**

1. Powell, Mark (2008). Deco for Divers. Southend-on-Sea: Aquapress. ISBN 1-905492-07-3.

**Keywords:** Decompression, deep stops, dive profiles, recompression.

# P-37 EXERCISE INTENSITY INFERRED FROM AIR CONSUMPTION DURING RECREATIONAL SCUBA DIVING

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**Introduction:** Episodic exercise is a risk factor for acute cardiac events and cardiac complications are increasingly recognized as common in fatalities during recreational scuba diving.[1] What is not known is the exercise intensity involved in a typical range of recreational diving.

**Methods:** This study used the pre- to post-dive gas cylinder pressure drop to estimate air consumption and, from that, exercise intensity during recreational dives. Dive profiles were captured electronically and divers self-reported cylinder pressure changes, perceived workload, thermal status and problems during the dives. The dive profile was used to determine mean depth, which was then used to derive mean surface air consumption (SAC) rate. Mean exercise intensity, reported in metabolic equivalents (MET, multiples of assumed resting metabolic rate [3.5 mL $kg^{-1}$ $min^{-1}$]) was then estimated in accordance with a method employed with divers in controlled open-water trials.[2] Data are reported as mean±standard deviation.

**Results:** A total of 959 recreational air dives (20 ± 9 msw maximum depth; 50 ± 12 min underwater time) by 139 divers (age 42 ± 10 y; 73% male; 11±10 y of diving; 12% smokers) were monitored. Problems were reported with 129 of the 959 dives (13.5%): buoyancy (45%), equalization (38%) and rapid ascent (10%) being the commonest. Assuming a conservative 10% overestimate due to cylinder cooling and uncontrolled gas loss, the estimated exercise intensity associated with monitored dives was 5 ± 1 MET (Table 1). Mean±2SD, 7 MET, captures the effort associated with the vast majority of dives monitored. This value is similar to recent expert opinion and consensus statements recommending a minimum 6-7 MET aerobic capacity for recreational diving.

Table 1: Gas consumption and inferred exercise intensity by perceived workload (mean [SD])

| | 'Resting/Light' (n=683) | 'Moderate' (n=247) | 'Severe/Exhausting' (n=9) | Pooled (n=959) |
|---|---|---|---|---|
| Weight (kg) | 84.4 (16.2) | 78.4 (16.8) | 78.0 (15.6) | 82.2 (16.9) |
| Mean depth (msw) | 10.6 (4.3) | 11.0 (4.4) | 10.4 (3.4) | 10.8 (4. 3) |
| SAC ($V_E$) ($L.min^{-1}$)[a] | 17.4 (5.4) | 17 .9 (5.5) | 22.3 (6.2) | 17.7 (5.4) |
| $VO_2$ ($L.min^{-1}$)[a] | 1.52 (0.14) | 1.53 (0.14) | 1.64 (0.16) | 1.52 (0.14) |
| SAC/kg ($L.kg^{-1}.min^{-1}$) | 0.21 (0.07) | 0.23 (0.07) | 0.29 (0.06) | 0.22 (0.07) |
| $VO_2$/kg ($mL.kg^{-1}.min^{-1}$)[a] | 0.019 (0.004) | 0.020 (0.004) | 0.022 (0.003) | 0.019 (0.004) |
| Exercise intensity (MET)[a] | 5.3 (1.0) | 5.8 (1.3) | 6.2 (0.9) | 5.4 (1.1) |

[a] Estimated

**Conclusion:** Our estimates suggest that a 7 MET aerobic capacity may generally be adequate to complete uncomplicated recreational dives. Higher levels of aerobic fitness are still strongly recommended to ensure ample reserves. Further research is needed to quantify energetic demands of recreational diving during both typical and emergent events.

**References**

1. Denoble PJ, Caruso JL, Dear GL, Pieper CF, Vann RD. Common causes of open-circuit recreational diving fatalities. *Undersea Hyperb Med* 2008; 35(6):393-406.
2. Dwyer J. Estimation of oxygen uptake from heart rate response to undersea work. *Undersea Biomed Res* 1983; 10(2):77-87.

**Keywords**: MET, recreational divers, workload estimate

## P-39 CARDIOVASCULAR RISK FACTOR PREVALENCE AMONG AUSTRALIAN RECREATIONAL DIVERS

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**Introduction:** At the turn of this century hypertension and coronary artery disease were reported by 10% of Australian recreational divers.[1] Cardiovascular risk factors are increasingly noted among diving fatalities.[2] Consensus at the DAN Diving Fatality workshop included that cardiovascular health is a priority for recreational divers.[3] More recently cardiovascular risk factors have been found in a majority of professional divers.[4]

**Methods:** Data from Australian recreational divers were extracted from the DAN Project Dive Exploration (PDE) database. Cardiovascular risk factors were identified.

**Results:** Anthropometric and dive experience data are presented as mean and standard deviation in Table 1. Among males (n = 161, 73%), median age 40 years (range 25-65), previous cardiovascular problems were reported by 12 (nine hypertension and three hyperlipidemia, 8%), smoking by 14 (10%), coronary-related medication by nine (six hypertension and three hyperlipidemia, 6%) and BMI > 30 in 34 (21%). Among females (n = 59, 27%), median age 36 years (range 22-58), a cardiovascular problem was reported by just one diver (high blood pressure, 2%), smoking by 8 (15%), coronary-related medication by the same diver and BMI > 30 in 5 (8%).

**Table 1.** Australian recreational divers (n=220) anthropometry (mean±SD)

| | Males (n= 161, 73%) | Females (n= 59, 27%) | Overall (n=220) |
|---|---|---|---|
| **Demography** (mean, SD) | | | |
| Age | 42 (9) | 39 (9) | 41 (9) |
| Height | 179 (7) | 167 (9) | 176 (9) |
| Weight | 87 (13) | 67 (11) | 82 (16) |
| BMI | 27 (4) | 24 (4) | 26 (4) |
| **Dive experience** (median, range) | | | |
| Years of diving | 8 (0-36) | 10 (0-39) | 10 (0-39) |
| Dives prev. year | 28 (0-200) | 28 (0-400) | 28 (0-400) |
| Dives 5 years | 135 (0-1500) | 101 (0-2000) | 120 (0-2000) |

**Discussion:** At least one cardiovascular risk factors was found in 62/220 (28%) of this sample of Australian recreational divers. This is lower than found among professional divers although this study relied upon self-reporting.[4] The prevalence of smoking approximated that found in Australian males (10% vs. 16%) and females (15% vs. 14%). BMI > 30 was less common among this sample than among Australian adult males generally, (21% vs. 30%, respectively).[5] We postulate that divers with more severe cardiovascular risk factors may be more likely to retire from diving and, therefore, the prevalence of risk factors among divers may be lower than in the general population. However, this is speculative and cardiovascular risk factors remain a concern for diving safety.

**References**

1. Taylor DM, O'Toole KS, Ryan CM. Experienced, recreational scuba divers in Australia continue to dive despite medical contraindications. *Wilderness & Environmental Medicine* 2002; 13(3):187-93.
2. Denoble PJ, Caruso JL, Dear GL, Pieper CF, Vann RD. Common causes of open-circuit recreational diving fatalities. *Undersea Hyperb Med* 2008; 35(6):393-406.
3. Mitchell S, Bove AA. Medical screening of recreational divers for cardiovascular disease: Concensus discussion at the Divers Alert Network Fatality Workshop. *Undersea Hyperb Med* 2011; 38(4):289-96.
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5. Stewart S, Tikellis G, Crrington C, Walker K, O'Dea K. A report on the long-term consequences of Australia's expanding waistline on cardiovascular disease. Melbourne, Australia: Baker Heart Research Institute.2008.

**Keywords**: Cardiovascular, survey, body mass index, Project Dive Exploration

## Sunday, September 29th, 2013

**International DAN Divers Day**
Risk assessment and mitigation in recreational diving – Principles and Tools
*Location: "Le Voilier" room, Tamarun*

| | | |
|---|---|---|
| 09:00 | **Registration** | |
| 09:30 | **Welcome and introduction (video presentation)** | |
| 10:00 | **The concept of duty of care: technical, medical and legal implications. Lessons from real cases –** *Alessandro Marroni* | |
| 10:30 | **Risk assessment and mitigation principles & tools** – *François Burman* | |
| 11:00 | **DAN risk assessment programs** – *Guy Thomas* | |
| 11:30 | **Break** | |
| 12:30 | **Applied ergonomy** – *Introduced by Jack Meintjes* | |
| 12:45 | Discussion | |
| 13:00 | **Divers' behaviour & risk** – *Introduced by Costantino Balestra* | |
| 13:15 | Discussion | |
| 13:30 | **Diving equipment & risk** – *Introduced by Michael Lang* | |
| 13:45 | Discussion | |
| 14:00 | **"Leave them alone!" Preventing marine life injuries** – *Introduced by John Lippmann* | |
| 14:15 | Discussion | |
| 14:30 | **Panel discussion and conclusive remarks** – *Chair: Ramiro Cali Corleo*<br>*Discussants: Costantino Balestra, François Burman, Jacek Kot, Michael Lang, John Lippmann, Alessandro Marroni, Jack Meintjes, Guy Thomas.* | |
| 15.00 | **Adjourn** | |

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# Diving and Hyperbaric Medicine

*The Journal of the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society*

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*Diving and Hyperbaric Medicine* (DHM) has been the joint journal of SPUMS and EUBS since 2008. Indexed on **MedLine** since the March 2011 issue, article abstracts are also accessible on PubMed back to March 2008. DHM publishes original articles, reviews, technical reports, short communications, case reports, work in progress, 'world as it is' and 'opinion' articles and 'letters to the Editor'.

Since 2008, DHM has also been indexed in the Thomson Reuters database **Sci Search**® – The Web of Science, and has an **Impact Factor** for 2012 of 0.668 (comparable to *Undersea and Hyperbaric Medicine*), and our first five-year IF is 0.541.

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