

# Diving and Hyperbaric Medicine

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

**SPUMS**

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**EUBS**



## Inner ear barotrauma

Controversies in hyperbaric medicine – Réunion2013

Submarine escape – the CO<sub>2</sub>-off effect and Valsalvas

Variations in no-stop times among dive computers

Holter 12-lead ECG monitoring of scuba divers

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To promote and facilitate the study of all aspects of underwater and hyperbaric medicine  
To provide information on underwater and hyperbaric medicine  
To publish a journal and to convene members of each Society annually at a scientific conference

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For further information on SPUMS and to complete a membership application, go to the Society's **website: <www.spums.org.au>**

The official address for SPUMS is:

c/o Australian and New Zealand College of Anaesthetists,  
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SPUMS is incorporated in Victoria A0020660B

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<b>Phone &amp; Fax:</b>	+44-(0)20-85001778

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## DIVING and HYPERBARIC MEDICINE

<www.dhmjournal.com>

#### Editor:

Michael Davis <editor@dhmjournal.com>  
c/- Hyperbaric Medicine Unit  
Christchurch Hospital, Private Bag 4710  
Christchurch, New Zealand  
**Phone:** +64-(0)3-364-0045 or (0)3-329-6857  
**Fax:** +64-(0)3-364-0817 or (0)3-329-6810

#### European (Deputy) Editor:

Lesley Blogg <lesley.blogg@eubs.org>

#### Editorial Assistant:

Nicky McNeish <editorialassist@dhmjournal.com>

#### Journal distribution:

Steve Goble <admin@spums.org.au>

#### Journal submissions:

Submissions should be sent to <submissions@dhmjournal.com>

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# Editorials

## Hydrophobicity: the link between bubbles, bubblebers and autoimmunity?

Costantino Balestra, President EUBS

Some data show that divers can be divided into two groups: ‘bubblebers’ and ‘non-bubblebers’; no hypothesis has proposed a generally accepted explanation for such a phenomenon.

Hills demonstrated on electron microscopy an oligolamellar lipid lining on the luminal aspect of ovine blood vessels.<sup>1</sup> He also provided evidence of hydrophobicity, using the measured angle to a small (5µl) drop of water. The hydrophobicity was reduced by rinsing these vessels with chloroform, which led to this lining being identified as phospholipids. Hills suggested that the deposition of lung surfactants created this hydrophobic lining. Arieli and Marmur demonstrated clearly defined areas on the surface of blood vessels that fit the suggestion of hydrophobic spots at which bubbles nucleate and grow after decompression from higher pressure.<sup>2</sup> Tiny, flat gas nanobubbles measuring 5–100 nm form spontaneously when a smooth hydrophobic surface is submerged in water containing dissolved gas.<sup>3</sup> One might suggest, therefore, that a permanent layer of nanobubbles covers these hydrophobic intra-vascular spots.

### Protein interactions

The chain of amino acids in a protein may include hydrophobic acids, and the  $\alpha$ -helices are also the most common structural elements of the protein to cross biological membranes. Because hydrophobicity is high for a gaseous phase, the hydrophobic regions in proteins will react with the gaseous phase. In contact with a gas phase, the configuration of a protein will be altered and the denatured protein will change its immunochemical properties. This process occurs with bubbles in the blood.<sup>4</sup>

### Surfactants act against proteins and cause autoimmune diseases

Large protein molecules are probably carried in different quantities and with different timing in the blood. When a large molecule containing a hydrophobic domain encounters the strong hydrophobicity of the nanobubble layer at a surfactant spot, it will adhere to the spot and its altered configuration will be recognized as a foreign molecule, setting in motion an autoimmune response. Autoimmunity increases with age,<sup>5</sup> which is itself considered a risk factor for decompression sickness (DCS) in human divers. If the hydrophobically active spots increase in area and number with age, when added surfactants are deposited, this may explain the concomitant increase in the risk of DCS and in autoimmune diseases.

A large variability in the prevalence of hydrophobic spots in humans may explain differences in sensitivity to

autoimmune diseases and to decompression stress (bubblebers vs. non-bubblebers). The presence of hydrophobic spots and the availability of certain proteins may determine the timing of onset of the autoimmune disease. This might explain the early appearance of Type I diabetes and the later onset of other diseases.

This explanation of the bubblebers and non-bubblebers division is consistent with the ‘de-nucleation’ processes as applied to humans before diving and explains why mechanical energy such as vibration, oxygen breathing (at a time interval before diving that is incompatible with desaturation), thermal ‘energy’ and possibly exercise before diving all appear to be protective for bubble production.<sup>6–9</sup> Once again, science and research in diving show increasingly wider applications and connections with other physiological and pathological processes.

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**E-mail:** <costantino.balestra@eubs.org>

### Key words

Bubbles, decompression sickness, endothelium, surfactant, editorial

## David Smart, President SPUMS

Since my last report, the SPUMS Executive has completed a rewrite of the Purposes and Rules to comply with the new Consumer Affairs Victoria Model Rules Structure. At a Special General Meeting on 01 November in Sydney, these were adopted unanimously. The completion of this tedious task has multiple flow-on effects. It allows the Journal to now officially be published jointly with EUBS. It also allows for more flexibility in the dates of our ASM (which hosts the SPUMS Annual General Meeting), in keeping with our strategic relationship with EUBS.

Planning is well under way for the 2015 ASM to be held in May in Palau, convened by Dr Cathy Meehan. Cathy is a stalwart of SPUMS, having convened multiple ASMs, and contributed as an executive member over many years. In keeping with our recent ASMs, the 2015 meeting promises to be of a high quality. The main theme is diabetes and diving. Dr Neal Pollock is confirmed as our Guest Speaker. I encourage as many members as possible to attend. Registration is now open via a link on our website.

An urgent matter for resolution is establishment of a separate DHM Journal governance committee with representatives of both SPUMS and EUBS. This will permit greater support for the Editor in relation to financial governance and strategic direction. The committee would not cut across the academic independence of the Editorial Board, but would be responsible for the operational aspects of the Journal. It would also enable clearer lines of responsibility and reporting, as well as joint ownership (EUBS and SPUMS)

of journal processes so that communication is enhanced.

At the time of writing there was still some uncertainty regarding the ANZCA Certificate in Diving and Hyperbaric Medicine. Commenced over a decade ago, this qualification is regarded as the highest level achievable in the field of diving and hyperbaric medicine in Australia and New Zealand. Because of multiple factors and small numbers in the programme, the ANZCA has questioned the viability of the certificate. A number of SPUMS members including the Past President are working with ANZCA in an effort to maintain the certificate programme. The SPUMS Diploma continues to be a recognised qualification in diving and hyperbaric medicine, and is complementary to the ANZCA Certificate.

A major focus over the coming months will be a revision of the SPUMS website. We plan to modernise the structure and its functionality so that it meets our future needs. This process is being led by webmaster Joel Hissink with the help of Nicky Leen-McNeish who provides web support to both SPUMS and DHM. Very soon, we will be appointing professional web designers to facilitate this task. While there may be some future interruptions to the website, it will be for a good cause and the end product should be a major improvement, with significantly improved functionality.

I wish all our membership and EUBS colleagues a happy Christmas and a safe and productive New Year.

### Key words

Medical society, general interest

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## The Editor's offering

This issue is bigger than usual, which is a healthy sign for any editor, but does mean that some things have had to be squeezed in a bit, such as this offering. Once again, authors have provided a rich variety of research and information that is sure to provide something of interest for every reader, no matter their medical and/or research background.

The 'controversies in hyperbaric medicine' session at the joint meeting, Réunion2013, was well received at the time, and three of the presenters have provided articles. Two of these deal with the provocative topics of unestablished indications for hyperbaric oxygen treatment (HBOT), sham treatments and the interpretation of results, with particular reference to the current 'HBOT in chronic brain injury debate'. Their carefully argued opinions are sure to generate further debate on these critical issues. The article by Elliott and Smart on inner ear barotrauma impressed its two reviewers, both very experienced in the clinical management of this injury, as a useful, comprehensive review of the topic. If you thought dive computers were much the same, then the data in the article by Sayer et al will give pause for thought.

Clearly, the choice of computer to use in an occupational diving setting needs careful consideration.

The most important academic news for the journal is that Lesley Blogg, PhD, has been appointed to the Editorial Board as European (Deputy) Editor as Peter Müller's successor. We welcome her warmly and I look forward to our working together. Peter, whose contribution has been very important, will continue to be involved as a member of the forthcoming Journal Governance Group. An early task will be the adoption of new software to manage the submissions and peer review processes electronically, i.e., to move into the 21st century! More on both matters in the March 2015 issue. A majority of the EB had a productive face-to-face meeting during the EUBS ASM in Wiesbaden; only the second time this has been possible since SPUMS and EUBS joined forces.

*Michael Davis*

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**Front-page photo: Closed-circuit rebreather divers checking their computers prior to a mixed-gas deep dive.**

# Original articles

## Effects of Valsalva manoeuvres and the 'CO<sub>2</sub>-off' effect on cerebral blood flow

Fiona Seddon, Julian Thacker, Karen Jurd and Geoffrey Loveman

### Abstract

(Seddon F, Thacker J, Jurd K, Loveman G. Effects of Valsalva manoeuvres and the 'CO<sub>2</sub>-off' effect on cerebral blood flow. *Diving and Hyperbaric Medicine*. 2014 December;44(4):187-192.)

**Introduction:** Previous research has shown that a rapid drop in inhaled carbon dioxide (CO<sub>2</sub>) partial pressure reduces cerebral blood flow and may induce faintness – the 'CO<sub>2</sub>-off' effect. The aims of this study were to investigate the effects of performing Valsalva manoeuvres while experiencing the 'CO<sub>2</sub>-off' effect and whether symptoms occur that are sufficient to jeopardise submarine tower escape.

**Methods:** Twenty male volunteers, mean (SD) age 34.7 (8.5) years each completed three tests. The first test was to perform Valsalva manoeuvres breathing air. The second and third tests involved breathing a high CO<sub>2</sub> mix (5% CO<sub>2</sub>/16% O<sub>2</sub>/79% N<sub>2</sub>) for 1 h prior to switching to breathe O<sub>2</sub> and performing Valsalva manoeuvres, or switching to breathe air for 1 min then O<sub>2</sub> and performing Valsalva manoeuvres. Blood pressure, cerebral blood flow velocity, electrocardiogram, and respiration were monitored throughout. A subjective questionnaire was administered at intervals to monitor symptom type and severity.

**Results:** Valsalva manoeuvres breathing air resulted in a 31% reduction in cerebral blood flow. Breathing high CO<sub>2</sub> caused a sustained increase in cerebral blood flow and symptoms of breathlessness and headache. Following the gas switch from high CO<sub>2</sub>, some subjects reported faintness, headache and nausea. Cerebral blood flow dropped by 34% when switching from breathing high CO<sub>2</sub> to O<sub>2</sub>, by 35% when switching to air then by a further 3% when switching from air to O<sub>2</sub>. In both circumstances there was a further drop of 14% after performing the Valsalva manoeuvres. The drop in cerebral blood flow in subjects that reported faintness was greater than that in the subjects who did not, but this difference was not significant.

**Conclusion:** Transient faintness or headache may occur in the escape tower during pressurisation, but this should be short-lived and not incapacitating.

### Key words

Hypercapnia, Valsalva, cerebral blood flow, Doppler, physiology, submarine

### Introduction

Royal Navy submarines are fitted with tower escape systems allowing survivors to escape from a distressed submarine (DISSUB). There may be a long wait in the submarine prior to starting tower escape during which the partial pressure of carbon dioxide (PCO<sub>2</sub>) may rise despite use of a CO<sub>2</sub> absorbent. Submariners may switch from breathing a hypercapnic and hypoxic atmosphere in the DISSUB to a normocapnic and normoxic atmosphere in the escape tower. The submariner is subject to rapid pressurisation in the escape tower to equalise with the surrounding sea pressure, and then decompression as he ascends to the surface. During the pressurisation the escaper will also be exposed to a hyperoxic atmosphere, with the inspired partial pressure of oxygen (P<sub>i</sub>O<sub>2</sub>) reaching as high as 398 kPa at the maximum permitted escape depth (180 m).

Fainting usually occurs when a person is in the upright position and can be provoked by anything that reduces cerebral perfusion.<sup>1</sup> CO<sub>2</sub> is a cerebral vasodilator whilst O<sub>2</sub> is a cerebral vasoconstrictor. Thus the switch from breathing a hypercapnic gas in the DISSUB to a hyperoxic gas whilst stood in the escape tower may lead to transient cerebral vasoconstriction resulting in cerebral hypoperfusion, which

could in turn result in fainting. Fainting in the escape tower could endanger the escaper and hinder escape for the rest of the crew by blocking the tower with the escaper's body.

A previous study examined the physiological effects of the rapid replacement of a hypercapnic breathing gas with 100% O<sub>2</sub> – the 'CO<sub>2</sub>-off' effect.<sup>2</sup> Subjects breathed a mixture of 5% CO<sub>2</sub>/16% O<sub>2</sub>/79% N<sub>2</sub> (high CO<sub>2</sub>) for one hour and then switched to breathing O<sub>2</sub> for 15 min. Mild or moderate faintness was the most frequently reported symptom following the gas switch. Transcranial Doppler (TCD) was used to measure middle cerebral artery blood flow velocity (MCAv). There was a significantly greater percentage drop in mean MCAv in subjects who had symptoms of faintness that developed after the switch to O<sub>2</sub> when compared with those who did not.

Submariners are trained to minimise ear discomfort by equalising pressure across the tympanic membrane using Valsalva manoeuvres (Valsalvas). Valsalvas are known to cause a drop in MCAv when in the upright position.<sup>3</sup> This is a mechanical effect of the raised intra-thoracic and intra-abdominal pressure causing reduced venous return and cardiac output. Therefore, we hypothesized that performing Valsalvas following a switch from breathing high CO<sub>2</sub>

might exacerbate the drop in MCAv caused by the switch in breathing gas previously observed and possibly worsen any symptoms such as faintness or nausea.

## Methods

The study was approved by the QinetiQ Ethics Committee (ethical protocol SP792 v 2.0), and carried out at the QinetiQ Hyperbaric Medical Unit, St. Richard's Hospital, Chichester, UK. Volunteers gave their written informed consent and the study was conducted in accordance with the principles of the Declaration of Helsinki (revised 2008).

## STUDY DESIGN

It was hypothesised that Valsalvas would further increase the observed drop in mean MCAv caused by a gas switch from high CO<sub>2</sub>. A power test (power = 0.8 and alpha = 0.05) using R statistical software (version 2.10.1) determined that 16 subjects would be required to detect a significant increase in the mean percentage drop in mean MCAv of a further 10% over that caused by the switch to 100% O<sub>2</sub> alone. To allow for possible subject withdrawal, or increase in the observed standard deviation in mean MCAv, 20 subjects were recruited.

## SUBJECTS

Twenty male volunteers participated in the study, with mean (SD) age of 34.7 (8.5) years; height 179.8 (4.9) cm; body mass 84.4 (14.5) kg. Subjects were requested to refrain from alcohol the day before each test. They were requested to have a light breakfast and their normal caffeinated drink on the morning of each test. The subjects performed each of three test conditions on separate visits with a period of at least 24 hours between each.

## PROCEDURES

All tests were carried out at normobaric ambient pressure. British Oxygen Company supplied cylinders of medical quality 5% CO<sub>2</sub>/16% O<sub>2</sub> balance N<sub>2</sub>, hereafter termed 'high CO<sub>2</sub>' (note that in the previous study this was termed 5CO<sub>2</sub>/16O<sub>2</sub><sup>2</sup>). Medical O<sub>2</sub> and air were obtained from the hospital supply. Breathing gases were contained in Douglas bags and breathed via plastic tubing and a silicon mouthpiece. A four-way gas switching block (Hans Rudolph Inc.) was used to control the gas delivered.

The three tests were conducted as shown in Table 1. Test 1 was conducted first for all subjects, allowing familiarisation with equipment and procedures. The order of Tests 2 and 3 was randomised.

## VALSALVA MANOEUVRES

A calibrated pressure transducer (General Electric, Druck, 800–1200 mbar range) was connected to the mouthpiece

**Table 1**

Procedures for Tests 1, 2 and 3 (see text for more details)

Time (min)	Test 1	Test 2	Test 3
0	Start air (standing)	Start high CO <sub>2</sub> (seated)	Start high CO <sub>2</sub> (seated)
5	6 x Valsalva		
10	End		
50		Stand up	Stand up
60		Switch to O <sub>2</sub>	Switch to air
61		6 Valsalva	Switch to O <sub>2</sub>
62		Switch to air	6 Valsalva
63		Sit	Switch to air and sit
68		End	End

assembly to ensure Valsalvas were performed consistently. Subjects wore a nose-clip throughout. Valsalvas were performed by the subject occluding the mouthpiece exhale valve with the right hand while attempting to breathe out to achieve a mouthpiece pressure of 40 mmHg (5.3 kPa) above ambient for 2 s. A traffic light system displayed when sufficient effort had been achieved. Six Valsalvas were performed in 30 s by each subject.

## INSTRUMENTATION

A flow meter (KL Engineering Spirometric module S430A) placed in the inhale tubing allowed measurement of respiratory rate and minute volume. Subjects were instrumented for the duration of the test allowing measurement of:

- brachial blood pressure (BP mmHg) (General Electric, DINAMAP® Pro 1000) from the right arm;
- O<sub>2</sub> saturation (General Electric, DINAMAP® Pro 1000) from a finger on the left hand;
- blood velocity in the middle cerebral artery (measured continuously) using Transcranial Doppler transducer (Comtec TCD II) held in position at either left or right temporal region with a Rimed probe holder LMY2;
- electrocardiogram (ECG) using two independent ECG monitors (LifePulse10 HME Ltd and General Electric DINAMAP® Pro 1000) showing leads I and II;
- inspired and expired O<sub>2</sub> and CO<sub>2</sub> concentrations via a capillary tube from the centre of the mouthpiece to a Servomex 1440 fast-response gas analyser.

## DATA RECORDING

Heart rate, BP, respiration rate, respiratory minute volume, and mean MCAv were recorded each minute for 5 min then every 5 min until 60 min then at 1 or 2 min intervals to the end.

A subjective symptoms questionnaire was administered each minute for the first 5 min of high-CO<sub>2</sub> breathing, then after a further 5 min and then at 10 min intervals until the switch, when it was administered more frequently. The subject

**Table 2**  
Mean  $\pm$  95% CI absolute and % change in physiological parameters during Test 2

Test 2	Air baseline	1 min CO <sub>2</sub>	5 min CO <sub>2</sub>	30 min CO <sub>2</sub>	Final min CO <sub>2</sub>	1 min O <sub>2</sub>	Valsalva	1 min air	Final min air
Resp rate (breath·min <sup>-1</sup> )	11 $\pm$ 1.8	12 $\pm$ 1.8	13 $\pm$ 2.2	16 $\pm$ 1.9	17 $\pm$ 1.8	15 $\pm$ 1.8	10 $\pm$ 1.9	14 $\pm$ 2.2	13 $\pm$ 1.9
% change		9	18	45	55	36	-9	27	18
Resp minute vol (L·min <sup>-1</sup> )	6 $\pm$ 1.0	11 $\pm$ 1.9	17 $\pm$ 2.5	25 $\pm$ 2.4	28 $\pm$ 3.3	20 $\pm$ 2.9	NA	10 $\pm$ 1.9	6 $\pm$ 1.0
% change		83	183	317	367	233	NA	67	0
Mean BP (mmHg)	91 $\pm$ 1.9	96 $\pm$ 3.1	95 $\pm$ 3.1	96 $\pm$ 3.3	105 $\pm$ 3.7	103 $\pm$ 3.3	NA	101 $\pm$ 3.7	96 $\pm$ 3.3
% change		5	4	5	15	13	NA	11	5
Heart rate (beat·min <sup>-1</sup> )	61 $\pm$ 3.7	64 $\pm$ 3.1	62 $\pm$ 3.5	63 $\pm$ 3.5	73 $\pm$ 4.3	72 $\pm$ 4.9	79 $\pm$ 6.5	74 $\pm$ 5.3	57 $\pm$ 4.7
% change		5	2	3	20	18	30	21	-7
MCAv (cm·s <sup>-1</sup> )	65 $\pm$ 4.9	75 $\pm$ 6.9	79 $\pm$ 7.6	75 $\pm$ 7.3	76 $\pm$ 8.4	50 $\pm$ 6.5	40 $\pm$ 4.7	46 $\pm$ 5.9	63 $\pm$ 6.1
% change		15	22	16	17	-23	-39	-29	-3
ETCO <sub>2</sub> (kPa)	5.3 $\pm$ 0.2	6.0 $\pm$ 0.2	6.4 $\pm$ 0.2	6.3 $\pm$ 0.2	6.3 $\pm$ 0.2	4.3 $\pm$ 0.8	NA	4.4 $\pm$ 0.4	5.0 $\pm$ 0.2
% change		13	21	19	19	-18	NA	-17	-6

**Table 3**  
Mean  $\pm$  95% CI absolute and % change in physiological parameters during Test 3

Test 3	Air baseline	1 min CO <sub>2</sub>	5 min CO <sub>2</sub>	30 min CO <sub>2</sub>	Final min CO <sub>2</sub>	1 min air	1 min O <sub>2</sub>	Valsalva	Final min air
Resp rate (breath·min <sup>-1</sup> )	11 $\pm$ 1.4	12 $\pm$ 1.4	13 $\pm$ 1.6	16 $\pm$ 1.4	16 $\pm$ 1.4	16 $\pm$ 1.4	15 $\pm$ 1.4	12 $\pm$ 2.4	13 $\pm$ 1.8
% change		9	18	45	45	45	36	9	18
Resp minute vol (L·min <sup>-1</sup> )	6.5 $\pm$ 1.2	11 $\pm$ 2.0	18 $\pm$ 2.0	24 $\pm$ 2.0	28 $\pm$ 2.9	22 $\pm$ 3.1	13 $\pm$ 2.0	NA	6.5 $\pm$ 1.2
% change		69	177	269	331	238	100	NA	0
Mean BP (mmHg)	92 $\pm$ 2.5	95 $\pm$ 2.9	96 $\pm$ 3.3	96 $\pm$ 3.1	106 $\pm$ 3.3	104 $\pm$ 2.9	101 $\pm$ 3.3	NA	97 $\pm$ 2.2
% change		3	4	4	15	13	10	NA	5
Heart rate (beat·min <sup>-1</sup> )	64 $\pm$ 5.3	70 $\pm$ 4.7	65 $\pm$ 4.5	66 $\pm$ 4.7	74 $\pm$ 4.7	76 $\pm$ 5.1	80 $\pm$ 5.3	81 $\pm$ 6.3	58 $\pm$ 3.7
% change		9	2	3	16	19	25	27	-9
MCAv (cm·s <sup>-1</sup> )	65 $\pm$ 8.0	76 $\pm$ 7.6	81 $\pm$ 9.6	74 $\pm$ 9.4	79 $\pm$ 10.2	51 $\pm$ 6.9	49 $\pm$ 6.3	37 $\pm$ 5.3	58 $\pm$ 6.8
% change		16	23	14	20	-22	-26	-43	-11
ETCO <sub>2</sub> (kPa)	5.2 $\pm$ 0.2	6.0 $\pm$ 0.2	6.5 $\pm$ 0.2	6.4 $\pm$ 0.2	6.4 $\pm$ 0.2	4.3 $\pm$ 0.6	4.4 $\pm$ 0.4	NA	5.0 $\pm$ 0.2
% change		16	25	23	23	-16	-15	NA	-4

was required to rate his level of discomfort as: none, mild, moderate, severe or intolerable in each of the categories: nausea, breathlessness, faintness and headache.

#### TEST TERMINATION CRITERIA

The test would be terminated:

- at the subject's request; on a subjective questionnaire response of 'intolerable' to any aspect;
- on failure of any equipment used to monitor withdrawal variables;
- on recording end-tidal CO<sub>2</sub> (ETCO<sub>2</sub>) > 8.5 kPa for more than five consecutive breaths;
- if the subject began to vomit;
- if the subject fainted or requested assistance feeling faint;
- on subjective signs of impending panic;
- if BP was greater than either a systolic of 180 or a diastolic of 110 mmHg, sustained for over 1 min.

#### STATISTICAL ANALYSIS

The relative percentage change from baseline values in six physiological parameters (respiratory rate, heart rate, BP, MCAv, ETCO<sub>2</sub> and respiratory minute volume) was calculated at different time points. The relative percentage change in mean MCAv was calculated from the value immediately preceding and those following the switch from high CO<sub>2</sub> for Test 2 and Test 3. Data were compared using either paired or unpaired, unequal variance Student's *t*-tests. Differences were considered significant if *P* < 0.05.

## Results

### SYMPTOMS

All subjects completed each of the three tests successfully; there were no withdrawals and no subject fainted or vomited or was otherwise incapacitated at any stage. Four subjects did not report any symptoms throughout the tests. Fifteen of the 20 subjects reported symptoms during high-CO<sub>2</sub> breathing, compared with seven reporting mild or moderate symptoms of faintness, with headache or nausea after the gas switch and performing Valsalvas.

### PHYSIOLOGICAL PARAMETERS

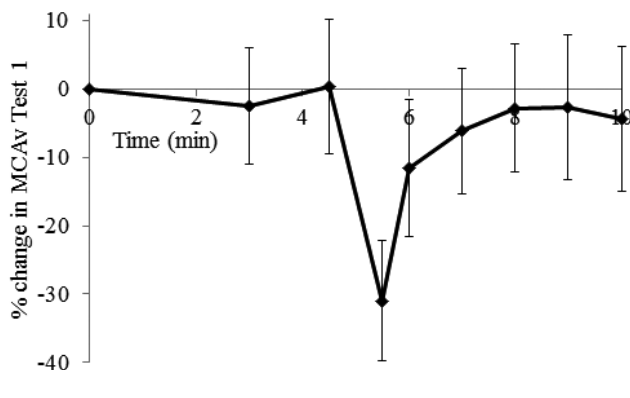
Tables 2 and 3 show absolute and percentage change in the mean physiological parameter values at defined points through Tests 2 and 3 respectively. No data for BP, respiratory minute volume and ETCO<sub>2</sub> are reported for the time at which the Valsalvas were performed, as the subjects were occluding the exhale valve, making these measurements inaccurate.

### MCAv

Figures 1, 2 and 3 show the change in mean MCAv during Tests 1, 2 and 3 given as a percentage change from the baseline measurement. During Test 1 mean MCAv dropped by 31% after performing the Valsalvas, and then recovered towards baseline values. The changes in mean MCAv were very similar for both Test 2 and Test 3: MCAv increased to reach a peak of about 23% above baseline at 5 min of

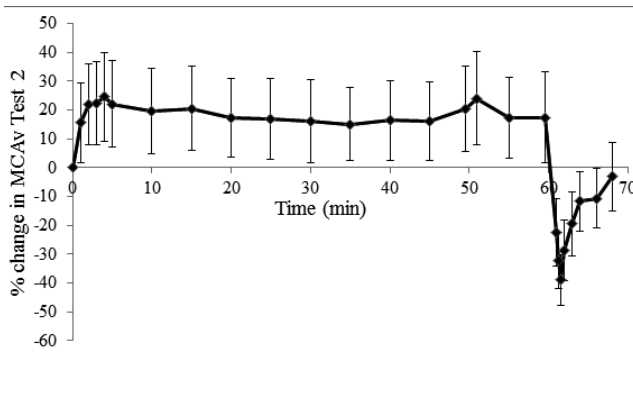
**Figure 1**

% change in mean MCAv (95% CI shown) from baseline during Test 1



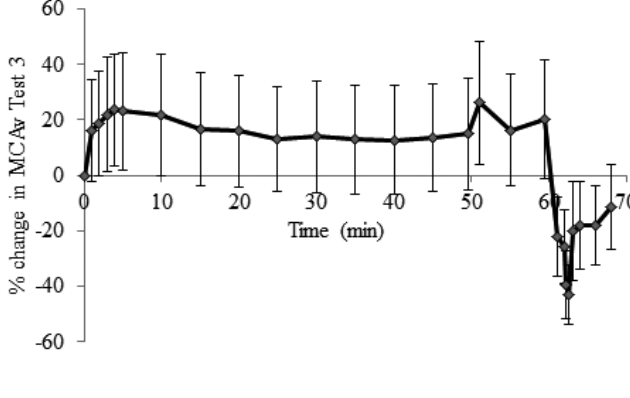
**Figure 2**

% change in mean MCAv (95% CI shown) from baseline during Test 2



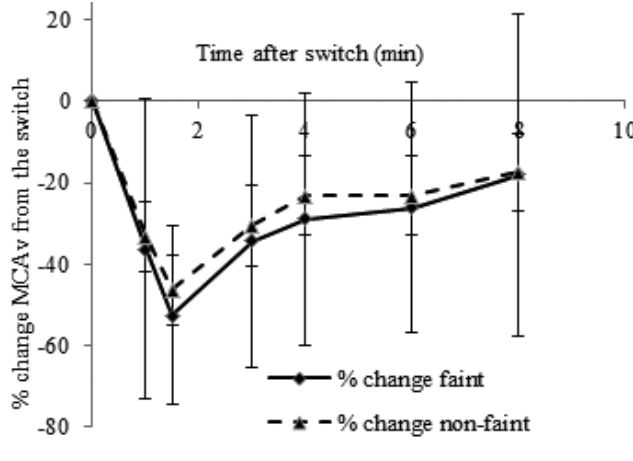
**Figure 3**

% change in mean MCAv (95% CI shown) from baseline during Test 3



**Figure 4**

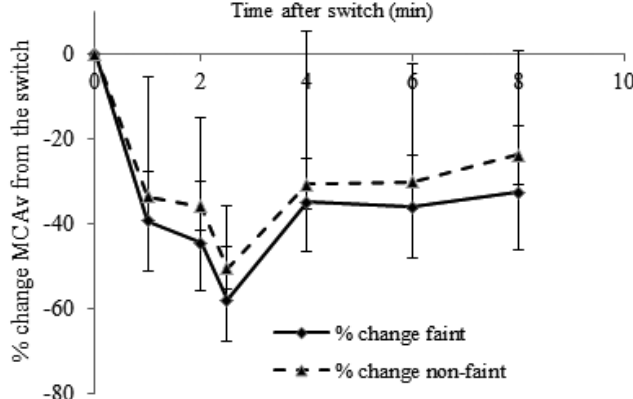
% change in mean MCAv (95% CI shown) post switch to O<sub>2</sub>; Test 2 comparing faint and non-faint subjects (not significant)



breathing high CO<sub>2</sub>. There was then a decline; the mean MCAv was around 16% above baseline from 15 min until the subjects stood up when it increased to around 25%. The switch from high CO<sub>2</sub> in both tests caused a drop to around 23% less than the baseline and there were further decreases in mean MCAv with Valsalvas before a recovery towards baseline.

**Figure 5**

% change in mean MCAv (95% CI shown) post switch to air; Test 3 comparing faint and non-faint subjects (not significant)



The percentage change in mean MCAv taken from the value immediately preceding the switch was calculated for Tests 2 and 3. During Test 2 the mean MCAv dropped by 34% when switching from high CO<sub>2</sub> to O<sub>2</sub>, by a further 14% after performing the Valsalvas and then recovered towards baseline over the final 5 min. During Test 3 the mean MCAv dropped by 35% when switching from high CO<sub>2</sub> to air, by a further 3% when switching to O<sub>2</sub> and then by 14.5% when performing the Valsalvas. Recovery towards baseline values then continued over the final 5 min.

**MEAN MCAv WITH OR WITHOUT FAINTNESS**

Figures 4 and 5 show the percentage change in MCAv taken from the value immediately preceding the switch from high CO<sub>2</sub> for Test 2 and Test 3. Subjects are grouped as those

who did or did not report feeling faint after the switch and/or Valsalvas. The drop in MCAv for the subjects reporting faintness or increased faintness following the switch was



generally greater than the drop in MCAv for those who did not. However, this difference was not statistically significant.

## Discussion

### CHANGES WHILE BREATHING HIGH CO<sub>2</sub>

The most frequently reported symptom while breathing high CO<sub>2</sub> was breathlessness, followed by headache and faintness, which is in agreement with our previous study.<sup>2</sup> The symptoms of breathlessness and headache were evenly reported between Tests 2 and 3 regardless of which was performed first, whereas symptoms of faintness were more likely to be reported on the first test with high CO<sub>2</sub> rather than on the second; possibly a learning effect, as subjects knew what to expect and therefore did not report as faint. Cerebral blood flow has been shown to increase when breathing 5% CO<sub>2</sub>.<sup>2,4</sup> In the present study, mean MCAv increased by 23% after 5 min of breathing 5% CO<sub>2</sub>.

### CHANGES AFTER SWITCHING FROM HIGH CO<sub>2</sub>

Transient mild or moderate symptoms of faintness, headache or nausea occurring after the switch to either air or O<sub>2</sub> were reported by seven subjects. Faintness or increased faintness was the most commonly reported symptom, being reported by 7/20 subjects (35%, 95% CI 15–59%). This is a higher incidence than in our previous study where 7/34 (20%, 95% CI 8–38%) subjects reported mild to moderate faintness after the switch to O<sub>2</sub>. Therefore, proportionately more subjects felt faint after the switch and Valsalva than just from the switch alone, but this was not statistically significant.

Three subjects reported mild headache starting after the switch to O<sub>2</sub> on Test 2; however, this was also around the same time as they were performing Valsalvas. Activity-related headaches are well documented and are reported by sufferers during or shortly after a physical activity which typically incorporates a Valsalva, such as coughing, sneezing or straining while lifting heavy loads.<sup>5,6</sup> These 'cough headaches' are generally short-lived, lasting between 1 s and 30 min, without other associated symptoms.<sup>5</sup> It would be unlikely that this would in any way prevent safe escape from a submarine. Any headaches reported by subjects in our trial were resolved by the end of the tests.

Pre-fainting symptoms include headache and nausea and these additional symptoms were reported by subjects who also reported feeling faint. Fainting or feeling faint is associated with a decrease in MCAv and this is most commonly provoked in the standing position.<sup>1</sup> Hyperoxia and hypocapnia both reduce MCAv and the decrease seen in our study could have been caused by cerebral vasoconstriction due to hyperoxia from switching to 100% O<sub>2</sub> (Test 2) and/or the return to normocapnia from ceasing to breathe high CO<sub>2</sub> (Tests 2 and 3) – the 'CO<sub>2</sub>-off' effect.

Differentiating between the symptoms reported after the

switch from high CO<sub>2</sub> and those symptoms reported after the Valsalvas was difficult, because of the exact timings of administering the questionnaire at 1 min intervals at this part of the trial. However, in the debrief at the end of the tests, some subjects reported definite symptoms after Valsalvas and two subjects noted light-headedness after performing Valsalvas alone (Test 1).

### MCAv AND SYMPTOMS FOLLOWING VALSALVA MANOEUVRES

Other studies have reported that decreases in MCAv of about 50% are associated with faintness. Passive head-up tilt of healthy subjects reduces MCAv and induces feelings of faintness.<sup>7</sup> In our study, the drop in percentage MCAv for the group that noted faintness or increased faintness following the switch was, in general, greater than the drop for those that did not. However, in contrast to our previous study, this difference was not significant. Our previous study demonstrated a significant difference in percentage drop in mean MCAv between the subjects who had symptoms of faintness after the switch to O<sub>2</sub> and those who did not report faintness (decrease in MCAv of 51% versus 44% respectively).<sup>2</sup>

Valsalvas performed in the standing position reduce the mean MCAv to 50% of the value obtained during supine rest, whereas during supine Valsalvas the reduction in MCAv is of the order of 35%.<sup>3</sup> The authors concluded that in the upright position, expiratory straining may critically compromise cerebral perfusion.

In our previous study, where the subjects switched to breathing 100% O<sub>2</sub> but did not perform Valsalvas, there was a large drop in percentage MCAv in the first minute following the switch to O<sub>2</sub> (similar to the effect observed with this study) – any further drop in percentage MCAv after the first minute following the switch was not significant when compared with the drop in the first minute. Therefore, it is assumed that in our present study, the significant drop in percentage MCAv observed following Valsalvas was, in fact, due to the Valsalvas and not to a continued/prolonged effect of the switch to O<sub>2</sub>. Although Valsalvas exacerbated the decrease in cerebral blood flow following the switch from high CO<sub>2</sub>, the accompanying symptoms of faintness, headache and nausea were transient and not incapacitating.

### RELEVANCE TO SUBMARINE TOWER ESCAPE

The procedure for performing the Valsalvas was a compromise between the operational scenario and achieving a reproducible effect. In submarine escape exercises conducted by RN instructors, the observed method of ear-clearing varies markedly between individuals but is likely to be more frequent.

During the debriefing of the subjects following each test there was a range of comments from the subjects regarding

how they felt, from “*nothing of note*” and “*didn’t notice any difference*” to comments that the transient faintness after the switch and Valsalva was “*pretty grim, I couldn’t have done any physical work at that point*” and “*I couldn’t have made a decision*”. Despite these reports, all subjects successfully completed the six Valsalvas. This required them to coordinate repeatedly closing off the mouthpiece outlet with their hand while simultaneously ensuring that they were reaching the required exhalation pressure, and following instruction on when to inhale and exhale. Submariners are trained in using escape towers and the procedures should be familiar. Following pressurisation, the submariner will be ascending through the water column to the surface, with no physical work or decision making to perform. Submariners simply need to breathe normally during the ascent and by the time they reach surface any transient faintness or headache due to changes in the breathing gas/Valsalva manoeuvres should have resolved.

In the escape scenario, it is likely that the submariners will be at least partially immersed and thus subject to hydrostatic pressure which should help to support systemic BP and cerebral perfusion. The time from the start of flooding of the tower to the start of compression can take up to 190 s, depending on depth and type of escape tower, and this period may give a protective effect on cerebral circulation, reducing the risk of fainting in the escape tower.

Our study examined the effect of acute high CO<sub>2</sub> exposure. The effect of switching to air from a chronic high CO<sub>2</sub> exposure, as may be experienced in a DISSUB environment, is unknown.

### Conclusions

The hypothesis that Valsalva manoeuvres would reduce MCAv over and above that caused by a switch from breathing high CO<sub>2</sub> was upheld; there was a further 14% decrease in MCAv. The percentage drop in MCAv occurring following the switch from high CO<sub>2</sub> to 100% O<sub>2</sub> (34%) was similar to that occurring following the switch to air (35%). Therefore, a ‘CO<sub>2</sub>-off’ effect seems the best explanation of the observed results.

Seven subjects reported faintness after the gas switch and performing Valsalvas, some with additional symptoms of headache or nausea. Those subjects who reported feeling faint had a slightly lower mean MCAv than those who did not, but this was not statistically significant. Transient faintness or headache may occur in the submarine escape tower during pressurisation, but this should be short-lived and not be incapacitating.

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*Fiona Seddon, Julian Thacker, Karen Jurd, Geoffrey Loveman  
QinetiQ, Maritime Life Support, Haslar Marine Technology Park,  
Hampshire, UK*

### Address for correspondence:

*Fiona Seddon,  
QinetiQ, Maritime Life Support,  
Haslar Marine Technology Park,  
Haslar Rd. Gosport,  
Hampshire,  
UK PO12 2AG  
Phone: +44-(0)2392-335157  
Fax: +44-(0)2392-335197  
E-mail: <fmseddon@qinetiq.com>*

# Decompression management by 43 models of dive computer: single square-wave exposures to between 15 and 50 metres' depth

Martin DJ Sayer, Elaine Azzopardi and Arne Sieber

## Abstract

(Sayer MDJ, Azzopardi E, Sieber A. Decompression management by 43 models of dive computer: single square-wave exposures to between 15 and 50 metres' depth. *Diving and Hyperbaric Medicine*. 2014 December;44(4):193-201.)

**Introduction:** Dive computers are used in some occupational diving sectors to manage decompression but there is little independent assessment of their performance. A significant proportion of occupational diving operations employ single square-wave pressure exposures in support of their work.

**Methods:** Single examples of 43 models of dive computer were compressed to five simulated depths between 15 and 50 metres' sea water (msw) and maintained at those depths until they 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 various levels of decompression were indicated or exceeded. Each depth profile was replicated three times for most models.

**Results:** Decompression isopleths 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. For dives requiring decompression, computers were predominantly more conservative than tables across the whole depth range tested. There was considerable variation between models in the times permitted at all of the depth/decompression combinations.

**Conclusions:** The present study would support the use of some dive computers for controlling single, square-wave diving by some occupational sectors. The choice of which makes and models to use would have to consider their specific dive management characteristics which may additionally be affected by the intended operational depth and whether staged decompression was permitted.

## Key words

Computers – diving, occupational diving, decompression, dive profile, decompression tables

## Introduction

Dive computers can be accepted in some occupational diving sectors as tools for managing decompression.<sup>1</sup> However, the choice of which dive computer could be used for occupational diving is difficult because the number of models available is considerable. The choice is further complicated by the many different decompression algorithms employed in dive computers, with some being modified by manufacturers in unspecified ways.<sup>2</sup> In Europe, standards and normatives that underpin CE marking for dive computers do not stipulate operational limits for decompression management.<sup>3,4</sup>

There are many potential advantages to using dive computers for occupational diving. They can control diver ascent rates and calculate decompression based on actual (rather than predicted) multi-level pressure exposures. Most have dive profile storage and download capabilities;<sup>5</sup> some have additional features such as: calculating for the use of mixed gases; wireless display of cylinder pressures and heart-rate monitoring, as well as a range of user settings (seawater/freshwater, conservatism, altitude, etc.).<sup>2</sup> However, without a detailed knowledge of how the dive computers are managing decompression, diving supervisors will not have the relevant information on which to base any management choices about which models could be accepted for operational use within a regulated occupational diving environment. Whereas conservatism of decompression schedules may be more

important for some diving operations, maximising bottom time safely may be the predominant reason for choice in others.

There have been a number of studies that have compared the performance of dive computers in managing decompression.<sup>6–8</sup> The present study follows previous ones in that it compares the performance of a range of dive computers standardised across a number of pressure/time profiles. However, all the models assessed are relatively modern being either currently on sale or remaining in common use in the UK and Europe.<sup>2,9</sup> Comparisons were made of a series of single, square-wave profile dives for the depth range of 15 to 50 metres' sea water (msw). It is assumed that the square-wave profile is more typical of most occupational diving operations where the divers are working on a single task at a single depth before returning to the surface. The chosen depth range is assumed to be representative of most compressed air scuba diving operations where decompression obligations become apparent.

## Methods

Single examples of 43 models of dive computer that are in common use in the UK (Table 1) were set to default settings and all were in sea water mode. The computers were compressed simultaneously to five simulated depths (nominally 15, 20, 30, 40 and 50 msw). In each test, all 43

**Table 1**

The 43 models of computer employed in the present study, listed alphabetically by brand name

<b>Brand</b>	<b>Model</b>
Apeks	Pulse
Apeks	Quantum
Beauchat	Voyager
Cressi Sub	Archimede 2
Cressi Sub	Edy II
Mares	Icon HD
Mares	Nemo Sport
Mares	Nemo
Mares	Nemo Excel
Mares	Nemo Air
Mares	Nemo Wide
Mares	Puck Wrist
Mares	Puck Air
Oceanic	Atom 2
Oceanic	Datamask Hud
Oceanic	Pro Plus 2
Oceanic	Veo 250
Oceanic	VT 3
Scubapro	Xtender
Seeman	XP 5
Suunto	Cobra 2
Suunto	Cobra 3
Suunto	D9
Suunto	D6
Suunto	D4
Suunto	Stinger
Suunto	Spyder
Suunto	Vyper
Suunto	Vyper 2
Suunto	Vyper Air
Suunto	Vytec DS black
Suunto	Vytec Silver
Tusa	DC Hunter
Tusa	DC Sapience
Uemis	SDA
Uwatec	Aladin One
Uwatec	Aladin Prime
Uwatec	Aladin Tec 2 G
Uwatec	Galileo Luna
Uwatec	Galileo Sol
Uwatec	Galileo Terra
Uwatec	Smart Tec
Uwatec	Smart Com

computers were immersed in fresh water to a depth of 20 cm in a tank located inside a standard two-compartment therapeutic recompression chamber (*Divex* chamber of 2,000 mm diameter). The chamber was compressed with air using manual control to the simulated nominal depth employing the fastest descent rate possible through the compression valve being fully opened each time. Depth was maintained and monitored using calibrated chamber gauges (+ 0.032 msw average error rising (0–50 msw,  $n = 5$ ); + 0.020 msw average error falling (50–0 msw,  $n = 5$ ); calibrated by a UKAS Calibration Laboratory-certified *Druck* MS-022

to the test standard BS EN 837–1:1998); there were some minor manual adjustments for depth ( $\pm 0.1$  msw) for the effects of temperature changes following compression and before stabilisation occurred as controlled by the chamber's environmental control unit. Barometric pressure before and during the tests was not recorded. The chamber is situated approximately 3 m above sea-level.

The chamber was maintained at nominal depth until it was known that all computers had registered over 30 minutes of decompression. At each depth, and for each model, downloaded data from the computers were used to collate the dive duration at which the unit was still registering “no decompression”, and the time of the dive 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; intervals between tests were at least 72 h to allow for the effects of the previous test to clear.

Depth/time isopleth relationships were generated for all the decompression end points examined over a 15 to 50 msw depth range for every model of computer. These were compared against isopleths constructed based on the Royal Navy Physiology Laboratory air decompression table 11 (RNPL 11), the Defence and Civil Institute of Environmental Medicine (DCIEM) air decompression tables, and the Sub-Aqua Association's modified version of the Bühlmann 1986 air decompression tables.<sup>10–12</sup> Linear interpolation was used to provide dive times where table increments did not match the nominal test depths.

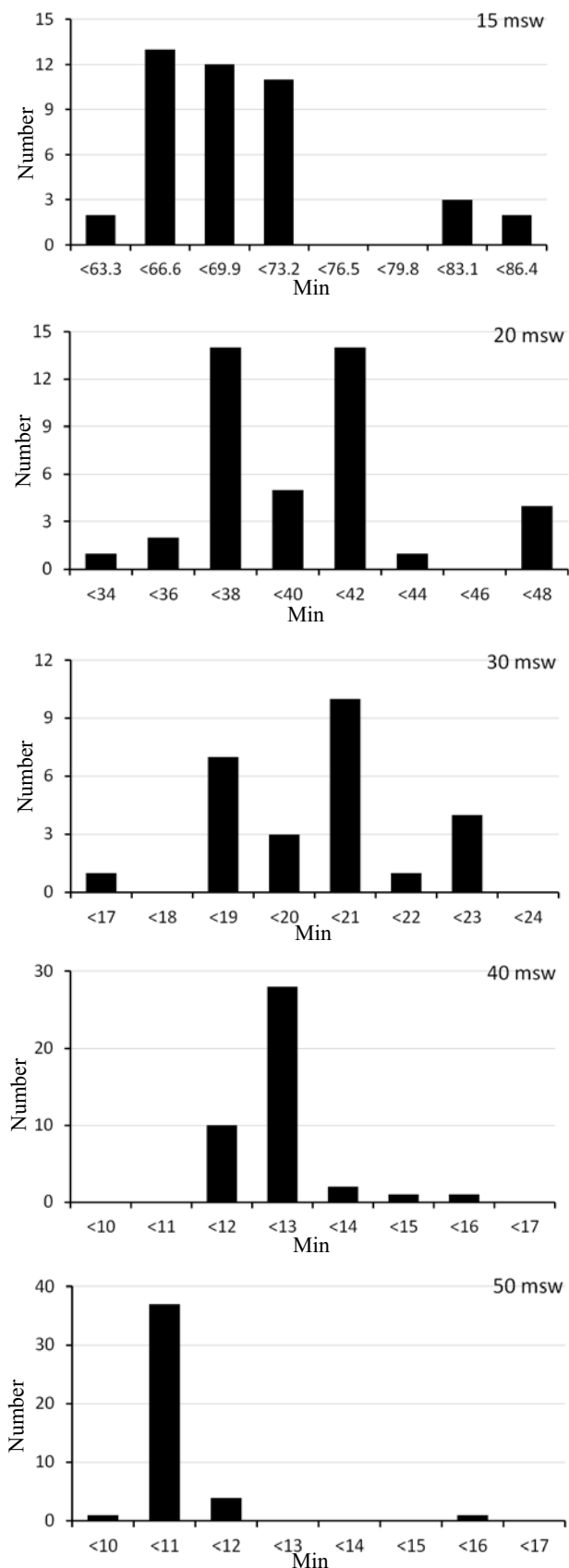
Frequency analyses were conducted based on the numbers of computer models falling within the time ranges required to reach designated decompression endpoints. The times recorded to reach all of the decompression/depth endpoints were converted into values of per cent deviation from the overall means. For decompression and non-decompression, the computers and tables were ranked based on their mean per cent deviation values.

The effects of two compression regimes that produced descent rates equivalent to 5.0–7.5 and 16.7–20.0 m·min<sup>-1</sup>, were tested on nine of the computer models (*Uwatec Galileo Sol*, *Uwatec Aladin Prime*, *Mares Nemo Wide*, *Mares Nemo*, *Suunto D9*, *Suunto Vyper 2*, *Oceanic Atom 2*, *Cressi Sub Edy II*, *Apeks Quantum*) at depths of 20 and 40 msw. The times on the downloaded profiles that indicated the maximum time for no-decompression (the time just before the recording showed a required decompression stop) and those needed to generate 10, 20 and 30 min of decompression were compared between the two descent rates at each depth using Student's *t*-test for paired samples.

The water temperature in all the tests was recorded using an immersed *Gemini Tiny Tag* data logger. A record of any computer malfunctions or failures was maintained.

**Figure 1**

Frequency analysis of number of tested computer units displaying maximum no-decompression stop times (Min) for square-wave dive profiles to maximum depths of 15–50 msw; for each unit,  $n = 1-3$  for each depth test



**Results**

Differences between replicated trials were examined for the 0, 5, 10, 20 and 30 minutes of decompression intervals (Table 2). In 95.5% of the comparisons, variation was within 10% of the average time for all tests; variation was zero in 47.4% of the comparisons examined. In 0.4% of comparisons, variation was greater than 25% of the average time. The maximum recorded variations for the five decompression times ranged between 19.0 and 33.3%. There was no consistency in the variations observed in terms of individual units, specific tests or minor depth changes.

A slower descent rate generated significantly longer times permitted before each of the nominal decompression end points (no-stop, 10, 20, 30 min) was reached at both depths tested (20 and 40 msw;  $P < 0.01$  and  $n = 9$  in all cases). The differences in the times to reach each end point were broadly attributable to the additional time taken during slower descents.

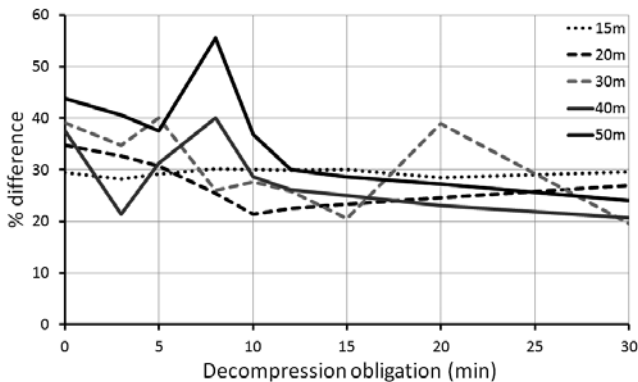
Frequency analyses showed that there was considerable variation in the times recorded by the computer units for all the depth/decompression combinations; an example for no-decompression-stop values is shown in Figure 1. With two exceptions (no stops and 8 minutes of decompression at 50 msw, differences of > 40% recorded) the variances between the maximum and minimum times permitted to reach all the nominal decompression end points at all the five test depths were between 20 and 40% of the maximum recorded times (Figure 2). The largest differences in permitted times were not always attributable to the same computer units. The trends for the 15 and 20 msw tests tended to be more consistent across the range of decompression endpoints tested (Figure 2).

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 (and particularly at 15 msw), but less conservative between 30–50 msw (Figure 3). However, these differences were not always consistent between computer models. Whereas in some comparisons there were relatively constant differences between the computers at all depths (Figure 4), in others there were quite large differences at shallower depths but these were not evident when the tests were deeper (Figure 5).

Differences in decompression management were also present across the depth range tested in per cent deviation from the mean. For no-decompression dives the *Oceanic Veo 250*, for example, gave times that were less than the mean at 15, 20 and 50 msw, but above the mean at 30 and 40 msw (Table 3). The *Mares Nemo Sport* was among the most conservative computers when tested at 15 msw, but was the least conservative at 40 and 50 msw (Table 3). Similar anomalies were present in the decompression dives; an example is the

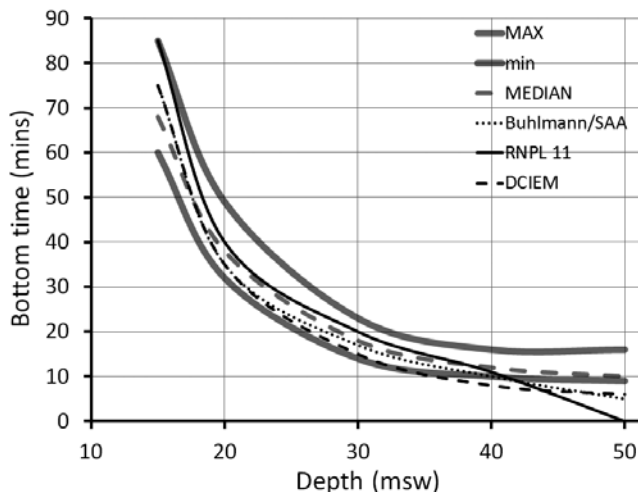
**Figure 2**

The difference in total dive times required to generate decompression penalties of 0–30 min expressed as a % of the maximum permissible time. Values are for 43 computer models tested across a depth range of 15–50 msw ( $n = 1-3$  for each point)



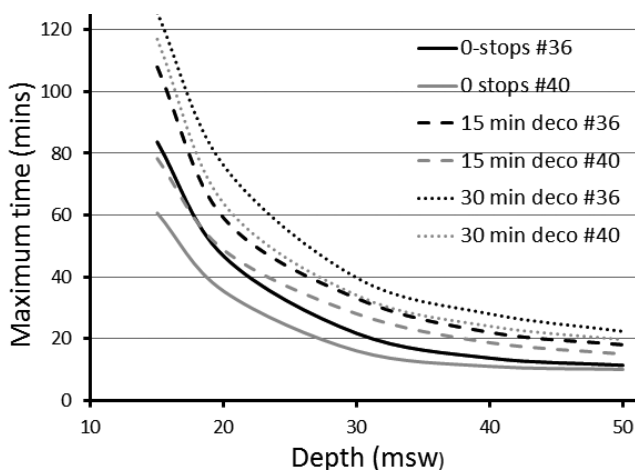
**Figure 3**

Isopleth relationships for the maximum times permitted by 43 models of dive computer and two air decompression tables before the dive would require any decompression over a depth range of 15 to 51 msw; all dive profiles were square-wave; isopleths for the dive computers are pooled to show maximum (MAX), minimum (min) and median values for all 43 models



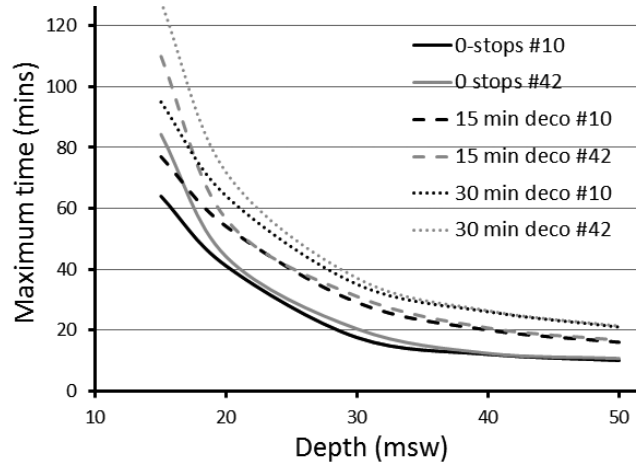
**Figure 4**

Decompression isopleths for two models of dive computer (#36 = *Oceanic Datamask HUD* and #40 = *Apeks Quantum*) compared at three levels of decompression stress (no-stops; 15 min of deco; 30 min of deco)



**Figure 5**

Decompression isopleths for two models of dive computer (#10 = *Mares Icon HD* and #42 = *Seeman XP5*) compared at three levels of decompression stress (no-stops; 15 min of deco; 30 min of deco)



**Table 2**

Variation in times recorded within three replicate tests for 41 models of decompression computers compared at 25 combinations of nominal depth and decompression interval. Values are for the number of test runs falling within 5% variation groups

Decompression time (mins)	% Variation in times recorded/average time					Max	n
	0	0.1–5	5.1–10	10.1–25	> 25		
0	79	51	60	7	3	33.3	200
5	79	60	30	9	1	26.5	179
10	107	70	14	9	0	19.0	200
20	97	79	18	7	0	19.7	201
30	104	84	7	6	0	22.3	201
Total	466	344	129	38	4		981
%	47.5	35.0	13.1	3.9	0.4		

**Table 3**

Mean maximum times ( $n = 1-3$ ;  $n = 3$  in 187/215 tests) permitted by 43 dive computers and three decompression tables without having to undertake decompression stops at each of five nominal depths (15–50 msw; the RNPL 11 recommended decompression for any dive to 50 msw). For each depth, the mean no-decompression times were expressed as % deviation from the mean value; the final table ranking is based on the overall mean % deviations; blank cells = missing data

	Mean maximum no-decompression times (min; $n = 1-3$ )					Overall mean deviation (%; $n = 2-5$ )
	15 msw	20 msw	30 msw	40 msw	50 msw	
<b>Decompression table</b>						
DCIEM	75.0	35.0	15.0	8.0	6.0	-19.1
Buhlmann/SAA	75.0	35.0	17.0	10.0	5.0	-15.6
RNPL 11	85.0	40.0	20.0	11.0	0.0	-15.4
<b>Computer model</b>						
UEMIS SDA	70.0	33.3	15.7	11.5	8.7	-9.5
APEKS Quantum	60.7	35.3	16.0	11.0	10.0	-8.9
TUSA DC Sapience	60.0			11.0	10.0	-7.3
TUSA DC Hunter	65.0	37.0	17.0	11.3	10.0	-5.2
SCUBAPRO Xtender	65.3	37.0	17.0	11.3	10.0	-5.1
CRESSI Sub Archimede 2	64.3	38.0	17.0	11.0	10.3	-4.8
CRESSI Sub Edy II	64.7	38.0	17.0	11.3	10.3	-4.1
APEKS Pulse	65.0	38.0	17.0	11.0	10.7	-3.9
MARES Nemo Excel	65.3	37.0	17.3	12.3	10.0	-3.1
OCEANIC Veo 250	65.0	35.0	19.0	12.5	9.7	-2.8
MARES Nemo	66.7	37.3	17.3	12.3	10.0	-2.5
MARES Nemo Wide	66.3	37.7	17.3	12.3	10.0	-2.5
MARES Puck wrist	66.3	37.7	17.3	12.3	10.0	-2.5
MARES Puck Air	65.7	37.3	17.7	12.3	10.0	-2.5
MARES Nemo Air	66.7	37.5	17.3	12.3	10.0	-2.4
MARES Icon HD	64.0	41.0	17.5	12.0	10.0	-1.8
UWATEC Smart Com	66.0	38.0	17.3	12.7	10.7	-0.5
UWATEC Aladin prime	67.3	38.0	17.0	12.7	10.7	-0.5
UWATEC Aladin Tec 2 G	67.3	38.0	17.3	12.7	10.7	-0.1
UWATEC Galileo Sol	67.3	38.3	17.3	12.7	10.7	0.1
UWATEC Galileo Terra	67.3	38.3	17.3	12.7	10.7	0.1
UWATEC Galileo Luna	67.7	38.7	17.3	12.7	10.7	0.3
UWATEC Smart Tec	67.3	38.3	17.7	12.7	10.7	0.4
BEAUCHAT Voyager	69.3	42.0	18.3	11.7	10.0	0.6
SUUNTO Vyper Air	70.3	40.7	19.3	12.0	10.0	1.8
SUUNTO Spyder	72.0	41.7	19.0	11.7	10.0	1.9
SUUNTO Cobra 3	70.0	41.0	19.3	12.0	10.0	1.9
SUUNTO Vyper	70.0	41.0	19.3	12.0	10.0	1.9
SUUNTO Cobra 2	69.3	41.0	19.7	12.0	10.0	2.1
SUUNTO Vyper 2	70.3	41.0	19.7	12.0	10.0	2.4
SUUNTO Stinger	72.3	41.3	19.0	12.0	10.0	2.4
SUUNTO Vytec DS black	69.7	41.0	20.0	12.0	10.0	2.5
SUUNTO D4	70.0	41.0	20.0	12.0	10.0	2.6
SUUNTO Vytec silver	70.0	41.0	20.0	12.0	10.0	2.6
SUUNTO D9	70.3	41.0	20.0	12.0	10.0	2.7
SUUNTO D6	70.3	41.0	20.0	12.0	10.0	2.7
UWATEC Aladin One	68.0				11.0	4.2
SEEMAN XP 5	84.3	44.0	20.3	12.3	10.7	10.5
OCEANIC VT 3	82.3	46.3	21.5	12.0	11.0	12.5
OCEANIC Pro Plus 2	80.3	46.7	21.7	14.0	10.7	14.9
MARES Nemo Sport	65.0	38.3	19.0	15.3	15.3	15.0
OCEANIC Datamask Hud	82.7	46.5	22.0	13.7	11.3	16.7
OCEANIC Atom 2	83.7	46.7	21.7	13.7	11.3	16.7
Mean	69.7	39.5	18.4	12.0	9.9	

**Table 4**  
The mean % deviation ( $n = 1-3$ ;  $n = 3$  in 561/645 tests) from the mean maximum times taken to generate three levels of decompression (10, 20 and 30 min) at each of five nominal depths (15–50 msw) for 43 models of dive computer and two air decompression tables (shaded); the results are ranked based on the overall mean % deviations; blank cells = missing data

Computer model	15 msw			20 msw			30 msw			40 msw			50 msw			Overall			
	10 min	20 min	30 min	10 min	20 min	30 min	10 min	20 min	30 min	10 min	20 min	30 min	10 min	20 min	30 min	mean	mean		
APEKS Quantum	-14.54	-12.29	1.17	-8.55	-8.74	-8.70	-8.56	-8.66	-9.38	-6.17	-9.38	-6.94	-16.51	-9.77	-8.83	-11.71	-7.49	-6.28	
MARES Nemo Excel	-9.28	-15.29	-15.55	-13.37	-5.34	-6.38	-12.87	-8.20	-4.11	-4.05	-5.74	-10.94	-10.94	0.41	0.03	-3.50	-14.60	-4.67	
MARES Nemo	-8.06	-14.29	-14.10	-12.15	-4.66	-6.38	-12.87	-7.97	-4.11	-5.83	-7.18	-10.94	-10.94	0.41	0.03	-3.50	-14.60	-4.67	
OCEANIC Veo 250	-10.90	-11.62	-11.80	-11.44	-8.74	-11.58	-15.26	-11.86	-2.05	-6.71	-4.26	-10.94	-10.94	0.41	0.03	0.72	-5.11	-6.70	
MARES Puck Air	-8.87	-14.63	-14.68	-12.73	-4.66	-6.38	-12.87	-7.97	-6.54	-1.02	-4.05	-3.87	-10.94	0.41	0.03	-3.50	-14.60	-4.67	
MARES Nemo Air	-7.66	-13.96	-14.39	-12.00	-5.00	-6.38	-12.39	-7.92	-6.54	-2.05	-4.05	-4.21	-10.94	0.41	-1.24	-3.92	-14.60	-4.67	
MARES Puck wrist	-8.47	-14.29	-14.68	-12.48	-3.98	-5.81	-12.39	-7.39	-5.27	-1.02	-4.05	-3.45	-10.94	0.41	0.03	-3.50	-14.60	-4.67	
MARES Nemo Wide	-8.06	-13.96	-14.39	-12.14	-3.98	-4.65	-11.91	-6.84	-5.27	-1.02	-4.05	-3.45	-10.94	0.41	-1.24	-3.92	-14.60	-4.67	
MARES Icon HD	-11.30	-16.96	-17.85	-15.37	2.15	-1.18	-8.08	-2.37	-9.06	-2.57	-6.71	-6.11	-5.38	-3.95	-5.04	-4.79	-7.49	-2.30	
SCUBAPRO Xtender	-8.06	-3.95	11.84	-0.06	-4.66	-4.07	-3.77	-4.17	-2.75	-2.05	-5.83	-3.54	-5.38	-3.95	-5.04	-4.79	-7.49	-5.75	
TUSA DC Sapience	-13.73	-9.96	3.77	-6.64															
BEAUCHAT Voyager	-4.01	-8.96	-19.29	-10.75	-1.93	-4.65	-8.56	-5.05	1.04	-4.11	-7.60	-3.56	0.19	0.41	-5.04	-1.48	-0.37	-4.47	
TUSA DC Hunter	-8.06	-3.95	11.84	-0.06	-3.98	-4.07	-3.77	-3.94	-1.48	-1.02	-4.05	-2.18	-5.38	-3.95	-5.04	-4.79	-7.49	-3.22	
CRESSI-SUB Archimede 2	-8.47	-4.62	11.26	-0.61	-3.29	-2.92	-2.33	-2.85	-1.48	-1.02	-4.05	-2.18	-5.38	-3.95	-5.04	-4.79	-7.49	-3.11	
CRESSI-SUB Edy II	-8.87	-4.29	11.26	-0.63	-3.98	-2.92	-2.33	-3.07	-1.48	-1.02	-4.05	-2.18	-5.38	-3.95	-3.77	-4.37	-7.49	-2.95	
SUUNTO Stinger	-1.18	0.72	-7.76	-2.74	-1.93	-3.49	-2.81	-2.75	-5.27	-2.05	-3.16	-3.49	-5.38	0.41	-1.24	-2.07	-5.11	-2.72	
UEMIS SDA	-0.37	8.05	13.28	6.99	-1.25	1.13	3.89	1.26	-12.85	-2.05	-2.27	-5.72	-8.16	-1.77	-3.14	-4.36	-14.60	-2.43	
UWATEC Smart Com	-5.63	-0.29	1.17	-1.58	-3.29	-2.92	-1.38	-2.53	2.31	-4.11	-6.71	-2.84	5.76	-2.50	-5.04	-0.59	6.75	-1.34	
UWATEC Aladin Tec 2 G	-4.42	0.72	1.46	-0.75	-3.29	-2.92	-0.90	-2.37	2.31	-4.11	-6.71	-2.84	5.76	-2.50	-5.04	-0.59	6.75	-1.14	
UWATEC Aladin Prime	-4.82	1.05	1.17	-0.87	-2.61	-2.34	-0.90	-1.95	2.31	-4.11	-5.83	-2.54	5.76	-3.95	-5.04	-1.08	6.75	-1.12	
UWATEC Galileo Terra	-4.82	0.72	2.04	-0.69	-3.98	-3.49	-1.85	-3.11	2.31	-4.11	-5.83	-2.54	5.76	-2.50	-5.04	-0.59	6.75	-1.11	
APEKS Pulse	-7.25	-4.29	11.84	0.10	-3.29	-2.92	-2.33	-2.85	-1.48	-1.02	-4.05	-2.18	-9.09	-6.86	-6.30	-7.42	4.38	-1.10	
UWATEC Galileo Sol	-4.82	0.05	1.75	-1.01	-3.29	-2.92	-0.90	-2.37	2.31	-4.11	-6.71	-2.84	5.76	-2.50	-5.04	-0.59	6.75	-1.09	
UWATEC Smart Tec	-4.01	1.72	2.04	-0.09	-3.29	-2.34	-1.38	-2.34	2.31	-4.11	-5.83	-2.54	5.76	-3.95	-5.04	-1.08	6.75	-1.04	
UWATEC Galileo Luna	-4.01	1.72	2.04	-0.09	-2.61	-2.34	0.06	-1.63	2.31	-4.11	-4.94	-2.25	5.76	-2.50	-5.04	-0.59	6.75	-0.74	
SUUNTO Vyper silver	0.85	0.05	-8.34	-2.48	0.11	3.15	4.85	2.70	2.31	-3.95	6.61	-7.68	5.76	1.87	3.83	3.82	6.75	0.13	
UWATEC Aladin One	-4.01	0.05	3.77	-0.07															
SUUNTO Vyper Air	0.44	0.38	-8.05	-2.41	0.11	2.86	4.85	2.61	2.31	2.07	5.72	3.37	2.05	0.41	2.56	1.67	6.75	1.78	
SUUNTO Spyder	4.09	1.72	-7.19	-0.46	2.15	3.44	3.41	3.00	1.04	0.01	-1.38	-0.11	3.90	3.32	0.03	2.42	6.75	1.95	
SUUNTO Cobra 3	1.25	0.38	-7.76	-2.04	0.79	2.86	4.85	2.83	2.31	2.07	6.61	3.66	2.05	0.41	2.56	1.67	6.75	1.96	
SUUNTO Vyper	0.85	0.55	-8.34	-2.32	0.11	3.15	4.85	2.70	2.31	2.07	6.61	3.66	2.05	0.41	2.56	1.67	6.75	2.00	
SUUNTO Cobra 2	0.04	0.05	-8.34	-2.75	0.11	3.44	4.85	2.80	2.31	2.07	6.61	3.66	3.90	0.41	2.56	2.29	6.75	2.06	
SUUNTO Vyper DS black	1.25	-0.29	-8.92	-2.65	2.15	4.02	4.37	3.51	2.31	3.11	5.72	3.71	5.76	1.87	2.56	3.40	6.75	2.45	
SUUNTO Vyper 2	1.66	1.05	-7.19	-1.49	1.47	4.02	6.29	3.93	3.57	2.07	6.61	4.08	3.90	1.87	2.56	2.78	6.75	2.72	
SUUNTO D4	0.44	0.38	-7.76	-2.31	1.13	4.02	5.57	3.57	4.20	2.07	6.61	4.30	5.76	3.32	2.56	3.88	6.75	2.74	
SUUNTO D6	1.66	0.72	-7.48	-1.70	1.47	4.02	5.81	3.96	4.83	2.07	6.61	4.51	5.76	3.32	2.56	3.88	6.75	2.99	
SUUNTO D9	1.25	0.72	-7.48	-1.84	1.47	4.02	5.81	3.77	6.09	2.07	6.61	5.22	5.76	3.32	2.56	3.88	6.75	3.06	
<b>DCIEM table</b>	<b>45.80</b>	<b>40.07</b>	<b>38.36</b>	<b>41.41</b>	<b>2.15</b>	<b>21.35</b>	<b>14.90</b>	<b>12.80</b>	<b>-9.06</b>	<b>11.35</b>	<b>11.94</b>	<b>4.74</b>	<b>-22.07</b>	<b>-3.95</b>	<b>6.36</b>	<b>-6.56</b>	<b>-35.95</b>	<b>-18.06</b>	<b>5.37</b>
SEEMAN XP 5	20.69	15.39	10.68	15.59	7.60	5.75	2.93	5.43	1.04	4.14	-1.38	1.26	5.76	3.32	0.03	3.04	9.12	2.82	5.94
MARES Nemo Sport	-7.66	-3.95	11.84	0.07	-1.93	-1.76	-1.38	-1.69	6.09	4.14	0.39	3.54	13.18	10.60	7.63	10.47	32.84	18.89	7.36
OCEANIC VT 3	16.24	12.39	7.80	12.14	11.69	10.37	8.20	10.09	6.09	8.26	3.95	6.10	2.97	0.41	-3.14	0.08	13.86	6.75	8.88
OCEANIC Datamask Hud	17.86	13.72	8.67	13.41	12.37	-26.03	9.16	-1.50	7.36	10.32	5.72	7.80	11.32	9.15	6.36	8.94	13.86	6.75	7.64
OCEANIC Pro Plus 2	12.59	10.39	6.36	9.78	11.69	10.95	9.16	10.60	7.36	9.29	4.83	7.16	13.18	9.15	5.09	9.14	13.86	6.75	8.88
OCEANIC Atom 2	18.67	14.05	8.95	13.89	12.37	10.37	8.68	10.47	6.09	8.26	3.95	6.10	11.32	9.15	6.36	8.94	13.86	6.75	9.66
<b>RNPL 11 table</b>	<b>45.80</b>	<b>45.07</b>	<b>47.00</b>	<b>45.96</b>	<b>22.58</b>	<b>30.02</b>	<b>29.27</b>	<b>27.29</b>	<b>13.67</b>	<b>23.73</b>	<b>33.26</b>	<b>23.55</b>	<b>11.32</b>	<b>0.41</b>	<b>32.95</b>	<b>14.90</b>	<b>-28.84</b>	<b>6.03</b>	<b>-9.65</b>



**Table 5**

The mean overall times needed to generate decompression times of 10, 20 or 30 min of decompression time at each of five nominal depths (15–50 msw) for 43 models of dive computer and two air decompression tables ( $n = 129$ – $135$  for each cell)

Depth (msw)	Decompression time (min)		
	10	20	30
15	82.3	100.0	115.6
20	49.0	57.7	69.6
30	26.4	32.3	37.5
40	18.0	22.9	26.3
50	14.1	18.1	20.8

*Uemis SDA* which gave longer than mean times at depths of 15 and 20 msw but generated shorter than mean times for the deeper depths (Table 4). Although there was a degree of scatter, there were some general trends within the main manufacturing brands, with *Mares* computers tending to produce the most conservative times, followed in order by *Uwatec* and *Sunto* models (Tables 3 and 4). In both the no-decompression and decompression tests, most of the *Oceanic* computers gave the longest times for many of the exposures (Tables 3 and 4). Overall, but particularly from 30 msw and deeper, the air decompression tables produced the most conservative dive times for no-decompression dives (Table 3); however, in the decompression dives, both of the tables tested (RNPL and DCIEM) were ranked in the lower levels for conservatism (Table 4).

Water temperatures ranged from 12.2–24.5°C ( $n = 1,467$ ) with a total run time of 1,030.6 computer-hours for all the tests. There were 28 battery changes and 19 computer failures during the trials. Some of the failures were minor and related to being unable to download the dive because of low battery power, or only part of or none of the dive had registered on the download. Some of the failures were where the download information simply did not equate to the dive profile; there was one unit that flooded. Results from any unit displaying any recording anomalies (including low battery readings) were rejected from the analyses. It is unclear whether the download errors were representative of real-time problems that could have affected the ability of the diver to continue to receive valid information and, therefore, could have resulted in a dive being aborted. If it is assumed they could be, then this equates to a battery change or failure every 37 or 54 h of diving, respectively.

## Discussion

The results from the present study show numerous scales of variation in how decompression following a single, square-wave exposure is being managed by the dive computers tested. Although 0.4% of all replications showed time differences between sets of greater than 25%, these were in the 40 and 50 msw trials where denominator values are

small and so errors are exaggerated. Irrespective of internal variations there were considerable ranges of times permitted to reach each of the depth/decompression end points. The study employed only single examples of each of the dive computer models tested and lack of replication may explain some of the differences that were observed. Although there was a recorded water temperature range of about 12°C, much of that change was linear and temporary, being caused by the heat of compression. Some dive computers are claimed to modify decompression management with changes in ambient water temperature; however, no detail is provided as to the scale of modification and how that would relate to the range in temperatures recorded in this study. The computers were set to sea water mode in all tests as this function (compared with fresh water) was present in all the units. Although the computers were immersed in fresh water, this would not affect comparative decompression computations as the changes in depth were achieved using pressurised air monitored in msw in all cases.

Some of the variation may be caused by the decompression time retrieved from the computer downloads not necessarily being reduced by all computers at similar rates. Previous studies have shown that the decompression penalties displayed on a dive computer at the start of an ascent may not equate to the actual decompression time that is eventually undertaken.<sup>13</sup> Similarly the rate of reduction in the eventual decompression penalty that occurs in most dive computers as the unit travels to the decompression stop depths is not always uniform between computer models.<sup>13</sup> So, although two computers may both be indicating the same duration of total decompression at the point of initiating an ascent, one may take much longer to reach a point where surfacing is permitted than the other. Dive computers that generate longer surfacing times may be compensating in part for the longer times that some units allow to reach the nominal decompression endpoints.

Additional variation in the results obtained may also have been caused by the relatively low resolution of the time units that were displayed in the downloaded profiles (never less than one minute). It is unknown how the displayed information was being controlled and whether threshold values or conventional rounding up was being employed, or if the methods for rounding up were consistent for all models. Relatively small differences in the recording or display methods could generate significant variance in the results.

It is acknowledged that the compression rates of the two chamber compartments used in the present study were much slower than rates that could be employed in profiles where the diver may be attempting to maximise bottom time. Compression rate produced significant differences in the decompression schedules recorded for the same depth. However, these differences were very small and consistent and did not alter the overall rankings; some of the differences almost certainly resulted from the difficulty in retrieving

high-resolution data from downloaded information alone.

The present study evaluated performance for single, square-wave dive profiles only. The real advantage of using computers to manage decompression is that they can easily control multi-level, multi-day and multi-dive diving.<sup>6,14</sup> Some of the variation must be attributed to the decompression theories being employed. Examination of Tables 3 and 4 does show approximate groupings for the main manufacturers. This is not surprising as the different manufacturers tend to employ the same form of decompression algorithm over their whole family of computers.<sup>2</sup> However, there is no consistent or predominant decompression model being used and several manufacturers are modifying the algorithms themselves but in the absence of published criteria supporting those modifications.

For example, all the *Oceanic* computers examined in the present study employ modified Haldanean algorithms using the Diving Science and Technology database; the *Suunto* computers use the Suunto reduced gradient bubble model (RGBM); the *Uwatec* computers use versions of the ZH-L8 ADT, which is *Uwatec*'s adaptive 8-tissue algorithm; and *Mares* use their Mares-Wienke RGBM which is not a true RGBM algorithm but a Haldanian model with some additional safety factors.<sup>2,15</sup> Although it could be hypothesised that some algorithms are modifying the test dive decompression management because it is being treated as the initial dive in an anticipated multi-dive series, the differences between the computers are not always consistent across the depth range investigated and so significant theoretical dissimilarities must exist. It is most likely that the computers treat a 'first' dive in isolation and make any subsequent adjustments if the dive series evolves. In that case, differences in how the computers are working are known. For example, the standard Bühlmann model does not penalise consecutive dives whereas the RGBM models from *Suunto* and *Mares* employ a safety factor for repetitive diving that does give a penalty.<sup>15</sup>

The rate of battery failures in the present study was similar to values published previously.<sup>9</sup> The amount and type of warning given to the diver of an impending battery failure varied markedly between models. This, and the relatively high rate of failures recorded that could impact the ability to control decompression, would suggest that carrying two computers should be standard for any occupational diver who is relying on this method for dive management.

The results from the present study are probably only pertinent to the working diver because of the single-dive, square-wave profile employed. In many diving industry sectors, there continues to be a degree of scepticism about using dive computers for managing decompression. Much of that will come from the perceived loss of control over the diver from the surface supervising team. Where the safe control of decompression management can be devolved to the diver,

then the present study would suggest that many models of dive computer deliver profiles that are as conservative as standard air decompression tables for non-decompression diving, but considerably more conservative for those dives that involve staged decompression. There is no evidence to imply that the longer exposures being indicated by some of the computers are not adequate although decompression sickness risks and probabilities will probably increase with prolonged bottom time.

In a computer-driven era, it remains disappointing that dive management decisions, needed to balance the operational benefits of longer dive times against the additional risk of decompression sickness, continue to be based largely on subjective assessment. This will remain an issue until there is an accepted 'gold standard' for decompression modelling. As long as no standardised decompression model exists it will remain difficult for there to be any consistent approach to the manufacture of decompression computers.<sup>4</sup>

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Centre for Diving and Hyperbaric Medicine.

#### Conflict of interest

One author (AS) develops diving computers but only for professional use and is not in competition with the brands employed in the present study.

*Martin DJ Sayer<sup>1</sup>, Elaine Azzopardi<sup>1</sup> and Arne Sieber<sup>2</sup>*

<sup>1</sup> *UK National Facility for Scientific Diving hosted at the Scottish Association for Marine Science Laboratories near Oban, Argyll, Scotland*

<sup>2</sup> *Chalmers University of Technology, Gothenburg, Sweden, and SeaBear Diving Technology, Austria*

#### Address for correspondence:

*MDJ Sayer, PhD*

*UK National Facility for Scientific Diving*

*Scottish Association for Marine Science*

*Dunstaffnage Marine Laboratories, Dunbeg, Oban*

*Argyll PA37 1QA, Scotland*

**Phone:** +44-(0)1631-559236

**E-mail:** <mdjs@sams.ac.uk>

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## 12-lead Holter monitoring in diving and water sports: a preliminary investigation

Gerardo Bosco, Elena De Marzi, Pierantonio Michieli, Hesham R Omar, Enrico M Camporesi, Johnny Padulo, Antonio Paoli, Devanand Mangar and Maurizio Schiavon

### Abstract

(Bosco G, De Marzi E, Michieli P, Omar HR, Camporesi EM, Padulo J, Paoli A, Mangar D, Schiavon M. 12-lead Holter monitoring in diving and water sports: a preliminary investigation. *Diving and Hyperbaric Medicine*. 2014 December;44(4):202-207.)

**Objective:** To demonstrate the utility of 12-lead Holter monitoring underwater.

**Methods:** A Holter monitor, recording a 12-lead electrocardiogram (ECG) underwater, was applied to 16 pre-trained volunteer scuba divers (13 males and three females). Dive computers were synchronized with the Holter recorder to correlate the ECG tracings with diving events. Our main objective was to demonstrate the utility of recording over a period of time a good quality 12-lead ECG underwater. The ECGs were analyzed for heart rate (HR), arrhythmias, conduction abnormalities and ischaemic events in relation to various stages of diving as follows: baseline, pre diving, diving, and post diving.

**Results:** The ECG tracings were of good quality with minimal artefacts. Analysis of variance (ANOVA) demonstrated a significant difference in HR during the various diving stages ( $P < 0.0001$ ). Other recorded ECG abnormalities included supraventricular ectopic beats (four cases), ventricular ectopic beats (eight cases) and ventricular couplets (two cases). Conduction abnormalities included rate-dependent right and left bundle branch block; however, these findings were previously known in these divers. No evidence of ischaemia was seen.

**Conclusion:** Continuous 12-lead Holter monitoring underwater can produce good quality tracings. Further studies are necessary to assess its usefulness in divers at risk for or with known coronary artery disease, and its comparison with other forms of cardiac stress tests.

### Key words

Scuba diving, cardiovascular, electrocardiography, physiology, pathology, diving research, patient monitoring

### Introduction

The analysis of sudden death rates in athletes during the last 30 years in Veneto, Italy showed a significant decrease believed to be the result of implementing 12-lead electrocardiography (ECG) screening to detect silent heart disease in young athletes.<sup>1-3</sup> Underwater activity is a known stressor to the cardiovascular system that can lead to myocardial ischaemia in predisposed subjects, and cardiovascular disease is the third-leading cause of death during diving.<sup>4</sup> This highlights the potential value of implementing a valid screening tool to identify divers at risk of cardiovascular death.<sup>5,6</sup> Immersion in cold water, unlike warm-water immersion which is associated with peripheral vasodilatation, increased venous return and cardiac output, causes reflex peripheral vasoconstriction and bradycardia, with a resultant reduction in stroke volume and cardiac output.<sup>7-10</sup> Together with this left ventricular diastolic dysfunction, the postural effects of weightlessness and hydrostatic pressure force blood from the peripheries to the pulmonary circulation.<sup>3,7</sup> These effects occur in both breath-hold and scuba diving.<sup>9,10</sup> Understanding the interaction of these haemodynamic effects is crucial, particularly in divers who may suffer from ischaemic or other heart disease.<sup>8,11</sup>

The 12-lead ECG is valuable in detecting channelopathies, hypertrophic cardiomyopathy and the Wolff-Parkinson-White syndrome, which altogether contribute to a significant proportion of sudden deaths in athletes. Exercise testing is

a tool to investigate divers with risk factors for coronary artery disease to rule out silent myocardial ischaemia and to assess their functional capacity.<sup>12</sup> Little attention has been paid to ECG monitoring in the aquatic environment. Since the cardiovascular stressors underwater are different from stressors during a standard exercise ECG, we thought that Holter monitoring during diving and water sports could be of value especially in divers older than 35 years who are more vulnerable to coronary atherosclerosis.<sup>13</sup> This presents several technical challenges such as ensuring that the instruments are water- and pressure proof and the need for electrode isolation to prevent the fall in electrical impedance with immersion in water.<sup>14</sup>

We hypothesized that recording a continuous 12-lead ECG underwater would detect the development of ischaemic changes that would help stratify patients at high risk for breath-hold and scuba diving. Our preliminary observations demonstrating the physiological and clinical utility of Holter monitoring underwater are reported.

### Material and Methods

#### SUBJECTS

Sixteen Caucasian divers, 13 males and three females (age  $35.1 \pm 9.2$  years, weight  $69.6 \pm 3$  kg and height  $172.6 \pm 3.4$  cm) volunteered to participate in this study. Written, informed consent was obtained and the study was approved

**Figure 1**

The position of the 10 electrodes in a diver using a one-piece wetsuit; a small hole in the suit at the level of the left flank allows the passage of the Holter cable



by the University of Padova Human Research Ethics Committee (approval number 1/2014). The study followed the principles of the Declaration of Helsinki (2008 revision). The divers used their personal equipment: seven used a wetsuit, four a semi-drysuit and five wore a drysuit.

#### ECG MONITORING

Continuous 12-lead ECG recordings on a Holter monitor were undertaken as follows. The diver's chest was shaved, if necessary, and degreased with denatured alcohol to ensure good adhesion of the electrode patches to the skin. At the points of attachment of the self-adhesive electrodes (Kendall Arbo H34SG Tyco Healthcare and 3M Red Dot 2255), a small amount of conduction gel (Eco supergel Ceracarta, Forli, Italy) was applied. The 10 electrodes were placed following the standard procedure used for recording 12-lead Holter as follows: slightly below the right and left clavicle, manubrium sterni, fifth intercostal space at the right and left sternal border, four electrodes along the left inframammary line positioned in the fifth intercostal space from parasternally to the mid-axillary line and a tenth electrode at the lower edge of the rib cage in the mid-axillary line (Figure 1). In divers using a one-piece wetsuit, a small hole at the level of the left flank was made to allow passage of the cable and the positioning of the Holter monitor (Figure 2).

After connecting the cables, the electrodes were covered with two layers of two different transparent film adhesive tapes. The first layer (Visulin, Hartmann) was used to protect the thin cables and was easily peeled off to allow the removal

**Figure 2**

The Holter recorder in its pressure proof housing strapped to the diver's waist



of the instrumentation without damaging it and to limit the discomfort during detachment from the skin. The top layer consisted of 3M Steri-Drapes; this second layer was omitted if a drysuit was used.

A digital Holter device (H12+, Mortara Instrument Europe Ltd, Milwaukee, Wisconsin) was used, weighing 125 g and capable of recording 12 channels in real time to be stored on a compact flash memory card. Its reliability in water compared to the surface was confirmed with a Bland-Altman test (unpublished data) with error < 2% of the recorded signals. The Holter recorder was placed in a pressure-proof anticorrosive aluminum housing, with a Plexiglas cover (Metralabs. r.l., Padova, Italy; Figure 2), pressure tested to at least 608 kPa. There were no flooding problems and the system was well tolerated by the divers. For preliminary tests in salt water and in a swimming pool, only a single layer of Suprasorb (Lohmann and Rauscher, an analogue of Visulin) was used for better comfort. At the start of the experiment, more than one dive was performed for technical purposes until optimal quality ECGs were obtained.

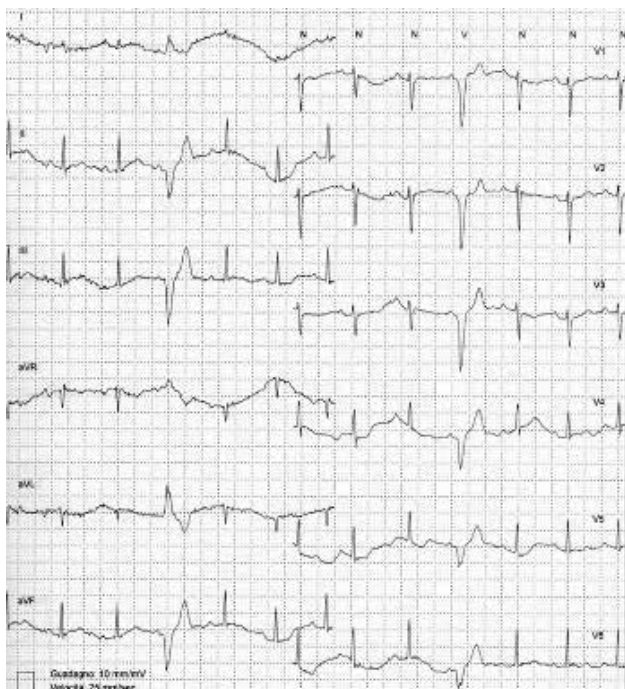
#### MEASUREMENTS

The ECGs were recorded during scuba air dives in the sea. Dive computers were synchronized with the Holter monitor to correlate the ECG tracings with diving events. The tracings were analyzed by a cardiologist using the software H-Scribe Enterprise (Mortara Rangoni Europe, Milwaukee, Wisconsin). Heart rate (HR, beats·min<sup>-1</sup>) was recorded continuously as follows: baseline before kitting up, pre dive, diving and post dive. The average HR for each individual subject during each of these four stages was then expressed as a percentage of the maximum theoretical HR for that subject, according to the formula:<sup>18</sup>

$$HR_{\max} = 208 - (0.7 \times \text{age}) \quad (1)$$

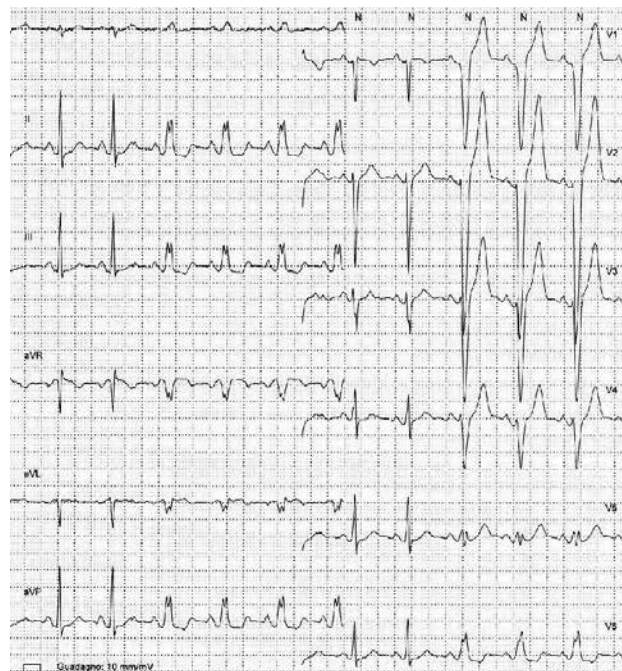
**Figure 3**

Ventricular extrasystole in Subject AA at 22.5 msw depth



**Figure 4**

Left bundle branch block in Subject LS at 26 msw



As well as HR, abnormalities of conduction, such as supraventricular ectopics (SVEs), premature ventricular contractions (PVCs) and bundle branch block, as well as evidence of ischaemia, were looked for in the recordings. SVEs and PVCs were classified according to Lown's criteria.<sup>15</sup> Diving data included depth and duration of the dive, water temperature, air consumption and diving conditions, such as swimming against a current or low visibility.

**STATISTICAL ANALYSIS**

The results are expressed as mean ± standard deviation (SD). In order to determine any significant difference in HR during the various stages of diving, a one-way ANOVA was applied. Assumption of normality was verified using the Shapiro-Wilk W-test. When a significant F-value was found, the least significant difference (Bonferroni) was chosen as the post-hoc procedure. Statistical analyses were performed using the software IBM SPSS Statistic, version 15.0 (IBM Corporation, Somers, New York). The level set for significance was  $P \leq 0.05$ .

**Results**

**QUALITY OF ECG RECORDINGS**

All participants completed the study without any complications. The ECG tracings during diving were comparable to standard ECG tracings on dry land. There were no differences between salt and fresh water (based on the pre-trial recordings) or related to the type of diving suit

worn. There were some artefacts in the recordings depending on the depth of the dive and movements of the upper limbs and trunk; however, they were insignificant and similar in extent to motion artifacts produced from athletes running on a treadmill. Generally, there was little or no loss of data.

**HEART RATE CHANGES**

The one-way ANOVA showed significant differences in HR during the various diving stages: Baseline–Pre-dive–Dive–Post-dive ( $F = 37.293, P < 0.0001$ ; Table 1). The average baseline heart rate was  $87 \pm 2$  beats·min<sup>-1</sup> which increased significantly during the pre-diving stage to  $135 \pm 20$  beats·min<sup>-1</sup>. This was detected from a few seconds to 26 minutes ( $7.4 \pm 7.9$  min) before the start of descent and represents a mean HR increase of  $48 \pm 15$  beats·min<sup>-1</sup>. In

**Table 1**

Heart rate (HR) recorded during the diving stages (Baseline – Pre – During – Post) expressed as the mean of the percentages of the maximum theoretical heart rate ( $HR_{max} = 208 - (0.7 \times \text{age})$ ) for each subject; i.e., the average baseline HR was  $47.7 \pm 9.93\%$  of  $HR_{max}$ . § This HR value represents the % difference in the mean HR between the four stages; \*  $P < 0.05$ , †  $P < 0.001$ , (i.e., compared to baseline, the average HR pre-dive (denoted in the table as a/b increased significantly by 48%)

Condition	HR % (compared with HR max)‡	Δ% §	Δ% §
Baseline (a)	$48 \pm 9.93$	a/b – 48†	a/c – 18 (ns)
Pre-dive (b)	$71 \pm 9.87$	b/c – 21†	a/d – 24*
During (c)	$56 \pm 8.20$	b/d – 49†	c/d – 35†
Post-dive (d)	$36 \pm 10.16$		

**Table 2**

Demographic and dive characteristics (dive duration, air consumption, maximum depth and minimum temperature achieved) of the 16 subjects; the recorded HRs during the 4 diving stages and the occurrence of ECG abnormalities; means and standard deviation (SD) shown in the bottom row; BMI – body mass index; msw – metres’ sea water; PAC – premature atrial contraction; PVC – premature ventricular contraction; BBB – bundle branch block; LBBB – left bundle branch block; RBBB – right bundle branch block

Subject	Sex	Age (yrs)	BMI (kg·m <sup>-2</sup> )	Certification	Suit	Temperature (°C)	Depth (msw)	Duration (min)	Baseline	Heart rate				BBB	
										Pre dive (beats·min <sup>-1</sup> )	Dive	Post dive	PAC		PVC (couplets)
DE	F	32	18.2	Advanced	wet	16	30.5	43	90	132	105	69	0	1	–
BC	M	33	25.4	Rescue	semi-dry	21	24.3	53	80	135	90	59	1	0	–
DF	M	39	22.9	Open	dry	12	22.9	39	120	170	120	100	0	14 (2)	–
EF	M	35	22	Open	dry	17	22.1	34	90	130	120	80	0	0	–
ED	M	36	20.3	Dive master	dry	17	19.2	38	70	129	90	49	0	0	–
CR	M	33	24.9	Open	dry	18	21.8	46	85	135	115	74	0	0	–
PE	M	39	31.1	Advanced	dry	16	28.4	36	100	154	115	93	0	0	–
AD	M	42	24.9	Dive master	wet	18	22.9	38	70	149	95	49	0	2	–
GD	F	36	19	Advanced	semi-dry	20.5	7.2	55	80	115	85	52	0	0	–
LS	M	37	26	Tech diver	wet	10.4	33.1	45	85	150	95	68	1	2	LBBB
AA	M	45	24.2	Instructor	wet	18	37.5	50	95	120	100	50	0	10	–
BA	M	27	22	Instructor	wet	18	32.3	46	85	95	95	75	0	0	RBBB
CL	M	19	22.5	Open	semi-dry	18	21.2	45	85	130	120	70	0	1	–
CG	M	57	24.2	Rescue	wet	17.5	19	36	115	167	120	80	12	9 (1)	–
CA	M	32	21.5	Advanced	semi-dry	18	26.6	45	87	141	85	59	1	4	–
RI	F	19	19.8	Advanced	wet	18	33.4	42	55	109	90	31	0	0	–
Mean (SD)		35.1 (9.2)	23.3 (3.1)			17.1 (2.7)	25.2 (7.4)	43.2 (6.2)	87.0 (16.0)	135.1 (20.1)	102.5 (13.7)	66.1 (17.9)			

four cases, the increase in the HR was recorded immediately prior to the descent and in one case while swimming at the surface to reach the point for the descent. The two professional divers in the group showed the smallest increases in heart rate (10 and 25 beats·min<sup>-1</sup> respectively).

Slowing of the HR developed during the descent and was maintained between 85 and 120 beats·min<sup>-1</sup> (102 ± 13.7 beats·min<sup>-1</sup>) throughout the dive, with no correlation to the depth of immersion. A decrease in HR to 66 ± 17.9 beats·min<sup>-1</sup> was observed post dive which represents a significant decrease compared to the baseline HR (19 ± 7.8 beats·min<sup>-1</sup> lower) and the pre-dive HR (68 ± 18.1 beats·min<sup>-1</sup> lower). The lowest HR was observed between 7 min before surfacing until 20 min after surfacing.

#### DYSRHYTHMIAS AND CONDUCTION ABNORMALITIES

Supraventricular ectopic beats were identified in four subjects, ventricular ectopic beats in eight subjects and ventricular couplets in two subjects. Two divers had one PVC during the ascent and after emergence (Figure 3), two divers had two PVCs both during the descent, one diver had four PVCs, one nine PVCs, one had 10 PVCs and one diver who was already known to suffer from PVCs had 14 (all Lown class 1). Two divers had two consecutive PVCs (couplets, Lown class 4a which carries a higher risk of degenerating into ventricular arrhythmia). The ventricular couplets were recorded 10 min before descent in one case and 2 min after surfacing in another case. Conduction abnormalities, including right bundle branch block and rate-dependent left bundle branch block (LBBB, Figure 4), were recorded; however, this diagnosis was not new for either of these divers.

None of the divers experienced chest pain during diving. There were no observed ST-segment shifts to suggest ischaemia in any of the recorded ECGs. Table 2 represents a compilation of the demographics, dive characteristics, the recorded HR during the four diving stages and the occurrence of arrhythmias in the 16 study subjects.

#### Discussion

The first underwater Holter studies, carried out in 1970 on breath-hold divers using magnetic tape recordings, correlated the breath-hold immersion-induced bradycardia with the duration of the apnea and the speed and depth of descent. Increased parasympathetic activity was demonstrated in healthy subjects during scuba diving and heart rate variability (HRV) assessed using 2-lead Holter monitoring.<sup>15,17-19</sup> Unlike patients with heart disease where a decrease in the HRV is associated with a higher risk of cardiac death, the significance of a reduced HRV in a healthy person is unknown. Despite the technical limitations, the occurrence of arrhythmias in elite breath-hold divers during deep diving in the sea has been recorded using 3-lead ECG monitoring.<sup>8</sup> Others have recorded ECG and depth simultaneously using

an ECG device with an integrated pressure sensor.<sup>18</sup> There are also reports of simultaneous recording of a 2-lead ECG, oxygen saturation, depth and temperature underwater up to a depth of 10.5 metres.<sup>20</sup>

The current study demonstrated the feasibility of recording 12-lead ECG underwater, with good quality tracings similar to those during exercise treadmill testing. We found a maximum increase in HR in the pre-diving stage, slowing at the end of the dive. This increase in the pre-dive HR was unrelated to physical effort and was mostly detected at rest during the waiting period prior to the descent. The two professional divers showed only minor HR increases before descent, confirming that this HR rise is likely linked to an emotional phenomenon causing sympathetic stimulation.<sup>21</sup> This emotional origin of tachycardia prior to performance is well known and correlates with the type of sport, being most significant with high-risk activities, such as motor racing, downhill skiing or skydiving.<sup>22</sup>

During the dive, HR was constantly maintained higher than baseline with minimal variability. An important finding was the bradycardia recorded at the end of the dive, a phenomenon reported previously after recreational scuba diving.<sup>6</sup> Hypothermia-induced bradycardia during cold-water immersion is a possible explanation for this phenomenon; bradycardia is a recognized haemodynamic finding in hypothermia.<sup>23</sup> Also, the shift of blood volume into the thoracic vasculature with immersion stimulates cardiopulmonary baroreflexes with a resultant parasympathetic effect and bradycardia.<sup>8</sup>

The occurrence of PVCs did not correlate with any particular stage of the dives, although they were most commonly seen immediately pre dive and post dive. The development of rate-dependent LBBB provoked a complete re-evaluation of this diver, confirming his unsuitability for diving. In the absence of underlying heart disease, the presence of PVCs usually has no impact on limiting activity and their numbers usually decrease during exercise owing to overdrive suppression of ectopic pacemakers by the fast sinus rhythm. An increase in PVC frequency during exercise should therefore prompt evaluation of the cardiac status of the diver.<sup>24</sup>

The main limitation of the study is that these dives were not standardized for immersion times and depth of exposure, as our main purpose was to demonstrate the feasibility of recording 12-lead ECG underwater. The need for meticulous placement of the electrodes and proper insulation for ideal signal transmission cannot be over-emphasized. We have demonstrated the feasibility of dynamic 12-lead ECG recording underwater producing good quality tracings, with no reduction in voltage, and minimal motion artifacts. Since Holter monitoring is the gold standard for detailed diagnosis of acute ischaemia and arrhythmias, it will be useful particularly for assessing the older diver, those with risk factors for coronary artery disease and those starting a professional water sports career. Its advantage over standard



exercise testing (e.g., Bruce protocol) is that it directly reflects the interaction between the underwater environment with its unique stressors and the cardiovascular system. Based on this experience, further studies under more strictly controlled conditions should be undertaken to assess the value of underwater Holter monitoring compared to other forms of cardiac stress tests.

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## Conflicts of interest: nil

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Gerardo Bosco<sup>1</sup>, Elena De Marzi<sup>2</sup>, Pierantonio Michieli<sup>3</sup>, Hesham R Omar<sup>4</sup>, Enrico M Camporesi<sup>5</sup>, Johnny Padulo<sup>1</sup>, Antonio Paoli<sup>1</sup>, Devanand Mangar<sup>6</sup>, Maurizio Schiavon<sup>2,3</sup>

<sup>1</sup> Physiological Laboratory, Department of Biomedical Sciences, University of Padova, Italy

<sup>2</sup> Scuola Superiore Sant'Anna, Pisa, Italy

<sup>3</sup> Sports Medicine and Physical Activities Unit - "aiColli" Social Health Department, Padova, Italy

<sup>4</sup> Internal Medicine Department, Mercy Hospital and Medical Center, Chicago, Illinois, USA

<sup>5</sup> Departments of Surgery/Anesthesiology and Molecular Pharmacology and Physiology, University of South Florida, Tampa, Florida, USA

<sup>6</sup> Florida Gulf to Bay Anesthesia, Tampa General Hospital, Tampa, Florida, USA

## Address for correspondence:

Professor Enrico M Camporesi, MD

University of South Florida College of Medicine

Tampa General Hospital

1 Tampa General Circle Suite A327

Tampa, Florida, 33606, USA

E-mail: <ecampore@health.usf.edu>

## Review article

# The assessment and management of inner ear barotrauma in divers and recommendations for returning to diving

Elizabeth J Elliott and David R Smart

### Abstract

(Elliott EJ, Smart DR. The assessment and management of inner ear barotrauma in divers and recommendations for returning to diving. *Diving and Hyperbaric Medicine*. 2014 December;44(4):208-222.)

Inner ear barotrauma (IEBt) constitutes a spectrum of pressure-related pathology in the inner ear, with antecedent middle ear barotrauma (MEBt) common. IEBt includes perilymph fistula, intralabyrinthine membrane tear, inner ear haemorrhage and other rarer pathologies. Following a literature search, the pathophysiology, diagnosis, and treatment of IEBt in divers and best-practice recommendations for returning to diving were reviewed. Sixty-nine papers/texts were identified and 54 accessed. Twenty-five case series (majority surgical) provided guidance on diagnostic pathways; nine solely reported divers. IEBt in divers may be difficult to distinguish from inner ear decompression sickness (IEDCS), and requires dive-risk stratification and careful interrogation regarding diving-related ear events, clinical assessment, pure tone audiometry, a fistula test and electronystagmography (ENG). Once diagnosed, conservative management is the recommended first line therapy for IEBt. Recompression does not appear to cause harm if the diagnosis (IEBt vs IEDCS) is doubtful (limited case data). Exploratory surgery is indicated for severe or persisting vestibular symptoms or hearing loss, deterioration of symptoms, or lack of improvement over 10 days indicating significant pathology. Steroids are used, but without high-level evidence. It may be possible for divers to return to subaquatic activity after stakeholder risk acceptance and informed consent, provided: (1) sensorineural hearing loss is stable and not severe; (2) there is no vestibular involvement (via ENG); (3) high-resolution computed tomography has excluded anatomical predilection to IEBt and (4) education on equalising techniques is provided. There is a need for a prospective data registry and controlled trials to better evaluate diagnostic and treatment algorithms.

### Key words

Inner ear, barotrauma, diving, pathophysiology, treatment, ENT, review article

### Introduction

Inner ear barotrauma (IEBt; see Table 1 for a range of acronyms related to this topic and used in this article) encompasses a spectrum of pathology in the inner ear resulting from pressure injury. This includes perilymph fistula (PLF), intra-labyrinthine membrane tear, inner ear haemorrhage, and other rarer pathologies (Figure 1). PLF is a subset of IEBt where an inappropriate communication occurs between the perilymph fluid in the inner ear and the middle ear via the labyrinthine structures of the round and oval windows.<sup>1-4</sup> A communication may also occur within the semicircular canals, vestibule, or cochlea via the intra-labyrinthine structures of the Reissner's, basilar and tectorial membranes, resulting in mixing of the endolymph and perilymph.<sup>3,5-7</sup>

### Anatomy

The oval window receives sound vibrations directly from the stapes footplate, converting them into waves which travel through perilymph to the organ of Corti for sound detection. The round window is also located in the vestibule of the membranous labyrinth, inferior to the oval window. Its function is to compensate for the changes in pressure in the unyielding fluid by stretching.<sup>8</sup> If a deficit involves either labyrinthine window, perilymph fluid leak occurs from the

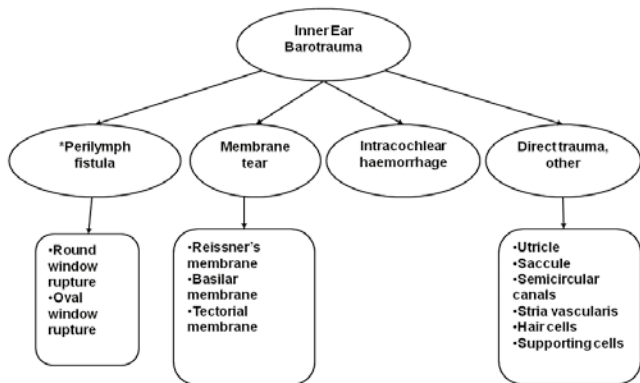
semicircular canals and/or cochlea into the middle ear (see <<http://www.dizziness-and-balance.com/disorders/unilat/fistula.html>>).<sup>2,7-10</sup>

Reissner's and the basilar membranes are located in the perilymphatic space between the internal structures of the cochlea, separating the three scala layers. Reissner's membrane divides the scala vestibuli and scala media; the basilar membrane separates the scala media and scala tympani and the tectorial membrane sits in the scala media.<sup>11,12</sup> The fluid in the scala vestibule and tympani is perilymph, with endolymph contained in the scala media above the organ of Corti (Figure 2).<sup>11,12</sup> Each has a significantly different ionic composition. All membranes are responsible for propagation of sound waves via the incompressible inner ear fluid.

### Diving-related barotrauma, hearing loss and IEBt

Aural barotrauma has been identified as the most common cause of long-term morbidity in divers, experienced by 52% in a sample of 709 experienced recreational divers.<sup>13</sup> It is estimated that 0.5–1.1% of divers will suffer IEBt in their lifetime.<sup>13,14</sup> In the absence of ear injury or noise exposure, diving per se may not cause hearing loss. No significant difference was found in pure tone audiometry (PTA) of 60 sport divers compared with controls; neither

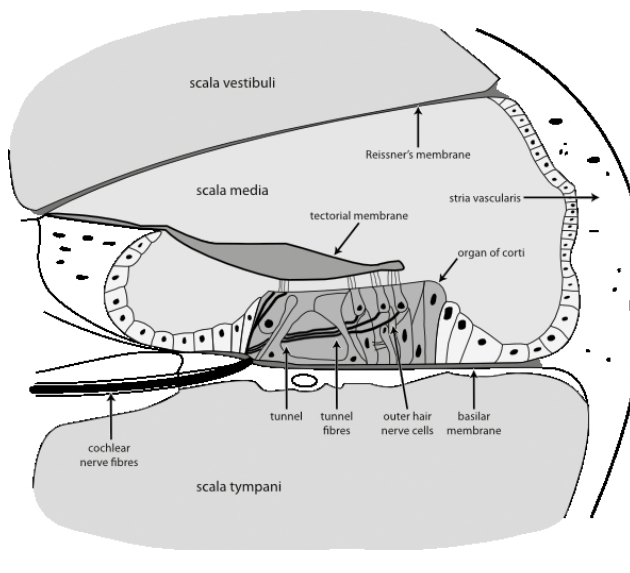
**Figure 1**  
Types of inner ear barotrauma



\* Note: Perilymph fistula can result from multiple other causes in non divers

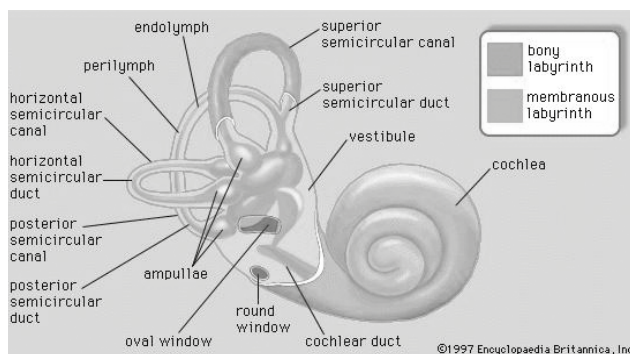
**Figure 2**

Cross section of the cochlea, illustrating the Organ of Corti  
<<http://en.wikipedia.org/wiki/File:Cochlea-crosssection.svg>><sup>12</sup>  
(reproduced with permission of *Wikipedia*)



**Figure 3**

Dissection of the inner ear <<http://www.britannica.com/EBchecked/topic/175622/human-ear/65040/Cochlea>><sup>32</sup>  
(reproduced with permission of *Britannica*)



**Table 1**

Acronyms for otorhinolaryngological and other terms relevant to inner ear barotrauma used in this article

Acronym	Medical term
ABR	Auditory brainstem response
BPPV	Benign paroxysmal positional vertigo
CCG	Craniocorpography test
CHL	Conductive hearing loss
ENG	Electronystagmography
ENT	Ear, nose and throat
HRCT	High-resolution computed tomography
IEBt	Inner ear barotrauma
IEDCS	Inner ear decompression sickness
MEBt	Middle ear barotrauma
MRI	Magnetic resonance imaging
OAE	Otoacoustic emission
OWR	Oval window rupture
PLF	Perilymph fistula
PTA	Pure tone audiometry
RWR	Round window rupture
SCD	Superior canal dehiscence
SHA	Smooth harmonic acceleration
SNHL	Sensorineural hearing loss
TM	Tympanic membrane
URTI	Upper respiratory tract infection
VEMP	Vestibular evoked myogenic potential

group had a past history of significant noise exposure or IEBt.<sup>15</sup> Another study comparing occupational divers with offshore workers also did not detect a higher incidence of hearing loss in divers.<sup>16</sup> In 64 entry-level occupational divers studied over six years, the only detected change in PTA was secondary to noise exposure. None suffered IEBt (or inner ear decompression sickness, IEDCS), although middle ear barotrauma (MEBt) was common.<sup>17,18</sup> One-hundred-and-twenty Navy divers had greater hearing loss than 116 non-diver controls.<sup>19</sup> Although cases were not split by cause of hearing loss (IEBt, IEDCS or industrial hearing loss), this study suggested that continuing to dive with known hearing problems may exacerbate hearing loss over time.<sup>19</sup> Any study of aural barotrauma or hearing loss in divers presents challenges – the spectrum and severity of injuries is broad. In addition, detection of injury may be retrospective via hearing and balance deficits, with no clear traumatic event recognised by the diver.

Hence, the true incidence of IEBt is difficult to calculate owing to minor symptoms not precipitating medical review, spontaneous healing and diagnostic uncertainty. Following either conservative or surgical treatment options, the diving specialist faces the challenge of assessing fitness to return to diving. There appears no current consensus to guide occupational divers returning to employment following IEBt, and this review was unable to identify guidance from learned societies, such as the South Pacific Underwater Medicine Society (SPUMS), the Undersea Hyperbaric Medical Society (UHMS) or the European Underwater and Baromedical Society (EUBS).<sup>7,12,20–26</sup>

Our aim was to review the literature on the pathophysiology, diagnosis and treatment of IEBt in divers and to develop best-practice recommendations for diagnosis, treatment and returning to diving.

### Literature search

A literature search from January 1972 to December 2012 was conducted using search terms 'inner ear barotrauma', 'perilymph fistula', 'round window rupture', 'oval window rupture', 'diving', 'SCUBA', 'diagnosis' and 'treatment'. The search was initially restricted to human studies only. Search engines employed included Ovid, Medline (PubMed), CINAHL, EMBASE and Google Scholar. In addition, a hand search was conducted of texts in diving medicine that covered IEBt, as well as proceedings and workshops from SPUMS, UHMS, EUBS and the International Congress on Hyperbaric Medicine. Finally a search was undertaken of references identified in papers located by the initial search providing access to other relevant articles.

### SEARCH RESULTS

Sixty nine papers/texts were identified, with 54 papers/chapters accessed. One paper was rejected as it was unable to be translated.<sup>27</sup> Six other papers were included for essential pathophysiological or anatomical information and the Australian/New Zealand standards.<sup>28-31</sup> Six webpages were accessed for supportive information and images.<sup>2,8,11,12,32,33</sup> Four key animal studies (dogs, cats, rabbits and guinea pigs) were included describing the historical development of pathophysiological understanding of IEBt.<sup>34-37</sup>

### Pathophysiology of IEBt in divers

Understanding pressure physiology is essential for an understanding of IEBt. Pressure increases by one atmosphere (101.3 kPa) for every 10 metres' sea water depth (msw). According to Boyles Law, during descent to 10 msw, an air space halves in volume, unless equalised by additional gas. The greatest rate of change in volume occurs in shallow water, hence divers may experience significant barotrauma even at shallow depths.<sup>14,23,24</sup>

From historical data on intraoperative assessments or forensic findings, three injury patterns were identified from IEBt in isolation or combination in scuba divers:

- fistula of the round or oval window;
- intralabyrinthine membrane tear;
- inner ear haemorrhage.<sup>23</sup>

### PERILYMPH FISTULAS IN DIVERS

A key factor in diving-related IEBt is the direct link between the intracranial cerebrospinal fluid (CSF) and the perilymph. IEBt results from unbalanced pressure changes between the structures of the inner ear and middle ear and/or CSF.

The relationship between changes in pressure from the intracranial space to the perilymph was first identified in rabbits in 1879, and further clarified in the mid-twentieth century.<sup>35-38</sup> An association was demonstrated between thoracic and intra-abdominal pressures, occlusion of neck blood vessels and CSF pressure.<sup>38</sup> Citing many early animal studies, it was concluded that changes in perilymph pressures correlated with intracranial pressure (ICP), and occlusion in the cochlear aqueduct inhibited this reflex.<sup>39</sup> However, it was not until the 1960s that the link between raised CSF pressure, perilymph pressure and PLF was observed in humans.<sup>39</sup> This coincided with the advent of stapes surgery. IEBt was first confirmed in divers in 1972 using pre- and post-incident audiograms.<sup>40</sup>

IEBt in diving may result from 'explosive' and/or 'implosive' forces on the vestibulocochlear apparatus due to pressure differences caused by a blocked Eustachian tube.<sup>3,6,7,26,41-44</sup> Explosive barotrauma is transmitted through the CSF to the perilymph of the cochlear aqueduct, vestibular aqueduct, and scala tympani.<sup>6,43,45,46</sup> The final endpoint of this pressure wave is the round or oval window. Implosive forces act from external sources upon the inner ear. Middle ear pressure changes may be transmitted to the labyrinthine windows, causing rupture and vestibulocochlear injury. Round window rupture (RWR) appears to be associated with barotrauma, whereas oval window rupture (OWR) is more commonly due to external forces to the head or auditory apparatus.<sup>43</sup>

Diving-related IEBt is thought to be due to pressure gradients on the tympanic membrane (TM) and middle ear during descent. From case histories, the main antecedent cause appears to be inability to pressurise the middle ear.<sup>10,21</sup> When the pressure differential between the middle ear and nasopharynx is > 90 mmHg (12 kPa), the Eustachian tube closes and 'locks', resulting in an inability to equalise the middle ear.<sup>10,46</sup> The TM bulges inwards, pushing the stapes into the oval window, displacing perilymph towards the round window, pushing it outwards into the middle ear cavity.<sup>3,7,43</sup> As perilymph and endolymph are incompressible fluids, pressure changes are transmitted to the weaker labyrinthine windows (Figure 3).<sup>7,32,43</sup> If a sudden pressure wave is transmitted to the perilymph, a RWR may result with leakage of fluid into the middle ear or an influx of air into the perilymph.<sup>3,20,47</sup> The above mechanism is a combination of implosive force (on the stapes and oval window), and explosive force due to the Valsalva manoeuvre. The process is augmented by a pre-existing negative pressure in the middle ear. This causes a sensorineural hearing loss (SNHL) and vestibular symptoms which may be progressive or fluctuating owing to leakage of perilymph.<sup>3</sup>

Excess pressure in the perilymph resulting from other processes, such as coughing, sneezing, straining or lifting, may also cause a RWR, OWR or tear of the basilar or Reissner's membranes.<sup>3,43,48,49</sup> Stresses are further amplified by external negative pressure (e.g., when removing a diving

hood).<sup>1,6,44</sup> Non-diving case series frequently include such mechanisms of injury. Most literature focuses on RWR and OWR which are subsets of IEBt. Both can occur as single pathologies, combined or occasionally bilateral. In the setting of diving barotrauma, RWR almost exclusively occurs and OWR is very rare.<sup>21,25,48,50</sup>

#### MEMBRANE TEARS

Other possible injuries from IEBt are tears and/or haemorrhage of the Reissner's, basilar or tectorial membranes.<sup>7,23</sup> These are termed intralabyrinthine or intracochlear tears and may be associated with MEBt.<sup>20,21,23,26</sup> It has been postulated that intracochlear ruptures are responsible for SNHL with additional interruption to the striae vascularis, hair cells and ancillary cells.<sup>10</sup> Injury of Reissner's membrane (also known as the vestibular window) may cause SNHL through a direct effect on the cells of hearing in the organ of Corti, or through perilymph/endolymph mixing, which changes the ion concentrations around the structures of hearing.<sup>7,23,39,45</sup> Basilar membrane damage can result in long-term SNHL by compromising the organ of Corti.<sup>7</sup> Membrane tears can be diagnosed clinically on PTA by a lingering SNHL isolated to the frequency that corresponds to the anatomical location of the membrane tear.<sup>23,26</sup> Membrane tears may also result in vertigo and nausea.<sup>7</sup> Symptoms do not appear pathognomonic for a specific membrane injury.<sup>11</sup> There also exists a 'double membrane break theory' that proposes a RWR or OWR may be combined with an intracochlear (i.e., Reissner's, basilar, tectorial) membrane tear.<sup>6,39</sup>

#### INNER-EAR HAEMORRHAGE AND GAS

With IEBt, there also may be direct physical damage to the hair cells or their blood supply caused by expanding gas in the inner ear, in particular the scala tympani and scala vestibuli from air forced from the middle ear through a PLF (pneumolabyrinth).<sup>7,10,44,47</sup> Clinical presentation is similar to RWR and OWR, with SNHL of varying severity and/or transient vestibular symptoms.<sup>23,26</sup> Inner ear haemorrhage tends to have a more sudden onset.<sup>3</sup> Isolated hearing deficits appear to be predominantly due to intracochlear membrane pathology and associated structures of hearing rather than RWR or OWR.<sup>39</sup>

#### Predisposing factors

Divers suffering IEBt may report difficulty in equalising on descent or ascent, or application of an internal or external force on the ear while diving or immediately post dive (e.g., forceful Valsalva, wave trauma, removing a wetsuit or hood, or lifting heavy equipment).<sup>3,16,21,22,25,40,51</sup> In a retrospective review of 50 Australian divers sustaining IEBt, 24 had acute or chronic ear, nose and throat (ENT) pathology.<sup>21</sup> This suggests that IEBt in divers results from problems in equalising, supported by a New Zealand case series in which

a history of difficulty in ear clearing and/or respiratory infection was identified in three-quarters of the divers.<sup>25</sup>

Some individuals appear prone to recurrent PLF, suggesting anatomical predisposition; one study identified recurrent IEBt in two of 44 divers.<sup>16</sup> Post-mortem histological examination of temporal bones has demonstrated an association between PLF and enlarged vestibular and/or cochlear aqueducts, permitting greater CSF-perilymph communication.<sup>6,45,52</sup> These anomalies may be detected on high-resolution computed tomography (HRCT).<sup>16</sup> This contrasts with the proposed hypothesis that a small cochlear or vestibular aqueduct could make an ear more prone to implosive or internal pressure changes.<sup>39</sup>

Aplasia or malformations of the cochlea, and weakness or malformation of the bony structures, otic capsule, or inner ear membranes may also predispose to IEBt.<sup>45,52,53</sup> Other congenital causes of IEBt (OWR) include weakness of the annular ligament of the stapes, and abnormalities of the stapes bone.<sup>6,23,39</sup> Anatomical abnormalities of the windows may also pose a risk for IEBt.<sup>39</sup> None of the aforementioned factors would be detected during routine physical examination. In a non-diving series of 44 cases of PLF, half had no clear precipitating cause, suggesting possible spontaneous PLF in some individuals.<sup>51</sup>

IEBt is a rare diving injury; hence screening for congenital risk factors is not appropriate at the time of health screening. There is merit in identifying divers who have difficulty with ear clearing or greater risk of MEBt, because the evidence is that it increases the risk of IEBt.

#### Diagnosis of IEBt in divers

##### ONSET OF SYMPTOMS

The timing of symptom onset in relation to a diving event is a key element in identifying IEBt. Symptom onset (vestibular dysfunction or hearing impairment) after difficulty equalising on descent is a consistent finding in many IEBt case series.<sup>14,21,23,24,26,40</sup> IEBt may also become manifest during ascent; secondary to pneumolabyrinth and gas expansion, injuring the cochlea or vestibular apparatus.<sup>12</sup> The symptoms of IEBt may even occur some days post dive, particularly if provoked by lifting or straining.<sup>1,54</sup>

Unfortunately symptom onset may be vague, particularly if SNHL is not accompanied by vestibular symptoms. IEBt has a spectrum of severity, depending on the anatomical site of insult and degree of injury. Tears in Reissner's or the basilar membrane (within the cochlea) without RWR may present as isolated SNHL. Some resolve spontaneously within days.<sup>6</sup> These cases are thought to be under-reported because of their less dramatic presentation and self-resolution. The existence of subclinical IEBt has been suggested, which may be detected as minor degrees of SNHL in divers measured by

otoacoustic emission (OAE) testing.<sup>54</sup> However, this study was limited by the absence of a non-diving control group.<sup>54</sup>

The major differential diagnosis of IEBt is IEDCS. The two conditions pose a diagnostic dilemma, sharing similar clinical presentations: nausea, vertigo, nystagmus, SNHL, and tinnitus.<sup>14,21,46,55</sup> The original descriptions of five divers with IEBt described SNHL occurring after very low-risk dives.<sup>40</sup> Dive risk stratification remains a significant part of clinical algorithms when assessing new vestibulocochlear symptoms in divers. A provocative dive profile, uncontrolled ascent or missed decompression stops, onset of symptoms after the dive, other manifestations of decompression illness (DCI) or mixed gas diving indicate a greater likelihood of IEDCS.

IEDCS commonly occurs in divers using gas mixtures other than air (e.g., heliox) during their dive, although IEDCS in air divers is being reported in increasing numbers.<sup>20,31,46,56</sup> Up to 48% of divers with IEDCS have other symptoms of DCI.<sup>56</sup> In contrast, low-risk, shallow dives, sudden onset of symptoms during ear clearing manoeuvres, a past history of ear barotrauma, symptom onset in relation to Valsalva/straining (during or post dive), and coexistent MEBt all suggest IEBt.<sup>20,21,31,46,54-56</sup> It is important to note however, that individuals with IEBt may have normal tympanic membranes.<sup>21,46</sup>

It has been suggested that recompression could theoretically exacerbate the symptoms of IEBt, whereas IEDCS symptoms should improve.<sup>9,31,46</sup> This assertion has not been confirmed. In the retrospective series of 50 divers, three were recompressed without incident.<sup>21</sup> Other studies also have found that recompression can be conducted on suspected PLFs without adverse effects, provided tympanostomy tubes are present.<sup>3,55</sup> The risks of tympanostomy in an already injured ear must be carefully weighed against any theoretical benefit. It has been hypothesised even that IEBt may be improved by recompression because gas that has entered the inner ear via a window rupture may be redistributed or removed during recompression.<sup>31</sup>

Other differential diagnoses include alternobaric vertigo, and non-diving medical causes. Alternobaric vertigo is due to differences in middle ear pressures between the right and left ears resulting in disequilibrium and vertigo which usually manifest during ascent.<sup>57,58</sup> MEBt was reported by 24 of 67 occupational divers, in whom transient dizziness and vertigo developed during (24 divers) or soon after (10 divers) working dives.<sup>57</sup> Symptoms were short-lived post-dive (hours only) and not associated with demonstrable hearing injury or long-term effects.<sup>57</sup> Persistent symptoms should raise the possibility of IEBt. Non-diving differential diagnoses include Meniere's disease, benign paroxysmal positional vertigo (BPPV), or vestibular neuritis.<sup>18,51</sup> Where symptom onset coincides with diving, diving pathology should be considered and/or treated before assigning a non-diving diagnosis.

## CLINICAL PATTERN OF SYMPTOMS

Vestibular injury is characterised by dizziness, constant dysequilibrium, positional vertigo, positional nystagmus, imbalance, ataxia, nausea and possibly vomiting.<sup>1,7,51</sup> These symptoms typically worsen with activity and loud noise (the 'Tullio effect'), and are improved or relieved with rest.<sup>51</sup> Disequilibrium caused by IEBt may fluctuate, provoked by actions that increase ICP, e.g., sneezing or straining.<sup>51</sup> Vestibular symptoms neither rule in nor rule out PLF, but do appear amenable to surgical repair.<sup>1,14,47</sup>

Hearing deficits may be perceived as aural 'fullness', 'muffled' hearing, tinnitus, hyperacusis, or complete hearing loss.<sup>1,24</sup> Often, impaired speech discrimination is noted.<sup>22,41,59</sup> The severity of the symptoms correlates with the extent of the injury and is a prognostic indicator for recovery.<sup>1</sup> The pattern of SNHL loss demonstrated at PTA for IEBt may be variable. The critical issue is that it is recognised. A pre-incident audiogram assists to define the degree of injury.<sup>21,40</sup>

## THE CHALLENGE OF PRECISE PATHOPHYSIOLOGICAL DIAGNOSIS

Given that the exact injury from IEBt may be microscopic and deep within the inner ear, it is challenging for the clinician to identify the specific pathophysiology in any affected individual. Diagnoses of PLF secondary to RWR or OWR dominate case series, and originate from the surgical literature. The exact cause may only be discovered during tympanotomy or at autopsy, or occasionally by high-resolution imaging techniques. Case series describe membrane tears and haemorrhages as diagnoses of exclusion, based on clinical findings, or when PLF has been ruled out at tympanotomy.<sup>23</sup> It is clinically difficult to differentiate between trauma to the cochlea or vestibular apparatus, haemorrhage in the inner ear and PLF.<sup>3,23</sup>

### Investigation of IEBt

When assessing the usefulness of investigation of IEBt, data are available from both diving and non-diving case series. Some mixed-aetiology series include only one or two divers, whilst most non-diving series originate from the surgical literature and focus on identifying PLF. A comprehensive table of all studies from our literature search is available from the authors. However, in Table 2, we document only those clinical series focusing solely on IEBt in divers.

Initial assessment of suspected IEBt should include a general head and neck examination, otoscopy, Rinne and Weber tests, cranial nerve testing and cerebellar testing, including sharpened Romberg and Hallpike manoeuvres. Where there is clinical suspicion of IEBt, numerous tests are available for otoneurological assessment (Table 3). Essential tests for IEBt include air and bone (if available) PTA to diagnose and quantify SNHL, and the fistula test to clinically assess

inducible vertigo. Positional PTA has been used to assist in the diagnosis of PLF in non-divers.<sup>47</sup> A positive test was defined as a hearing gain of at least 10 dB in two or more frequencies when the subject lay supine with the affected ear uppermost.<sup>47</sup>

#### AUDIOMETRY

Audiometry findings may be variable in IEBt. Non-divers with PLF present with all three patterns of audiometric abnormalities: ascending curves 22.5%, descending curves 40% and flat global curves in 37.5%.<sup>60</sup> Both global and descending curves have been reported in divers with IEBt.<sup>40</sup> SNHL involving the higher frequencies (descending curve) has been associated with RWR.<sup>43</sup> The traumatised area in the membranous labyrinth can be mapped using audiometry in increments of 100 Hz, which demonstrates isolated damage to the cochlea in a narrow frequency range.<sup>6</sup> Audiograms are also useful to monitor recovery from the injury.<sup>41</sup> Hearing loss appears more likely to improve if low to mid frequencies are affected (250–1,000 Hz).<sup>3</sup> This is useful functionally as it reflects the speech frequencies.

PTA is currently required prior to clearance for occupational (and recreational) diving medicals in Australia (AS/NZS 2299 Standards and AS4005.1).<sup>61,62</sup> This assists in the assessment of diving-related ear injuries by providing a reference baseline for comparison.<sup>21,40</sup> The Australian recreational industry recently abandoned AS 4005.1, which will result in a diagnostic challenge of IEBt in recreational divers as they will be less likely to possess reference audiograms in the future.

#### FISTULA TEST

The fistula test has been classically used to detect RWR or OWR. The test involves a variable pneumatic pressure applied to the external ear with the aim of inducing vestibular symptoms or nystagmus. A positive test led to exploration in a number of series; however, its limited sensitivity and specificity have prevented the test becoming a 'gold standard'.<sup>4,41,51,60</sup>

#### ELECTRONYSTAGMOGRAPHY AND COMPUTER TOMOGRAPHY

ENG has been used to diagnose and quantify vestibular injury.<sup>4,6,21,22,41,49,51,52,57,63,64</sup> ENG may be combined with a pneumatic test (fistula test, Valsalva), or positional changes to increase its sensitivity.<sup>5</sup>

HRCT of the temporal bones has been recommended for IEBt to identify individuals at risk of an enlarged CSF-perilymph communication via a widened cochlear aqueduct orifice and enlarged internal auditory canal.<sup>10</sup> Two of 44 divers with recurrent IEBt were found to have anatomical abnormalities, and HRCT was stated to assist assessment

in returning to diving.<sup>10</sup> It was not recommended for initial diagnosis of IEBt, but may have potential in identifying pneumolabyrinth.<sup>44</sup>

A key conundrum is whether or not to undertake surgical exploration and repair of the subset of IEBt that is due to PLF. It is difficult to develop clear guidelines from the non-diving literature regarding investigation of PLF in divers. Many of the series are of mixed aetiology including external trauma cases in non-divers.<sup>1,4,5,47,48,60,65</sup> A number of series report the findings of tympanotomy, but only loosely report the selection criteria for performing the procedure.<sup>1,4,41,47,48,51,53,60,65</sup> Clinical algorithms included PTA; however, there was variable reporting of other investigation even in recent series.<sup>1,48</sup> Most of the series are retrospective, introducing selection bias.

#### SURGICAL EXPLORATION

In 26 of a series of 51 cases, a fistula was demonstrated intraoperatively.<sup>4</sup> No diagnostic tests confirming PLF preoperatively could be identified.<sup>4</sup> Using clinical criteria to diagnose barotraumatic PLF, combined with PTA, nine suspected cases were exposed surgically, with eight having PLF confirmed intraoperatively.<sup>1</sup>

#### IEBt vs. IEDCS

Two clinical series of divers have documented diving events that produced cochlear (hearing or tinnitus) and/or vestibular (dizziness, balance, vertigo) symptoms, where there was a low risk of DCI.<sup>21,14</sup> In one, a significant percentage of divers who had problems with equalisation had subsequent onset of vestibulocochlear symptoms occurring within 24 hours of the dive, but mostly on ascent.<sup>21</sup> ENG demonstrated subclinical vestibular injury in four of 50 divers.<sup>21</sup> In the other, ENG, auditory brainstem response (ABR) and the vestibulo-ocular response smooth harmonic acceleration (SHA) test were used to differentiate central from peripheral causes of vestibular abnormality in divers.<sup>14,46</sup> The algorithm used was not well explained, making it difficult to separate the groups clinically other than via diving risk stratification and ear symptoms.<sup>14,46</sup>

In another series, clinical criteria were used to differentiate IEDCS from IEBt; however, once again the precise algorithm was not described.<sup>55</sup> From these data, 18 divers diagnosed with IEDCS appeared to have vertigo as a major symptom, and 26 diagnosed with IEBt had mainly hearing impairment (although nine had vestibular symptoms).<sup>55</sup> Only five of the 18 divers in the IEDCS group had audiograms, which makes interpretation difficult.<sup>55</sup> Large right-to-left cardiac shunts were detected in 15 of the 18 divers with IEDCS using bubble contrast echocardiography.<sup>55</sup> Unfortunately the study was weakened by failure to perform this test on divers identified as IEBt. Video oculography also was used in their diagnostic cache, but too inconsistently for analysis.<sup>55</sup>

**Table 2**  
Summary of case series of inner ear barotrauma in divers (see Table 1 for definition of acronyms)

Author (Year)	Study	Diagnostic criteria	Indication for surgery	Treatment	Outcome
Freeman P, Edmonds C 1972 <sup>40</sup>	Series of 5 Navy divers with SNHL	Clinical criteria associated with diving; IEDCS ruled out by risk stratification of dives; audiometry pre and post incident.	No surgery	Conservative: all	5/5: divers: persistent SNHL 2/5: global hearing loss 3/5: high frequency SNHL
Parell J, Becker G 1985 <sup>23</sup>	Series of 14 divers	Clinical criteria associated with diving; PTA, fistula test and ENG.	Vestibular symptoms and severe hearing loss	Conservative: 12/14 Surgical: 2/12 tympanotomy + repair	Conservative: 7/12 improved Surgery: 2/2 improved, 1 fully
Shupak A et al 1991 <sup>46</sup>	Series of 5 recreational divers	Clinical criteria; cases separated from IEDCS by risk stratifying the dive + ENG, ABR and SHA test. Algorithm not clearly defined.	Persistent vestibular symptoms	Conservative: all for at least 72 h initially Surgical: 2/5 1 confirmed RWR + PLF, patched; 1 not confirmed; both windows patched	Conservative: 3/5 cases, 2 improved hearing, 3 improved vertigo Surgery: Both improved vertigo and hearing, 1 fully
Parell J, Becker G 1993 <sup>24</sup>	Long-term follow up of 20 divers (7 professional, 13 recreational) with confirmed IEBT who continued diving; may include some of 1985 series)	History ear injury/symptoms associated with diving; clinical criteria + PTA, fistula test and ENG. (all SNHL, 10 vestibular +/- tinnitus, 7 tinnitus only)	Vestibular symptoms and/or severe hearing loss	Conservative: 16/20 cases (not PLFs) Surgical: 4/20 cases	Conservative: 9/16 improved Surgery: 2/4 improved Long term outcomes: 19/20 remained stable (median follow up 54 months; 8–2,000 dives completed post injury)
Roydhouse N 1997 <sup>25</sup>	Series of 20 divers; 19 RWR, 1 OWR (16 cases personally managed by author)	Clinical criteria – onset vestibular symptoms +/- deafness during/after diving.	Positive fistula sign or worsening signs/symptoms	Conservative: 6/20 cases Surgical: 14/20 (4/20 managed by surgeons other than author)	Conservative: unclear Surgery: 8/10 PLF confirmed; 10/16 continued diving without issues (loose follow up) Surgical candidates stated to have better prognosis but not supported by presented data
Sheridan M et al 1999 <sup>26</sup>	Series of 3 divers	Clinical criteria – onset during/after diving of vestibular symptoms +/- deafness + fistula tests.	Worsening vestibular symptoms	Conservative: 1/3 (suspected intracochlear haemorrhage) Surgical: 2/3 cases (1 OWR, 1 RWR); fistula tests equivocal or -ve in surgically diagnosed PLFs	Conservative: hearing improved (resumed diving) Surgery: improved vestibular symptoms and hearing (neither resumed diving)



**Table 2** (continued)

Shupak A et al 2003 <sup>14</sup>	Series of 43 recreational divers (24 IEDCS, 19 IEBt); 9 IEBt cases were followed up long term	Same methodology to separate IEDCS/IEBt as Shupak (1991), but clinical criteria unable to distinguish IEDCS/IEBt. Follow up: mean 28 months, range 1–72 months	Deterioration of hearing or severe vestibular symptoms, 2 cochlear, 3 cochlear + vestibular	Conservative: 7/9 Surgical: 2/9 tympanotomy with both windows repaired (these cases were not specifically identified)	Conservative vs. surgical outcomes unable to be identified Cohort outcomes reported only: 6/9 fully recovered, 2/9 residual SNHL, 1/9 residual vestibulocochlear symptoms
Edmonds C 2004 <sup>21</sup>	Series of 50 cases IEBt	Occurrence in low-risk-DCI air diving: clinical criteria (40 had SNHL, 43 tinnitus, 16 vertigo, 4 dysacusis); PTA ( $\geq$ 20dB loss in $\geq$ 2 frequencies), or ENG +/- confirmed PLF at surgery	Persistent tinnitus and vestibular symptoms	Conservative: 33/50 cases Surgical: 13/50 cases 4/50 cases: unable to identify treatment	Conservative: 3/33 full recovery, 21/33 improved tinnitus and vestibular symptoms Surgery: 2/13 cured, 9/13 improved, 1/13 worsened
Klingmann, C et al 2007 <sup>35</sup>	Series of 44 divers with 46 cases of acute inner ear disorders 26 divers diagnosed IEBt	Not stated how separated IEBt from IEDCS; appeared to use clinical criteria (vertigo precluded diagnosis of IEBt)	SNHL > 40 dB or severe vertigo with nystagmus	Conservative: 23/26, including high-dose prednisolone, decongestants and rheologic therapy Surgical: 3/26 tympanotomy + RWR closure	Conservative: 18/23 persisting vestibulocochlear symptoms Surgery: 1/3 complete recovery, 2/3 no hearing improvement

**Treatment of IEBt**

The traumatic aetiologies of PLF have been classified into head trauma, acoustic trauma (external trauma), trauma caused by physical exertion, and barotrauma (internal trauma).<sup>66</sup> Surgical trauma due to stapedectomy would fall under the first group. It may not be valid to generalise outcomes from treatment of external traumatic causes to barotrauma-induced injury. The internal trauma group is applicable to diving barotrauma and, where relevant, outcomes from clinical series of mixed aetiology are included in this section.

**CONSERVATIVE MANAGEMENT**

Conservative management is recommended by a number of authors as initial treatment for IEBt because of its broad pathology.<sup>1,24,26,45,46,51</sup> Generally bed rest is recommended, with the head elevated to 30–40 degrees for one to two weeks, and avoiding significant physical activity for two weeks.<sup>39,53,67</sup> Conservative interventions also include; resting prone, taking decongestants and vestibular suppressants and avoiding activities that provoke an increase in CSF or ICP (e.g., heavy lifting, straining, nose blowing, coughing, sexual intercourse, ear clearing, air travel, high-speed elevators, and loud noises).<sup>14,20,39,45,49,51,53,67</sup> Even subtle changes in ICP caused by moving from a supine to erect position produce some stretch on the round window membrane.<sup>39</sup> Ear plugs have been used to protect against loud noise, and external ventilation tubes also known as ‘ear planes’ may aid by damping changes in air pressure in the external canal.<sup>2</sup>

Steroids have been used concomitantly with conservative management. The logic behind steroid treatment is its anti-inflammatory properties and to reduce swelling.<sup>30,65</sup> Steroids are recommended management for sudden SNHL and vertigo of possible vascular aetiology.<sup>30</sup> Prednisolone 60 mg for 10 days has been recommended for occupational divers within three weeks of injury but high-level evidence is lacking.<sup>54</sup> Trans-tympanic application of steroids has also been trialled.<sup>30,54</sup> To date, there are no studies supporting or refuting the efficacy, precise dosing or period of administration of steroids in the treatment of IEBt.<sup>1</sup> Vasodilators, anticoagulants (aspirin and NSAIDs) and nicotinic acid are contra-indicated in the treatment of suspected IEBt.<sup>21,39</sup> Other medications (anti-emetics, decongestants, sedatives), may be prescribed ‘as required’ for symptom control, and may reduce episodes of raised ICP.<sup>22,54</sup>

**TYMPANOTOMY**

The justification to undertake an exploratory tympanotomy is supported by clinical suspicion of PLF, persistent vestibular symptoms, and severe hearing loss. A positive fistula test may support the decision.<sup>6,20,25,44,46,53,60,65</sup> If vestibular symptoms are debilitating or strongly suggestive of a PLF, relatively urgent surgical repair is recommended by various

**Table 3**  
Summary of the available diagnostic tests and features consistent with inner ear barotrauma

Investigation	Indication	Features/limitations
Rinne test	512 Hz tuning fork used to determine a conductive hearing loss (CHL). For IEBt the response will suggest sensorineural hearing loss (SNHL); both bone conduction and air conduction will be impaired. <sup>29</sup>	Useful if bone conduction PTA is not accessible.
Weber test	For SNHL, the vibrations from 512 Hz tuning fork are perceived louder in the non-affected ear (for CHL they are louder in the affected ear). <sup>29</sup>	Mixed pathology of SNHL and CHL (e.g., associated MEBt) causes difficult test interpretation. <sup>29</sup> SNHL with IEBt may present as a normal ear as affected frequencies can be higher than 512 Hz. <sup>29</sup>
Fistula test	Aims to induce nystagmus (Hennebert's sign) or subjective dysequilibrium (Hennebert's symptom). Positive and/or negative pressure is applied against the eardrum, commonly with a finger on the tragal cartilage, with a rubber bulb or pneumatic otoscope. <sup>11,13,51,63,64</sup> PLF is likely if symptoms are produced. <sup>41</sup>	Accurate in reportedly < 50–70% of cases with PLF. <sup>51,52</sup> Fistula tests may be a useful non-operative diagnostic technique if positive; not sensitive nor specific. <sup>51</sup>
Pure tone audiometry (PTA)	Detects and quantifies SNHL. Air and bone conduction audiograms are the best way to differentiate between MEBt, 8th cranial nerve involvement in IEDCS and IEBt, with IEBt presenting with a mostly high tone hearing deficit (compared to a global SNHL). <sup>20,26</sup> MEBt demonstrates CHL. <sup>57</sup> IEBt and PLF may demonstrate SNHL in the higher frequencies (4,000 Hz plus) or all frequencies. <sup>20</sup> Speech audiometry may demonstrate difficulties with word recognition in PLF. <sup>66</sup> Can aid in anatomically isolating SNHL to the cochlea (secondary to IEBt) or the auditory nerve when compared with PTA. <sup>29</sup> Useful in monitoring recovery. Successful treatment of IEBt = improvement in hearing to within 15 dB of the pre-incident audiogram. <sup>20</sup>	Bone conduction audiometry is not as available or as sensitive as air conduction testing (requires a transducer). <sup>29</sup> Useful in excluding a CHL, particularly in excluding MEBt. <sup>27</sup> Comparison of pre- and post-incident PTA provides invaluable data supporting a confident diagnosis of PLF, as SNHL suggests IEBt. IEBt on a post-incident audiogram is defined as hearing loss $\geq 20$ dB in two or more frequencies, compared to the initial PTA. <sup>20</sup> Positional PTA (PTA conducted when lying with affected ear up) is deemed positive if $\geq 2$ frequencies improve by at least 10 dB. <sup>47,66</sup> Vertical PTA shows a greater hearing loss to a horizontal PTA after 30 minutes waiting period owing to displacement of air trapped in the cochlea affecting the conduction of sound. <sup>9,20,47,66</sup> Positional PTA is specific for PLF. <sup>9,47</sup> Higher frequency SNHL, i.e., > 4,000 Hz, may also be caused by industrial deafness (a confounder, but is usually bilateral).
High resolution computed tomography (HRCT) – temporal bones	Excludes inner ear and intracranial lesions. Excludes inner ear anomalies that may predispose the person to ongoing IEBt. In acute situations, may identify air in the cochlea or vestibular apparatus (pneumolabyrinth), thereby confirming the presence of a PLF peri-operatively. <sup>8,44,47</sup> Can also exclude the presence of superior canal dehiscence (SCD) which has implications for management (surgery is less successful in the presence of a SCD). <sup>8</sup>	HRCT (1.0–1.5 mm slices) allows the ability to differentiate otic cortical bone from air (which MRI fails to do). <sup>44</sup> Not available in all hospitals and not readily accessible in a timely manner. Requires radiation exposure. Negative test does not rule out IEBt.
Electronystagmography (ENG)	Detects and quantifies vestibular disturbance, as eye movements are electronically recorded in response to provocation. <sup>13,28</sup> Particularly useful if vestibular symptoms are not overtly subjective. <sup>20</sup> Differentiates peripheral from central (cerebral and cerebellar) causes. <sup>20</sup> Nystagmus can be spontaneous, or provoked by eye movements, positional changes, optokinetics, and caloric testing. <sup>38</sup>	In 50-case series, four cases who denied vestibular symptoms had positive ENG on testing. <sup>20</sup> Accurate in 35–91%. <sup>10</sup>

**Table 3 (continued)**

Caloric test	Tests vestibular function. Ear canal is exposed to water hotter or colder than body temperature, i.e., 30–44°C, stimulating a compromised labyrinth and inducing nystagmus which can be directly observed or recorded via ENG. <sup>38</sup>	Cannot be conducted on perforated tympanic membrane. <sup>9</sup> Low specificity in identifying PLFs. <sup>10</sup>
Tullio phenomenon	The affected ear is exposed to a burst of noise (95 dB at 500 Hz) for a few seconds to induce dysequilibrium and/or vertigo = positive test, which may be a sign of PLF. <sup>44,51</sup> Initiates a vestibulospinal reflex from otolith organs. <sup>48</sup>	Low sensitivity in identifying PLFs. <sup>44</sup>
Otoacoustic emission (OAE) testing	OAEs are spontaneous or responsive sounds made by the cells within the inner ear, giving an appreciation of functionality of the inner and middle ear. <sup>54</sup> Helps differentiate cochlea injury from other forms of IEBt. <sup>42</sup> Can detect subclinical IEBt in divers, identifying 'transient emission shifts' as a more sensitive assessment of objective SHL. <sup>54</sup>	Only accessible in specialist laboratories.
Tympanotomy	A diagnostic procedure and treatment for PLF.	Direct vision of PLF possible. High correlation with resolution of vestibular symptoms if PLF confirmed at operation then repaired. <sup>1,3,48,53</sup>

authors.<sup>6,14,20,23,39,42,46,51,52,65</sup> Likewise, if regular PTA testing (daily in some studies) demonstrates deterioration, surgical treatment may be indicated.<sup>47</sup> A number of studies show significant improvement in vestibular symptoms post surgery (Table 2).<sup>1,6,22,26,46,51,53,65</sup>

Tympanotomy confirms the diagnosis of PLF when leaks are seen from either window at exploration. False positives may occur: an iatrogenic fistula, pooling of local anaesthetic, or misinterpretation of the tissue architecture (e.g., adhesions) for a window rupture.<sup>47,60,68</sup> A number of techniques provoke leakage of the perilymph during tympanotomy: Valsalva manoeuvre in a conscious patient; Trendelenberg positioning; Quekenstedt manoeuvre; internal jugular vein compression or increasing the intrathoracic pressure in an intubated patient.<sup>6,45,48,51,60</sup> PLF may not always be observed; however, a graft placed over both windows may improve symptom severity.<sup>1,6,13,26,51</sup> It may also indirectly promote healing of intracochlear structures. Grafting carries a potential risk of inducing further CHL in an ear with SNHL, hence it is usually undertaken in the setting of more severe symptoms.<sup>45</sup> Despite this caveat, tympanotomy and grafting appears a relatively safe intervention.<sup>1,4,6,48,51,60</sup>

According to studies of divers, ataxia and vertigo are significantly improved with surgical intervention, and tinnitus is mostly relieved; hearing recovery is less consistent.<sup>3,6,39,44,47,65,67</sup> However, the procedure has not been tested in a randomized controlled trial, and any comparative trials are limited by selection bias (Table 2). A theoretically increased risk of labyrinthitis in the setting of a PLF has not been proven; however, case studies have documented a link with chronic PLF.<sup>39,45,65</sup> Out of positive PLFs confirmed surgically, 96% had a resolution in vestibular symptoms with window closure.<sup>4</sup> Even those identified clinically at risk of PLF but not confirmed at surgery had a 68% recovery from vertigo, dizziness, nystagmus, and other symptoms after surgical repair.<sup>4</sup>

A key finding from this and other studies was that no individual had deterioration in their symptoms despite all cases receiving tympanotomy and repair irrespective of whether a PLF was confirmed. The associated symptoms of nausea, vomiting and motion intolerance also are improved, as is cognitive memory.<sup>48</sup> Surgical intervention for RWR was successful in treating vertigo for all patients in another series, but had variable effect on tinnitus and hearing loss.<sup>53</sup> Similar findings were found in a small series of nine cases.<sup>1</sup>

In the retrospective series of 50 divers with IEBt, two thirds were treated conservatively with improvement in two thirds of these, but complete recovery in only five divers.<sup>21</sup> A quarter underwent tympanotomy with mixed results. One patient reporting worse symptoms post-operatively.<sup>21</sup> In one diver; the delay to surgery was 10 weeks post injury, yet symptoms of vertigo and tinnitus improved markedly.<sup>21</sup> Small numbers and multiple other confounders prevented

**Table 4**  
Other investigations of limited use in the diagnosis of inner ear barotrauma

Investigation	Indication	Features/limitations
Auditory brainstem response (ABR)	Distinguishing cochlear from retrocochlear lesions <sup>30,46</sup>	Aids with determining auditory and vestibular pathway pathology in the brainstem; useful in isolating inner ear versus central nervous system causes <sup>46</sup>
Smooth harmonic acceleration test (SHA)	Assessment of the vestibulo-ocular response <sup>46</sup>	Used alongside ABR to isolate central versus peripheral pathologies; <sup>46</sup> requires highly specialised laboratory
Magnetic resonance imaging (MRI)	Excluding differential causes	Sensitive in detecting soft tissue lesions, e.g., acoustic neuroma, vestibular schwannoma, cholesteatoma, or multiple sclerosis plaques; <sup>8,14,30</sup> not widely available
Biochemical markers e.g., 2-transferrin	Visualisation of 2-transferrin intraoperatively (when applied as an assay); <sup>44</sup> 2-transferrin only produced in CSF and perilymph <sup>45</sup>	PLF is otherwise too small to detect directly. Time consuming (hours) laboratory analysis, therefore, impractical for intra-operative diagnosis <sup>45</sup>
Craniocorpography (CCG)	An objective test in which the upper body is recorded with the patient marching on the spot while blindfolded <sup>60</sup>	Not sensitive or specific for IEBt <sup>60</sup>
Electrocochleography	Aids in delineating Meniere's disease from PLF <sup>8</sup>	Technically challenging; limited access to testing; difficult interpretation
Vestibular evoked myogenic potential (VEMP)	Identifies Tullio phenomenon from SCD <sup>8,14</sup>	Operator dependent; high error risk = poor quality

formal statistical comparison between surgical and conservative groups. Normal hearing was also reported post-operatively in two of 13 cases; the two with resolution were either operated on shortly post injury or had only a very small fistula.<sup>53</sup> Exploratory tympanotomy may provide diagnostic and curative management of a PLF, although there is some debate as to the indication and timing of exploratory surgery.<sup>47,60</sup>

#### TIMING OF REPAIR OF PLF

Timing of surgical exploration and repair of PLF has received considerable attention, derived from studies of both diving-related and non-diving-related trauma. The general opinion is that urgent tympanotomy is unnecessary.<sup>1,6,22,44,45,46,51,53,55,65</sup> One to three weeks is recommended to allow for spontaneous healing or resolution of acute ear pathology and facilitation of access to the labyrinthine windows, depending on the severity of SNHL and vertigo.<sup>6,22,23,44,46,53,67</sup> For incapacitating vertigo, severe hearing loss or deteriorating hearing post injury, tympanotomy within 24–48 h of injury has been recommended.<sup>3</sup> It appears that little improvement in hearing is achieved if exploration occurs greater than two weeks post injury.<sup>3</sup>

With strict bed rest, spontaneous healing should occur within four days, with five to 10 days as the limit to conservative management for suspected MEBt and IEBt not including PLF before review and consideration for surgery.<sup>23,39,53</sup> Others recommend early (< 48 h) surgery if the tympanic membrane is also ruptured, because OWR was more likely in this setting.<sup>65</sup> Some recommend antibiotics, sometimes with steroids, prior to any repair undertaken later than 48 hours post injury.<sup>65</sup> In one series, hearing improved within 10 days of injury compared with those who waited four to six weeks.<sup>1</sup> Ten days post injury seems to be accepted as the best time to intervene, with no maximum time. The ability to improve hearing does depreciate with time.<sup>1,26,53,67</sup> The conservative approach allows time over weeks to months for central vestibular compensation to occur, correcting peripheral vestibular dysfunction.<sup>19,57</sup>

There is an up to 10% risk of recurrence after surgical repair.<sup>4</sup> Tympanostomy tubes have been used to aid resolution of PLF symptoms by reducing external pressure effects on the stapes.<sup>2</sup> However, this is not an appropriate management option for occupational divers. If symptoms persist or return following PLF repair, one author recommended repeating

the repair no more than twice.<sup>2</sup> The severity of symptoms correlates negatively with prognosis, irrespective of the treatment.<sup>1</sup>

### Prognosis for the injured diver and returning to diving

It has been proposed that divers with IEBt are prone to further incidents that exacerbate their tinnitus and hearing loss,<sup>3</sup> but this is not supported by others.<sup>14,24,39</sup> One group stated; *“Although the older literature clearly suggests otherwise, we believe that scuba divers who completely recover from inner (or middle) ear barotrauma may return to diving as long as they exercise caution and care”*.<sup>26</sup> However, this advice was not backed up by what these authors reported, as two of their three cases (both undergoing tympanotomy) were advised not to continue diving. Long-term follow up of IEBt cases (seven conservatively and two surgically managed) reported complete recovery of symptoms in six patients, and return to diving as early as one month post tympanotomy repair was permitted, provided that predisposing anatomical abnormalities were excluded, hearing was stable, and their balance was normal.<sup>14</sup> This study has some limitations as it included both IEDCS and IEBt cases and lacked a clear clinical algorithm for making the diagnosis. In addition, follow-up was reported for only nine of the 19 divers, creating significant risk of positive selection bias.<sup>14</sup>

Of 20 divers who returned to diving against medical advice, only one sustained a further episode of IEBt.<sup>24</sup> Seven of these were professional divers and four had undergone surgical repair of their PLF.<sup>24</sup> These findings suggest that divers may not be at increased risk of recurrence of IEBt when returning to diving. It was not reported whether these divers were part of a larger cohort of divers with IEBt who received the standard counselling post injury *“to discontinue diving”*.<sup>24</sup> Once the manifestations of IEBt have settled with no disequilibrium, hearing is within normal range and other clinical parameters normalised, it has been suggested that the diver can return to light duties after 10 days, full duties at six weeks, and diving after three months.<sup>3</sup>

In the retrospective series of 50 divers with IEBt, return to diving was not supported, on the basis that a high proportion of them had predisposing risks, such as MEBt and ear clearing difficulties.<sup>21</sup> In contrast, the recommendation that *“after careful and probably radical repair of a round window membrane rupture, the diver can return to diving”* was on the proviso that the diver received counselling from an ENT surgeon knowledgeable about diving, and adhered to safe diving practices, including ear clearing.<sup>25</sup>

In 917 patients responding to a survey sent to 2,222 past stapedectomy patients, 208 had gone snorkelling, scuba diving or sky diving post procedure.<sup>69</sup> Twenty-eight provided detailed responses regarding symptoms in relation to diving activities and of 22 who had scuba dived, four had experienced otalgia, tinnitus or vertigo and one had SNHL

and vertigo unrelated to diving. It was concluded that there were no significant diving-related long-term effects when diving after stapes surgery.<sup>69</sup> These results may be affected by selection bias.

Characteristics that would exclude divers and who should avoid further risk or aggravation of IEBt from returning to diving are:

- symptomatic non-compensated vestibular damage;
- anatomical risk factors identified on HRCT or tympanotomy;
- persistent difficulties with ear clearing and MEBt;
- persistent, significant global hearing loss.

The risk not only involves compressed gas diving, but also other activities such as free diving, skydiving, and flying, where pressure changes can be pronounced.<sup>3</sup>

From this literature review, the recommendation for returning to diving depends on satisfying five criteria:

- Hearing loss is in a narrow frequency band and stabilised.
- Vertigo or imbalance is not a feature.
- Risk factors for MEBt are mitigated.
- No anatomical risk factors have been identified.
- No further surgical intervention is required.

Residual hearing loss is not a contraindication to diving per se, although the deficit could worsen. Occupational hearing loss is a common work-related (compensable) injury in Australia. Diving is contra-indicated where there is active vestibular disturbance.

There are valid reasons to differentiate between occupational divers and recreational divers. The decision not to undertake further diving has less impact on recreational divers who do not rely on diving for their livelihood. Occupational divers are dependent on diving for income, and are usually keen to return to diving where possible. Occupational divers also have different risk profiles to recreational divers. They are task-focused, have less control over the diving conditions and frequency, and their equipment is less conducive to ear clearing (e.g., full face masks). This may place them at greater risk of recurrence of IEBt. The overlap in symptomatology between recurrence of IEBt and vestibular DCI may also cause future diagnostic dilemmas. Occupational divers may be under greater incentive to return to diving after an upper respiratory tract infection (URTI), adding to risk of IEBt recurrence. An additional recommendation to avoid diving for at least two weeks following an URTI has been proposed.<sup>23</sup>

Returning to diving for occupational divers requires negotiation and risk acceptance by all stakeholders. Despite medical clearance to return to diving, employers may not be prepared to accept the risk.

### Recommendations from this literature review

- All divers require a baseline assessment of hearing prior to commencing diving (compulsory with diving medicals AS4005.1 and AS/NZS 2299).<sup>61,62</sup> Where IEBt is suspected, reference to the baseline audiometry can identify and monitor cochlea injury, thereby assisting diagnosis and management.
- Audiometry should be repeated at each subsequent health assessment because of the risk of subclinical IEBt and other aural pathology in divers, and to allow an up-to-date reference point for new injuries.
- All divers require education regarding the rationale and technique for equalising their middle ear effectively and regularly during their dive. The potential impact of ear clearing difficulties should be emphasised and, if persistent, dives terminated and medical advice sought.
- Investigations that will assist to identify IEBt include PTA (air conduction/bone conduction) with comparison to previous audiograms, the fistula test, an ENG, and combining PTA or ENG with pneumatic manoeuvres.
- A diagnosis of IEBt and particularly PLF is achieved by a thorough history assessing the risk of the dive profile, rapidity of onset of vestibular symptoms or hearing loss, problems with equalising, timing of symptoms in relation to ear equalisation manoeuvres or other manoeuvres, such as straining, coughing or lifting, and the use of the fistula test.
- If IEDCS cannot be excluded, a trial of hyperbaric oxygen treatment does not appear to worsen IEBt (case data) or its prognosis for recovery, provided the diver can equalise their ears. Careful and gentle ear clearing is advised.
- Conservative management is first-line care for almost all suspected IEBt, except with profound global hearing loss and/or major vestibular symptoms. This allows for healing of mild IEBt (inner ear haemorrhage, membrane tears) and MEBt and the development of some central compensatory mechanisms.
- Use of steroids has little supporting evidence in the management of IEBt, but is commonly used.
- Indications for explorative tympanotomy and closure of a confirmed or suspected PLF include severe or persisting vestibular symptoms or hearing loss, and/or deterioration of these symptoms, or lack of improvement in these symptoms over 10 days.
- HRCT of the temporal bones may identify predisposing anatomical abnormalities and exclude any congenital propensity to IEBt. This should be performed before clearing an occupational diver for return to diving.
- Returning to diving as early as one to three months post injury may be considered after stabilisation of hearing, absence of disequilibrium, normalisation of all vestibular symptoms, exclusion of anatomical predisposition and education to prevent MEBt.

Occupational divers may return to full diving capacity after PLF repair (provided the above criteria are satisfied). This would require extensive counselling as to the need

and techniques for equalising, and future impacts of any concurrent URTI. It would also require risk acceptance by the employer.

### Conclusion

IEBt constitutes pathology of the inner ear induced by failure to equalise pressure changes secondary to diving. IEBt produces tinnitus, vertigo and impaired hearing and balance. Diagnosis requires a combination of clinical suspicion, dive risk assessment, clinical assessment and investigation, including reference to baseline PTA. There is no definitive clinical algorithm or single diagnostic test that objectively confirms IEBt. Diagnosis of the PLF subset of IEBt is supported by positive fistula test and direct observation at tympanotomy. Conservative management is recommended first-line therapy for IEBt, with exploratory surgery indicated for severe or persisting vestibular symptoms or hearing loss, and/or deterioration of these symptoms, or lack of improvement in these symptoms over 10 days.

Provided that the SNHL is stable and not severe (measured by PTA), and there is no sign of uncompensated vestibular involvement (via ENG) and HRCT has excluded anatomical predilection to IEBt, it may be possible for divers to resume diving. This requires careful counselling of the diver regarding ear clearing, risk acceptance by all stakeholders, and detailed, informed consent before actioning. There is need to establish a data registry and long-term follow up of divers returning to their occupation post IEBt in order to gain a better understanding of their functional recovery, risk of recurrence or morbidity.

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Elizabeth J Elliott, David R Smart  
Faculty of Health Sciences, University of Tasmania, and Medical  
Co-directors, Department of Diving and Hyperbaric Medicine,  
Royal Hobart Hospital, Hobart, Tasmania

**Address for correspondence:**

Dr Elizabeth Elliott

PO Box 744, North Hobart

Tasmania 7002, Australia

**Phone:** +61-(0)414-344744

**Fax:** +61-(0)3-6222-7268

**E-mail:** <elizabeth.elliott@dhhs.tas.gov.au>

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# Controversies in hyperbaric medicine – Réunion2013

## Medical devices and procedures in the hyperbaric chamber

Jacek Kot

### Abstract

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The aim of this paper is to present current controversies concerning the safety of medical devices and procedures under pressure in a hyperbaric chamber including: defibrillation in a multiplace chamber; implantable devices during hyperbaric oxygen treatment (HBOT) and the results of a recent European questionnaire on medical devices used inside hyperbaric chambers. Early electrical defibrillation is the only effective therapy for cardiac arrest caused by ventricular fibrillation or pulseless ventricular tachycardia. The procedure of defibrillation under hyperbaric conditions is inherently dangerous owing to the risk of fire, but it can be conducted safely if certain precautions are taken. Recently, new defibrillators have been introduced for hyperbaric medicine, which makes the procedure easier technically, but it must be noted that sparks and fire have been observed during defibrillation, even under normobaric conditions. Therefore delivery of defibrillation shock in a hyperbaric environment must still be perceived as a hazardous procedure. Implantable devices are being seen with increasing frequency in patients referred for HBOT. These devices create a risk of malfunction when exposed to hyperbaric conditions. Some manufacturers support patients and medical practitioners with information on how their devices behave under increased pressure, but in some cases an individual risk-benefit analysis should be conducted on the patient and the specific implanted device, taking into consideration the patient's clinical condition, the indication for HBOT and the capability of the HBOT facility for monitoring and intervention in the chamber. The results of the recent survey on use of medical devices inside European hyperbaric chambers are also presented. A wide range of non-CE-certified equipment is used in European chambers.

### Key words

Hyperbaric medicine, safety, equipment, implantable devices, patient monitoring, ventilators, resuscitation, review article

### Introduction

The aim of this paper is to present current controversies concerning the safety of medical devices and procedures inside a hyperbaric chamber. The presentation has been divided into three sections:

- defibrillation inside a multiplace hyperbaric chamber;
- implantable devices during hyperbaric oxygen treatment (HBOT) and
- results of a European questionnaire on medical devices used inside hyperbaric chambers.

### Defibrillation inside a hyperbaric chamber

Electrical defibrillation is well established as the only effective therapy for cardiac arrest caused by ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT). The scientific evidence to support early defibrillation is overwhelming; the delay from collapse to delivery of the first shock is the single most important determinant of survival. The American Heart Association (AHA) has given a strong recommendation for immediate defibrillation as the treatment of choice for VF of short duration, as seen in

witnessed cardiac arrest. The goal for early defibrillation in the hospital and ambulatory clinics is for the shock to be delivered within the first few minutes after the victim's collapse.<sup>1</sup>

The need for rapid defibrillation should mean that every medical hyperbaric facility treating patients with life-threatening conditions should have the potential to perform defibrillation while inside the chamber. However, the need for defibrillation during HBOT is rare. In our centre, we conduct almost 7,000 person sessions per year including intensive care (mostly with septic shock) and emergency patients (mostly with carbon monoxide (CO) poisoning) – a total of more than 120,000 person exposures during the past 20 years. Excluding one case of a patient with CO poisoning being resuscitated for VF during compression, with spontaneous restoration of a regular heart rhythm after a few minutes breathing 100% oxygen at 253 kPa, we have seen only a few fatal cases inside the chamber or immediately following HBOT when there was a need for defibrillation. Such a low incidence of cardiopulmonary resuscitation inside the hyperbaric chamber is probably because of careful medical examination of critically ill patients by a hyperbaric physician before each HBO session,

monitoring of ventilation and circulation while at pressure and significant hyperoxygenation during the session, which prevents cardiac insults occurring under hyperbaric conditions. On the other hand, hyperoxygenation extends the length of circulatory arrest that can be tolerated, giving additional time for accelerated decompression and out-of-chamber defibrillation. However, there are still clinical situations where having the means for defibrillation inside the chamber is highly recommended, for example, long recompression tables, including saturation exposures, when fast decompression would be deleterious for the patient and for medical attendants.

It must be remembered that the procedure of defibrillation is inherently dangerous owing to the risk of fire caused by electrical discharges and voltaic arcing which may be generated between the paddles, high flow of current in older types of defibrillators and the risk of enhanced combustion from high local oxygen concentrations from leakage of oxygen from the patient's respiratory circuit. While it is an absolute contraindication to conduct defibrillation in the pure oxygen atmosphere of a monoplace chamber, the procedure for multiplace chamber defibrillation has been described previously.<sup>2</sup> Important requirements to be fulfilled before discharge include:

- the chamber is compressed with air and the oxygen fraction is kept below 21.5 vol%;
- large surface adhesive plates are attached to the patient's chest and the area around the plates is kept free from flammable materials;
- the standard defibrillator (including switches) is located outside the chamber and transmission cables pass through the chamber wall to the chest pads;
- additional personnel – an external defibrillator operator who controls the discharge unit located outside the chamber.

Quite recently two defibrillators have been introduced into hyperbaric medicine, which could be used inside the chamber (including the discharge unit). This makes the procedure of in-chamber defibrillation much easier. The Physiocontrol LifePak 1000 has been approved for hyperbaric use by the classification body Germanisher Lloyd in close cooperation with the Biomedical Engineering Department of the Karolinska Institute in Stockholm, Sweden (Kronlund P, Lind F, personal communication, 2013). The other is the Corplus3 (GS Elektromed, Geräte G. Stemple GmbH, Germany). The former device is a popular automated external defibrillator (AED) well known to emergency medical service (EMS) teams as well as for in-hospital services. The latter device is a combined wireless monitor of physiological parameters with embedded defibrillator to be used inside a hyperbaric chamber. Both devices have been approved by Germanisher Lloyd and, among other aspects, the safety approval for these devices is based on the assumption that using a lower current in bi-phasic impulse mode during defibrillation with self-adhesive pads does

not create a risk of sparking. Indeed, as reported in 2010, "*there were no case reports of fires caused by sparking when shocks were delivered using adhesive pads*".<sup>3</sup> The same statement has appeared in several other national guidelines, for example, those published by the Australian, New Zealand and United Kingdom Resuscitation Councils.

However, sparking during defibrillation even with adhesive pads has been observed several times and reported to the FDA Manufacturer and User Facility Device Experience (MAUDE) database.<sup>4</sup> In this database, there is also a description of a recent event (MDRFOI ID = 2922391, dated 12 October 2012), when a fire was ignited during defibrillation, which burned the patient's side in the EMS ambulance. The defibrillation shock of 200 joules was delivered for VT. There was no explicit statement that the impulse was delivered through the self-adhesive pads but, in the description of the event, there is information that the AutoPulse Non-invasive Cardiac Pump was used during resuscitation and transportation of the patient. The standard procedure in such cases is to attach self-adhesive pads for defibrillation, so one may assume that this event happened with such pads attached. If so, this event shows that fire can start during defibrillation, at least in specific circumstances.

In conclusion, having a modern, stand-alone defibrillator inside a hyperbaric chamber makes defibrillation under hyperbaric condition technically easier than ever, but such a procedure still presents a risk of fire. Therefore, every precaution must be taken while delivering a defibrillation shock in a hyperbaric environment.

### Implantable devices

Implantable devices are being seen with increasing frequency in patients referred for HBOT. A review of such devices has been published previously and updated by direct contact with manufacturers to determine maximum allowable pressure for specific devices (from 151–709 kPa).<sup>5,6</sup> While all implantable devices that are exposed to the ISO-compatible ETO-standard sterilization process are exposed to pressures up to 253 kPa, this testing by manufacturers is not giving full legal standing against health providers for increased risk of damage by overpressure. Fortunately, there is direct support from at least one large manufacturer of implantable cardioverter defibrillators (ICD), which has provided a statement setting out the correct application and pressure tolerances of their pacemakers and implantable defibrillators both in diving activities and under hyperbaric conditions.<sup>7,8</sup> According to this statement, it is assumed that devices produced by this manufacturer will operate safely up to 253 kPa, but that performance may change at pressures in excess of 303 kPa (with return to normal operation after decompression). The device chassis will start to deform significantly only at pressures close to 507 kPa. For other implantable devices, for example, brain stimulators or implantable infusion systems, the pressure limitations are stricter, limiting maximum allowable pressure to only 203

kPa.<sup>9</sup> Interestingly, when looking at national guidelines from the same manufacturer it appears that the recommendations vary from region to region. An example is an implantable infusion system with either a programmable pump with a catheter access port or non-programmable pump without a catheter access port. The manufacturer advises a pressure limit to 203 kPa in one region of the world and 233 kPa or 303 kPa in others!<sup>10</sup>

In the FDA MAUDE database there is at least one incident reported of an implantable pump inside a hyperbaric chamber (MDRFOI ID = 765802, dated 10 October 2005).<sup>4</sup> In this event, during a 2-hour session at a pressure of 152 kPa, a “bang noise at the implant side” was reported by the patient, with malfunction of delivery of an analgesic. X-ray confirmed dislocation of the catheter from the infusion pump. The units of pressure reported in this event are not documented precisely, but were probably gauge pressure, which is equivalent to 253 kPa.

Yet another problem with implantable devices is the potential for external electrical arcing during delivery of a shock. Such a risk has been raised by one manufacturer, leading to a statement that, although there has been no reported incidence of ICD shock-triggered ignition, it may be advisable, pending further study to the contrary, to disable the defibrillation mode while patients are undergoing hyperbaric treatments.<sup>8</sup> There are reports that malfunctioning implanted devices may lead to skin burns over automated ICD sites or even transmitting the shock to external rescuers performing external chest compression.<sup>11,12</sup> Theoretically there is a possibility of external arcing during shock delivery from an implanted defibrillator, but this would need sufficient current leakage from the intended path and an air gap to allow an alternate pathway (to ground) that would complete the circuit. Experiments on dogs assessed worst-case scenarios for external leakage of current during internal defibrillation.<sup>13</sup> These demonstrated energy outflow (estimated to be in the order of 0.4 W when defibrillating with 30 joules, which resulted in mean 0.0184 amp of current with 19.3 V of voltage) which would be significantly lower than that currently allowed by the NFPA 2010 for battery-operated devices for use in hyperbaric conditions (not exceeding 12V and 48W).<sup>13,14</sup>

In conclusion, when faced with a patient referred for HBOT, who has an implanted device, it is recommended that the manufacturer is contacted for confirmation that this specific device can be safely exposed to the treatment pressure and time. Alternatively, one can use any existing reference in the literature looking for specific data on that type of device. Regardless of this, an individual risk-benefit analysis should be conducted on the patient, taking into consideration his/her clinical condition, the indication for HBOT and the capability of the HBOT facility for monitoring and intervention in the chamber. In any such case, both patient and physician should sign a consent form accepting increased risk for either malfunction or damage to the device. It is highly advisable

to constantly monitor implanted devices during every HBO session and to report any untoward events or malfunction to either national or international databases.

### Survey of medical devices inside hyperbaric chambers in the European Union

In Europe, there is a Medical Device Directive (MDD 93/42) that defines a medical device as “*any instrument (...), including the software necessary for its proper application intended by the manufacturer to be used for human beings for the purpose of: (...) treatment or alleviation of disease, (...) treatment or alleviation of or compensation for an injury (...)*”.<sup>15</sup> According to this definition, the hyperbaric chamber itself and all its equipment should be approved for hyperbaric conditions and this confirmed by appropriate CE certification. Unfortunately the list of medical devices that are CE-marked for use in hyperbaric conditions is very short. At present, it consists of only two ventilators (Italian Siaretron 1000 Iper [60 VF] and the Maquet Servo-i HBO), one syringe pump (Pilot Hyperbaric, Fresenius Vial S.A.) and two systems for internal monitoring (Haux HMMS, Germany, and Corplus3, Germany). For any other device that is introduced into hyperbaric conditions, a formal risk assessment must be conducted, but the user still takes the full responsibility for any malfunction of the device that is exposed to environmental conditions other than those specified in the operating manual for that device.

Because the list of CE-marked medical devices used for intensive care during a hyperbaric session is so short, it is well known that many European medical hyperbaric facilities are using different unlisted devices inside hyperbaric chambers. In order to obtain a clearer picture of these practices, a survey on the use of medical devices inside hyperbaric chambers in Europe was conducted in 2013.

The list of European medical hyperbaric facilities included in the OXYNET registry <[www.OXYNET.org](http://www.OXYNET.org)> was used as a contact list. The OXYNET database is administered by the European Committee for Hyperbaric Medicine, <[www.ECHM.org](http://www.ECHM.org)>. At the time of the survey (May 2013), there were 246 facilities included in the database. There was no e-mail address for 30, so 216 e-mails were sent with the questionnaire. Fifty-two e-mails were undeliverable and of the remaining 164 e-mails, 49 responses were received (only 30% of the e-mails successfully delivered). At the same time, the same survey was conducted in the USA giving a similar response rate (24%, 46 responses out of 192 centres; James Bell, personal communication, 2013). Out of the 49 centres, that completed the survey, 36 centres (73%) used only multiplace chambers; six centres used only monoplace chambers and seven centres were using both mono- and multiplace chambers.

It is interesting that out of 49 facilities that responded, only 33 reported that they were using any medical equipment inside the hyperbaric chamber. The remaining 16 centres,

including 11 centres with multiplace chambers and five with monoplace chambers, do not use any medical devices in their chambers. Of the 33 centres using medical devices inside the chamber, only six use solely CE-marked devices for ventilation, monitoring and infusions. The other 27 centres rely on some sort of risk assessment being conducted by the external company alone (11 centres), internally within the institution (five centres) or by both external and internal entities (eight centres).

#### MECHANICAL VENTILATION

Only 14 centres reported that they solely used CE-marked ventilators (Siaretron 1000 Iper, Maquet Servo i-HBO or the now obsolete Draeger Hyperlog). Other centres are using: Servo 900C modified for hyperbaric conditions; Evita 4; Draeger Oxylog; Penlon Oxford MKII; Brian Avian transport ventilator (for backup only) or Newport HT50 transport ventilator (for backup only).

#### PATIENT MONITORING

Among the 33 centres, there were a variety of different solutions for physiological monitoring other than the CE-certified devices (Haux HMMS and Corplus3). These included: the Kontron system (monitor outside, modules inside); Siemens Sirecust Monitor 620; Datex/Ohmeda (monitor outside, modules inside); GE Solaris 800i (with nitrogen flush); GE PDMS transmitting units; Propaq Encore; 106 EL/102; Marquette Tramscope or Draeger Infinity Delta PMS. Other medical devices used for monitoring of patients during HBOT included: Kontron TcpO<sub>2</sub>; Radiometer TINA TcpO<sub>2</sub>; Wright's spirometer; Magtrack respiration monitor; Life Pack 20 (discharge unit outside, defibrillation pads inside); Physiocontrol Lifepack 1000 defibrillator (certified by Germanischer Lloyd); Heine Minilux otoscope; Abbott Optimum Exceed or Accu-Check Performer glucometers and tonometer Omron M4-1 Intellisense (with manual start). Infusion devices included: Braun Perfusor Secura; Argus syringe pump 600 series; modified Alaris SE and IVAC P1000 peristaltic pumps; IVAC/Wellmed syringe P3000 and Terumo TE371 TIVA syringe.

With regard to policies for implantable devices, 26 centres had an explicit policy, including 13 centres requesting external approval (from the manufacturer). One centre has a policy for deep brain stimulators (up to 203 kPa).

Amongst the free text comments sent, there were many for an appropriate European organisation to officially tackle the problem of the lack of medical equipment that is CE marked for use in the hyperbaric environment, as well as the need for a shared repository of information about equipment used for medical purposes in different hyperbaric centres.

In conclusion, it is clear that the list of medical devices to be used inside hyperbaric chambers and approved by European regulations is deficient and does not fulfil the needs of many

European hyperbaric centres. In this situation, medical directors take the responsibility of using non-CE marked medical devices, either based on a formal risk assessment (external or internal) or simply based on their personal experience and general knowledge. It is highly advisable to convince manufacturers of the need for testing their devices for hyperbaric conditions with appropriate CE marking for the European market. In the meantime, any risk assessment should be published or otherwise made available for the guidance of other hyperbaric facilities. This journal, *Diving and Hyperbaric Medicine*, is an appropriate vehicle for the publication of such technological reports.

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*Jacek Kot is an Assistant Professor at the National Centre for Hyperbaric Medicine in Gdynia, Medical University of Gdansk, Poland*

#### Address for correspondence:

*Jacek Kot, MD, PhD*

*National Centre for Hyperbaric Medicine*

*Institute of Maritime and Tropical Medicine in Gdynia*

*Medical University of Gdansk*

*Powstania Styczniowego 9B*

*81-519 Gdynia, Poland*

**Phone:** +48-(0)58-6225163

**Fax:** +48-(0)58-6222789

**E-mail:** <jkot@gumed.edu.pl>

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### Appearance of gas collections after scuba diving death: a computed tomography study in a porcine model

Laurent PE, Coulange M, Bartoli C, Boussuges A, Rostain JC, Luciano M, Cohen F, Rolland PH, Mancini J, Piercecchi MD, Vidal V, Gorincour G

**Introduction:** Postmortem computed tomography can easily demonstrate gas collections after diving accidents. Thus, it is often used to support the diagnosis of air embolism secondary to barotrauma. However, many other phenomena (putrefaction, resuscitation maneuvers, and postmortem tissue offgassing) can also cause postmortem gas effusions and lead to a wrong diagnosis of barotrauma.

**Objectives:** The aim of this study is to determine topography and time of onset of postmortem gas collections respectively due to putrefaction, resuscitation maneuvers, and tissue offgassing.

**Methods:** A controlled experimental study was conducted on nine pigs. Three groups of three pigs were studied postmortem by CT from H0 to H24: one control group of nonresuscitated nondivers, one group of divers exposed pre-mortem to an absolute maximal pressure of 5 b for 16 min followed by decompression procedures, and one group of nondivers resuscitated by manual ventilation and thoracic compression for 20 min. The study of intravascular gas was conducted using CT scan and correlated with the results of the autopsy.

**Results:** The CT scan reveals that, starting 3 h after death, a substantial amount of gas is observed in the venous and arterial systems in the group of divers. Arterial gas appears 24 h after death for the resuscitated group and is absent for the first 24 h for the control group. Concerning the putrefaction gas, this provokes intravenous and portal gas collections starting 6 h after death. Subcutaneous emphysema was observed in two of the three animals from the resuscitated group, corresponding to the thoracic compression areas.

**Conclusion:** In fatal scuba diving accidents, offgassing appears early (starting from the first hour after death) in the venous system then spreads to the arterial system after about 3 h. The presence of intra-arterial gas is therefore not specific to barotrauma. To affirm a death by barotrauma followed by a gas embolism, a postmortem scanner should be conducted very early. Subcutaneous emphysema should not be mistaken as diagnostic criteria of barotrauma because it can be caused by the resuscitation maneuvers.

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#### Key words

Scuba diving, deaths, radiological imaging, animal model, reprinted from

# Unestablished indications for hyperbaric oxygen therapy

Simon J Mitchell and Michael H Bennett

## Abstract

(Mitchell SJ, Bennett MH. Unestablished indications for hyperbaric oxygen therapy. *Diving and Hyperbaric Medicine*. 2014 December;44(4):228-234.)

Unestablished indications are conditions in which systematic clinical use of hyperbaric oxygen treatment (HBOT) is not supported by adequate proof of benefit. HBOT is vulnerable to use in many such conditions for various reasons, perhaps the most important being that a placebo or participation effect may create an impression of efficacy. The systematic use of HBOT in unestablished indications raises ethical concerns about provision of misleading information, giving false hope, and taking payment for therapy of doubtful benefit. Any practice perceived as unethical or unscientific has the potential to draw the wider field into disrepute. Of substantial contemporary relevance is the use of HBOT in treatment of various forms of chronic brain injury; in particular, cerebral palsy in children and the sequelae of mild traumatic brain injury in adults. There are now multiple, randomised, blinded, sham-controlled trials of HBOT in both indications. None of these studies showed benefit of HBOT when compared to sham control, though the sham and HBOT groups often both improved, indicating that a placebo or participation effect influenced outcomes. These results almost certainly explain those of open-label trials (lacking sham controls) in which HBOT frequently seems beneficial. Advocates for HBOT in chronic brain injury claim that the sham treatments (usually 1.3 ATA\* pressure exposure whilst air breathing) in the blinded trials are actually active treatments; however, the same dose of oxygen can be achieved at 1 ATA breathing 27% oxygen. To counter this argument, advocates also claim that the extra 0.3 ATA of pressure is somehow independently beneficial, but this notion has limited biological plausibility and there is little supporting evidence. Chronic brain injuries remain unestablished indications at this time and, in our opinion, should not be systematically treated with HBOT.

## Key words

Hyperbaric oxygen therapy, hyperbaric research, trauma and stress, central nervous system, children, evidence, ethics, review article

## Introduction

Hyperbaric oxygen treatment (HBOT) is a therapeutic modality that has long struggled for credibility within 'mainstream' medicine. In large part, this has been due to a lack of high-quality evidence to support HBOT in its various indications. Thanks to the efforts of practitioners and researchers who recognise the centrality of evidence-based practice for credibility, the last two decades have seen maturation of the evidence base for a limited number of indications, and a concomitant improvement in perceptions of HBOT amongst many of our 'mainstream' colleagues. A tangible manifestation of this was the appearance in 2011 of the first chapter on hyperbaric and diving medicine in an iconic general medicine textbook.<sup>1</sup>

Unfortunately, advocacy for HBOT in indications that are either unsupported by an appropriate evidence base, or that have largely been disproved, threatens the credibility of the field. In particular there is growing controversy around the use of HBOT in treatment of various forms of chronic brain injury and we will return to this specific subject later. This prompted the convening of a session on controversies in hyperbaric medicine at the 2013 tripartite meeting of the South Pacific Underwater Medicine Society (SPUMS), the European Undersea and Baromedical Society (EUBS), and the Southern African Underwater and Hyperbaric

Medical Association (SAUHMA). One paper, intended as an overview of the issue of 'unestablished indications', is summarised here.

We begin with a brief mention of relevant historical events in the field, and we define an 'unestablished indication' in the modern context. We comment on why HBOT is vulnerable to use in unestablished indications and enumerate the reasons we consider deviation from rational, evidence-based practice to be harmful to the field. Finally, we will discuss cerebral palsy and the sequelae of mild traumatic brain injury (mTBI) as examples of unestablished indications in which the arguments for and against HBOT exemplify important principles.

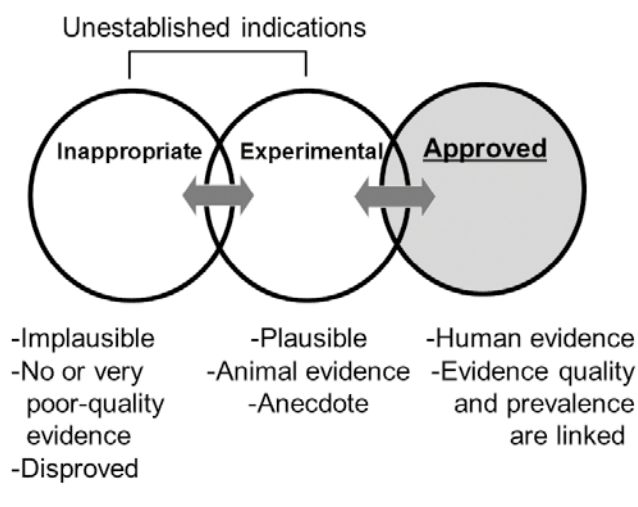
## What is an unestablished indication?

The history of hyperbaric medicine dates back centuries to the 'hyperbaric spas' or 'air baths' of Europe; an era in which exposure to mildly elevated pressures of air was advocated for treatment of a wide variety of ills.<sup>2</sup> This tradition of intuitive and speculative practice was continued into the 20th century with arguably the most conspicuous example set by

\* **Footnote:** 1 ATA = 101.3 kPa. Since all the trials described in this article report the pressure used in ATA, these units rather than kPa will be used here.

**Figure 1**

Three categories of potential indications for HBOT (see text for explanation)



an anesthesiologist in the United States, Orval Cunningham, who ran a practice based on exposure of patients to hyperbaric air, which he used to ‘treat’ a variety of disease processes. The bizarre zenith of his activities came with the construction of a large and luxuriously appointed residential chamber perhaps best described as a ‘hyperbaric hotel’. While quaint and perhaps even funny, the Cunningham saga provided an early example of how unconventional practice can attract the derision of conventional colleagues. Cunningham himself was deregistered following repeated refusal to provide any evidence to back up his claims of benefit from hyperbaric treatment and his residential chamber was closed. After his death, the facility was dismantled for scrap, and these events were announced to the medical world in a *JAMA* news column under the banner headline “Useless tank to become useful tanks”.<sup>3</sup>

This article, among other things, stated:  
 “The tank here referred to was originally constructed some 13 years ago by the late Dr Orval J Cunningham of Kansas City, Mo, for the purpose of instituting his preposterous pressure treatment for diabetes, pernicious anemia, and carcinoma.”<sup>3</sup>

In relation to the project’s funding by a wealthy industrialist the author asked:

“Why do people of great wealth who are unacquainted with scientific fact and apparently unwilling to consult scientific authority so frequently support strange notions in the field of medical care?”<sup>3</sup>

We will return to the issue of harm to the field later, but it is obvious that this characterisation of hyperbaric therapy as “preposterous” and a “strange notion” in one of the world’s most influential medical journals could only have been extremely damaging to the efforts of anyone trying to advance the modality in a rational manner.

Thankfully there are few practitioners as overtly unconventional as Cunningham in the present era, though there is little doubt that unestablished indications are being systematically treated with HBOT. This, of course, begs the question ‘what defines an unestablished indication?’ We categorise the potential indications for HBOT into three groups (Figure 1), each of which is characterised by several descriptors.

‘Approved indications’ are supported by human evidence of efficacy, and the quality of the supporting evidence should reflect the prevalence of the disease in question. Thus, not all approved indications require support by high-quality, large randomised trials. Sporadic, rare, and catastrophic diseases such as necrotising fasciitis are a good example. Such conditions are difficult to study in randomised trials, and the evidence quality bar may consequently be set lower than would be the case for a prevalent indication like ‘problem’ wounds.

An obvious point of contention in application of this model is who determines whether an appropriate standard of evidence has been met for an indication to be ‘approved’? The Undersea and Hyperbaric Medical Society (UHMS), an independent and responsible scientific society, has approached the problem by convening a standing committee of experts who periodically review the available evidence and make determinations on the status of new or existing ‘approved’ indications.<sup>4</sup> This process does not eliminate potential for contentious decisions, but it seems a pragmatic solution to a difficult problem. The double-ended arrows in Figure 1 are intended to indicate that this process of regular review ensures no indication is immutably categorized in the face of emerging evidence. Thus, for example, an ‘experimental’ indication can become ‘approved’ if sufficient evidence emerges to justify this.

‘Experimental indications’ are typically those in which there is a plausible biological rationale for application of HBOT and perhaps some supportive animal evidence or human anecdote. However, there is insufficient human evidence to achieve ‘approval’.

‘Inappropriate indications’ are typically those with little face validity or biological rationale, and little or no supporting evidence. This categorisation would also be applied to well-researched indications which may have once seemed plausible, but in which the overwhelming weight of available evidence is unsupportive.

As indicated in Figure 1, the ‘experimental’ and ‘inappropriate’ indications collectively constitute what we refer to as ‘unestablished indications’. Such indications may, of course, continue to be studied if it is deemed justified. However, we strongly believe that HBOT should not be represented as a proven treatment in these conditions. Nor should medical

practitioners systematically treat unestablished indications with HBOT outside the context of research, or (in our opinion) receive payment for such treatment.

### **Why is HBOT vulnerable to use in unestablished indications?**

There are a variety of reasons why HBOT is frequently utilised in treatment of unestablished indications. Firstly, the status of oxygen as a drug, and the regulations around who may administer it are uncertain or ambiguous in some countries. It is not uncommon to find so-called 'hyperbaric medicine units' owned by members of non-medical craft groups (like former commercial divers) who see nothing wrong with applying their recompression chamber operational experience to the medical field. Such people may enter into relationships of convenience with local doctors for the purposes of billing state-sponsored funding agencies, but the doctors sometimes know even less about hyperbaric medicine than the chamber operators. Not surprisingly, these scenarios often result in particularly bizarre claims of efficacy. A relatively recent example from New Zealand resulted in a full-page newspaper advertisement claiming efficacy for HBOT in around 100 medical conditions, many of which were spelled incorrectly.

Secondly, oxygen is easily marketed to the general public as being essential for life. In this paradigm, HBOT is portrayed simplistically as 'more of a good thing'. The mainstream public are vulnerable to such claims and levels of knowledge about these matters are poor. A recent brochure extolling the virtues of an oxygen café in Brisbane, Australia claimed that oxygen levels in the atmosphere of a typical large city hover around 12–16%, and that this is even lower in buildings. One of the present authors was contacted by a television station researcher to check the veracity of the claim!

Thirdly, the application of HBOT is technical and dramatic. It usually takes place in a positive, supportive and affirming clinical environment; and it requires considerable commitment from highly motivated patients who are invariably hopeful of a good effect. This is a perfect collection of circumstances for the emergence of a substantial placebo or participation effect and under such circumstances it is not surprising that HBOT often appears to work. This is particularly so for problems where outcomes are subjective, amenable to psychological manipulation, or where the results of confirmatory investigations can be easily misinterpreted. Under these circumstances it is not surprising that well-meaning practitioners may earnestly believe that they are achieving good results for their patients. Perhaps not surprisingly, there is a substantial body of emerging evidence that a placebo effect might be responsible for apparent improvement in particular unestablished indications and we will return to this issue later in consideration of chronic brain injuries.

Finally, desperate patients with chronic or progressive

problems are frequently willing to 'try anything', and it is not difficult to convince such patients to try HBOT. This gives rise to several of the ethical concerns we have about systematic and remunerated treatment of unestablished indications.

### **What are the concerns about treatment of unestablished indications for HBOT?**

We have two major concerns with the treatment of unestablished indications using HBOT. The first relates to the ethics of unintentional (or intentional) exploitation of vulnerable patients that we alluded to in the final point above. Given the (at best) uncertain benefit from HBOT in treatment of unestablished indications, any insinuation of benefit is potentially misleading. Similarly, the acceptance of payment for unproven therapy when the patient has unrealistic or unfounded expectations is widely regarded as unethical. For example, in a standards document, the College of Physicians and Surgeons of Saskatchewan specifically states: *"It is unethical to engage in or to aid or abet in treatment which has no scientific basis, may be dangerous, may deceive the patient by giving false hope, or which may cause the patient to delay in seeking proper care until his or her condition becomes irreversible."*<sup>5</sup>

The ethics of exposing patients to a therapy with risks when the benefit is unknown or even unlikely are highly questionable.

The second concern relates to the perception that treatment of unestablished indications creates among our mainstream medical colleagues. The use of HBOT in indications where there is little biological rationale let alone convincing human evidence creates the very real risk that hyperbaric physicians come to be seen as 'alternative medicine' practitioners (or worse). The 'Cunningham experience' described earlier in this article exemplified the derision that indiscriminate non-evidence-based practice attracts, and there have been more recent examples.

Experienced hyperbaric physicians will remember the 1987 Gabb and Robin article in *Chest* which famously labelled HBOT *"a therapy in search of diseases"*.<sup>6</sup> In support of their thesis, these authors cited a typical long list of indications claimed by enthusiastic advocates (similar to the one that we earlier described from a New Zealand newspaper), and predictably proclaimed that *"the broad range of conditions speaks for itself"*.

In 2013, the Federal Drug Administration became concerned enough about claims relating to HBOT in unestablished indications that it saw fit to issue a communication entitled *"Hyperbaric oxygen therapy: don't be misled."*<sup>7</sup> Although the communication was targeted against claims of efficacy in treating unestablished indications like autism, AIDS, cancer, stroke and depression rather than the approved indications, many readers will have neither grasped the distinction nor advanced beyond the pejorative title.



Thus, over the years, advocates for HBOT in unestablished indications have attracted ridicule in prominent journals like *JAMA* and *Chest*, and provoked admonishment from the FDA. This sort of negative attention from the mainstream medical community is damaging. We confidently predict that virtually all contemporary hyperbaric physicians will have struggled in the promotion of HBOT to at least some of their colleagues; usually based on the latter harbouring suspicions of the field as ‘alternative’ or lacking in evidence. Conspicuous promotion of HBOT for treatment of unestablished indications reinforces such prejudices, and almost certainly makes it less likely that patients who would benefit from treatment of approved indications will be referred.

### Contemporary issues

In recent years, the use of HBOT for the treatment of various forms of chronic neurological injury has been at the forefront of debate over unestablished indications. The evolution of the debate and the related research it has stimulated illuminates many of the issues we have discussed above and we provide a summary of it here. This account is, of necessity, relatively superficial and readers are encouraged to read the various references and judge relative merits for themselves.

The ‘HBOT in chronic brain injury debate’ first came to prominence in relation to cerebral palsy (CP) in children. Based on anecdotal observation of alleged improvement in behavioural and motor parameters, a number of enthusiasts promoted HBOT treatment for CP during the 1990s. The explanations offered for the alleged benefits focussed on unproven and vague concepts described in terms like the activation of ‘dormant’ or ‘idling’ neurons lying adjacent to areas of previous damage. There were also reports of putative improvements in cerebral blood flow patterns on SPECT scanning in association with HBOT treatments (vide infra).

The first definitive study was published in 2001.<sup>8</sup> This was a randomised, sham-controlled study of 111 children who received either 40 HBOT treatments at 1.75 ATA for one hour, or 40 air exposures at 1.3 ATA. Follow up was at three months after treatment. Both groups improved in respect of all outcome measures; most notably motor function, but there was no difference between the groups. The authors ascribed the general improvement to a placebo or participation effect, as did an independent scientific advisory committee.<sup>9</sup> This study created a storm of controversy which included emergence of the argument that 1.3 ATA of air is actually an active treatment. HBOT advocates opined that the study merely compared one active dose of oxygen with another, and that 1.3 ATA of air cannot be used as a sham control. We will address this issue in more detail later.

A second, randomised, sham-controlled study in CP patients was published in 2012.<sup>10</sup> In this case, 49 children were

**Table 1**

Key characteristics of the US military studies of HBOT for mild traumatic brain injury (mTBI); note that the Navy study was designed to factor out any effect of elevated inspired PO<sub>2</sub> in the control group; 1 ATA = 101.3 kPa

Service	Sessions	Control	HBOT
Army <sup>14</sup>	40	Air, 1.2 ATA	100% O <sub>2</sub> , 1.5 ATA
Air Force <sup>15</sup>	30	Air, 1.3 ATA	100% O <sub>2</sub> , 2.4 ATA
Navy <sup>16–18</sup>	40	10.5% O <sub>2</sub> , 2.0ATA	100% O <sub>2</sub> , 2.0 ATA 75% O <sub>2</sub> , 2.0 ATA

randomised to receive 40 HBOT treatments at 1.5 ATA or 40 exposures at 1.5 ATA breathing an inspired fraction of oxygen of 14% (equivalent inspired PO<sub>2</sub> to 21% oxygen at 1 ATA). The notable feature of this design is the elimination of any therapeutic effect of increased inspired PO<sub>2</sub> in the sham controls. Follow up was out to six months post treatment. There were no improvements in motor function scores, but this study did find significant improvement in a disability inventory in both groups, but (once again) no difference between the groups.

A second related area of recent interest has been the use of HBOT in chronic mild traumatic brain injury (mTBI). This has received much attention in the USA where large numbers of affected servicemen and women have returned from overseas conflicts. In 2013, Harch and colleagues published a series of 16 returned servicemen with sequelae of mTBI who all received 40 HBOT treatments at 1.5 ATA.<sup>11</sup> These patients exhibited improvements in various neuro-cognitive tests, and improvements in regional cerebral blood flow measured by SPECT scans. A second observational study in 63 mTBI patients treated similarly reported a common subjective perception of benefit but no clinically important changes on more objective neurocognitive testing.<sup>12</sup> A small subset of these patient had SPECT and CT angiographic studies which, as in the Harch series,<sup>11</sup> demonstrated an apparent improvement of regional cerebral blood flow after HBOT.

Several studies under the aegis of the US military (approximately corresponding to one per service) were subsequently undertaken in response to strong lobbying for systematic use of HBOT in veterans with mTBI. Whilst it is beyond the scope of this paper to describe these studies in detail, some trial characteristics are germane. The methodologies are summarised in an article by Weaver et al<sup>13</sup> and in the individual papers themselves.<sup>14–18</sup> All three were randomised, double blinded, sham-controlled trials, but with variation between studies in both treatment and sham protocols (Table 1).

The outcome measures in all studies included symptom inventories and neuropsychological testing. Results are reported at one month for the Army study; at one and six weeks for the Air Force Study, and immediate post-treatment,

one week and three months for the Navy study. The results for all three studies were presented at the Undersea and Hyperbaric Medical Society annual meeting in 2013, and have now been published.<sup>14-18</sup> None of the studies demonstrated any benefit for HBOT when compared to the sham protocol. In the Army and Air Force studies both sham and HBOT groups improved more than expected, but there was no difference between the groups. As was the case in the cerebral palsy trials previously discussed, the various authors considered a placebo effect most likely to account for parallel improvements in both sham (control) and HBOT patients.

These outcomes have disappointed enthusiasts.<sup>19</sup> It is notable the negative results contrast sharply with those reported from two recent studies of HBOT (versus standard care) in chronic stroke and mTBI that used an open-label, randomised design with no blinded sham hyperbaric exposures.<sup>20,21</sup> These studies demonstrated benefit when patients randomised to receive HBOT were compared to those randomised not to receive it. Not surprisingly, their authors have devoted considerable effort to explaining the different results in comparison to those of the US military mTBI studies.<sup>19-21</sup> They focus particularly on the contention that the US military sham exposures were actually effective treatments, and that this accounted for the equivalent results when sham and HBOT groups were compared.

The argument that a low-pressure air sham exposure is an effective treatment (and, therefore, an inappropriate control) is poorly supported. No-one has objectively demonstrated that exposure to 1.3 ATA of air is either neuroprotective or capable of resurrecting chronically 'idling' neurons in an injured brain. Moreover, there is no body of basic science evidence suggesting that small elevations in inspired pressures of oxygen and nitrogen (or small elevations of pressure itself) would be expected to exert a relevant therapeutic effect. 'Explanations' of the mechanisms underpinning the alleged efficacy of low-pressure air are rarely more sophisticated than the observation that there is a very modest elevation of the arterial  $PO_2$  when breathing air at 1.3 ATA, and that this has effects on completely different (usually pulmonary) pathologies in unrelated settings.<sup>20,21</sup> We have seen no cogent arguments to explain why this, of itself, would improve a chronic brain injury. Known effects of higher dose HBOT (such as stem cell mobilisation and effects on nitric oxide synthase) are often cited in the context of these debates, but to our knowledge such effects have never been demonstrated at these minimally elevated oxygen tensions.

One significant problem in relation to the 'active air sham' argument is that the same inspired  $PO_2$  achieved breathing air at 1.3 ATA could also be achieved by breathing 27% oxygen at 1 ATA, without the risks and costs of hyperbaric exposure. This begs an obvious question. If proponents of HBOT for chronic TBI believe that a 1.3 ATA air sham is actually an active treatment, why do they not simply treat

TBI patients with 27% oxygen at room pressure (or at least test this intervention; something they have all avoided doing to this point)?

A cynic might suggest this has much to do with the respective billing potential of the two modalities, but the response from advocates is that the putative neuro-rehabilitative effect of air at 1.3 ATA depends not only on the elevated arterial  $PO_2$  but also on the small elevation of ambient pressure.<sup>19</sup> To our knowledge, this argument is unsupported by any data demonstrating neuroprotective or neuro-rehabilitative benefit from exposure to pressure alone, and the notion lacks biological plausibility. Advocates attempt to address this concern by quoting the transduction of small pressure changes by certain cells in marine invertebrates,<sup>22</sup> and by citing pressure effects on mammalian neurons<sup>23</sup> revealed in studies whose outcome measures had nothing to do with neuro-rehabilitation and whose methods involved exposure to far greater pressures than 1.3 ATA.

This is sloppy citation and poor science, yet it is tenaciously promoted because the notion that pressure is a key contributor to the apparent benefit accrued from air at 1.3 ATA is crucial to two arguments advanced by those promoting HBOT for mTBI. The first, introduced above, is that even if air at 1.3 ATA is as effective as higher doses of HBOT, the hyperbaric approach cannot be replaced by breathing the equivalent  $PO_2$  (27%  $O_2$ ) at room pressure because the patient would not receive the alleged 'benefit' of pressure. The second is that the assumed benefit of pressure alone allows a circular argument which conveniently invalidates the randomised sham-controlled trials that show no benefit from HBOT in chronic brain injury,<sup>8,10,14-16</sup> including those designed to exclude any elevation of inspired  $PO_2$  in the sham groups.<sup>10,16</sup> Essentially, this argument holds that while proper blinding of controls cannot be achieved without some pressure exposure, any pressure increase means the controls are receiving an active treatment rather than an inactive sham. If one was to accept this argument, it would make sham-controlled trials virtually impossible to conduct – thus justifying the inferior non-blinded cross-over designs employed in recent studies of stroke and mild TBI as 'the best we can do'.<sup>20,21</sup>

Based on present evidence, we reject the argument that pressure per se is an active treatment in mTBI. We acknowledge the small increase in inspired  $PO_2$  to 0.27 ATA that occurs when air is breathed at 1.3 ATA, but we consider there is no convincing evidence for a neuro-rehabilitative effect of this dose of oxygen. On that background, we reiterate the fact that without exception, every randomised sham-controlled (blinded) study of HBOT in chronic brain injury to date has demonstrated equivalent improvement in patients receiving both HBOT and sham. Importantly, these include two studies designed to exclude any elevation of inspired  $PO_2$  in the sham groups.<sup>10,16</sup> The corollary is that unless the reader truly believes small increases in ambient pressure or the inspired  $PN_2$  alone can restore function to the

chronically injured human brain (notions that are currently unsupported by evidence), the appropriate interpretation of the sham-controlled study results is that there is no true therapeutic effect of HBOT in chronic brain injury. We are puzzled that advocates for HBOT in mTBI cite these studies as proof that the shams are not inert.<sup>21</sup>

Based on the available evidence and applying the principle of Occam's razor, we believe the most plausible explanation for the results of sham-controlled studies in chronic brain injury is a substantial placebo or participation effect. Given the demonstrated efficacy of cognitive rehabilitation therapy in TBI,<sup>24</sup> it seems very plausible that at least some sequelae of chronic brain injury may improve when highly motivated patients are given a dramatic prolonged course of treatment in a stimulating, positive, and optimistic clinical environment. It follows that we are not surprised by a recent non-blinded, non-randomised study in cerebral palsy comparing patients treated with: conventional methods; air at 1.3 ATA; HBOT at 1.5 ATA; and HBOT at 1.75 ATA, which found that all 'hyperbaric' groups (including air at 1.3 ATA) improved more than conventionally treated controls.<sup>25</sup> The authors stated: "*The very important difference observed in treated vs. controlled children can only be a genuine beneficial effect of HBO<sub>2</sub> therapy.*" It is extraordinary that the reviewers allowed this conclusion to be published because it is patently unjustifiable. Indeed, we believe that studies investigating HBOT in chronic brain injury that do not include a sham control group are deeply flawed.

Before concluding this discussion it is appropriate to mention SPECT scan detection of positive changes in regional cerebral blood flow (rCBF) following HBOT for mTBI.<sup>11,12,21</sup> These changes are sometimes cited as proof of an HBOT effect that cannot be due to placebo. In fact, it has been shown that rCBF as measured by SPECT may be influenced by cognitive therapy for mTBI and a placebo effect on SPECT results would therefore not be surprising.<sup>24</sup> Indeed, SPECT changes in response to placebo have been demonstrated,<sup>26,27</sup> with one analgesic study concluding: "*CBF changes appeared to correlate with the perception of pain or pain relief and not to the actual treatment administered per se.*"<sup>26</sup> The literature contains many high-quality references to placebo-induced changes in rCBF measured by other functional brain imaging techniques, and these are arguably relevant to SPECT. For example, functional magnetic resonance imaging has demonstrated that placebo analgesia causes decreased brain activity in pain-sensitive brain regions.<sup>28</sup> We accept that such results cannot be extrapolated directly to brain injury, but equally, we do not think that changes in SPECT scans following HBOT for mTBI constitute a convincing argument against placebo effects.

In the broader context of 'unestablished indications' the object lesson arising from the chronic brain injury saga is that there are some prevalent conditions in which HBOT may

appear to work when observational evidence is considered in isolation. Different conclusions may be drawn if sham-controlled studies are undertaken. Uncritical interpretations of observational data or data from trials without blinded sham controls<sup>25,29</sup> could result in massive expenditure on an expensive time-consuming 'therapy' that may, in fact, only work through a placebo effect. This should be of concern to all hyperbaric physicians who base their practice on evidence, and who are striving to build collaborations with sceptical mainstream colleagues.

We conclude this paper with an acknowledgement that research is ongoing in this area. Our commentary is based on the current state of the field, and we accept that evidence in respect of some of the 'unestablished indications' discussed here may evolve to a point where we revise our opinions in either of the directions indicated in Figure 1. In respect of chronic brain injuries, after multiple sham-controlled studies in which controls and HBOT subjects improved equally, any argument in support of HBOT now hinges on acceptance of the theory that the control intervention (air breathed at 1.2–1.3 ATA) is an active treatment with equivalent effects to higher doses of hyperbaric oxygen. We are unable to find either plausible explanations or substantive evidence to support this hypothesis. We accept that the matter has not been definitively studied and indeed, for this reason, we consider the current claims of therapeutic benefit across an extraordinary range of hyperbaric exposures to be premature, and most likely a misinterpretation of a placebo effect. As hyperbaric physicians there is nothing we would appreciate more than new, evidenced-based indications for HBOT, but we owe it to ourselves, the field and our patients not to actively promote unproven or ineffective therapy.

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*Associate Professor Simon Mitchell is a consultant anaesthetist and hyperbaric physician at Auckland City Hospital. He is the Head of the Department of Anaesthesiology at the University of Auckland, Auckland, New Zealand*

*Associate Professor Michael Bennett is a consultant anaesthetist and hyperbaric physician at Prince of Wales Hospital, Sydney, Australia*

#### Address for correspondence:

*Associate Professor Simon Mitchell  
Department of Anaesthesiology  
School of Medicine, University of Auckland  
Private Bag 92019  
Auckland, New Zealand*

**E-mail:** <sj.mitchell@auckland.ac.nz>

# Hyperbaric medicine and the placebo effect

Michael H Bennett

## Abstract

(Bennett MH. Hyperbaric medicine and the placebo effect. *Diving and Hyperbaric Medicine*. 2014 December;44(4):235-240.) The placebo in medicine has a long and interesting history. Despite the widespread use of placebo medication and sham interventions in clinical research, surprisingly little is known about how placebos work. There is evidence the administration of placebo preparations can induce measurable changes in physiology including the production of endorphins. Placebos usually involve some form of deception, but have been shown to work even when their lack of 'active' ingredients is declared to the patient. The relevance of the nature of placebo effects has become a central debate in the field of hyperbaric medicine with the recent suggestion that 131 kPa of air may be an active therapeutic intervention rather than a convenient and convincing sham. This paper discusses the nature of placebo and participation effects and the implications for hyperbaric oxygen therapy if low-pressure air is regarded as therapeutic.

## Key words

Placebo, research, hyperbaric research, evidence, general interest

## Introduction

With the rising profile of evidence-based medicine over the last 30 years, physicians have an increasing appreciation of the advantages of high-quality evidence in clinical decision making. In this context, it is generally accepted that for the assessment of efficacy of new treatments, appropriately powered, blinded, randomised controlled trials (RCTs) are the design least prone to bias. This design is, therefore, the least likely to lead to false conclusions. Indeed, the very word 'random' has taken on a talismanic quality such that some investigators have included this descriptor even when it is inappropriate.

When well-planned and conducted, RCTs with blinding and allocation concealment have many advantages. Most importantly, they eliminate bias in the allocation of subjects to the alternative treatment arms. That is to say, a properly random allocation method ensures the only reason for differences between the subjects in each treatment group at the start of a trial are those due to random chance. The magnitude of this chance is dependent on sample size and is measurable using standard statistical approaches. An RCT that also ensures allocation concealment (where the individual responsible for enrolling the subjects cannot be aware of the group to which any individual will be allocated) and the blinding of subjects, investigators and outcome-assessors to the actual treatment received by each individual is even less likely to be subject to bias. In these trials, the outcome cannot be systematically affected by the conscious or subconscious bias of either the subject or the investigators because there is no way they can be aware what treatment any individual is receiving.

Of course few, if any, trials are in practice perfect in design and implementation. It is the job of well-informed critical appraisal to determine the reliability of a trial outcome

and, therefore, the degree to which those outcomes should influence practice. Apart from a meticulous and thorough investigation of the methods and conduct of a trial, one further way to appraise treatment outcomes is to evaluate the robustness of apparent treatment effects (good or bad) across a range of studies in similar populations. This is the aim of systematic review and meta-analysis – both of which require appraisal of their design and conduct.

Except in rare circumstances where deliberate misconduct can be demonstrated, we have little choice as consumers of studies than to accept that trials are performed as described. Even given this, we should appreciate there are a number of subtle influences that only meticulous trial design and execution can avoid. This is particularly true of trials of human subjects where important outcomes are either subjective or require interpretation by outcome assessors.

One fascinating aspect of human trials is the potential for biases due to expectations about the effectiveness of treatments and the way in which they are administered. In particular, there are three well-described potential such influences – the 'placebo effect', the 'Hawthorne effect' and the 'nocebo effect'. They are sometimes summarised by the umbrella term 'participation effects'. These effects can make the interpretation of randomised trials problematic unless exemplary trial design is employed and the potential for participation effects acknowledged.

This paper will discuss the interaction of these effects and trial design with particular reference to how one may avoid systematic bias and misinterpretation of outcomes.

## The placebo effect

A placebo has been defined as "*a substance or procedure... that is objectively without specific activity for the condition*

*being treated*”.<sup>1</sup> Inherent in the concept of the placebo is an intention to deceive the patient and sometimes the investigator and the outcome assessor engaged in a trial. Often, patients given a placebo treatment will have a perceived or actual improvement in a medical condition; this is commonly called the ‘placebo effect’. The placebo effect is simply the patient response that cannot be attributed to an investigational intervention. While most often thought of in terms of ‘the power of the mind’, there are a number of potential explanations, any of which may be operating singly or in combination. These include a direct effect of altered levels of hormones or endorphins, expectancy effects, regression to the mean and a flawed trial methodology.

## HISTORY

The word placebo has an interesting origin. Derived from the Latin *placēbō*, meaning “*I shall please*”, the use of this term began with St. Jerome’s translation of the bible from the ‘Old Latin’ to that in use in the Christian church in the fourth century (the translation came to be known as the *Vulgate Bible*, referring to the use of the ‘common’ form of Latin). Here Jerome chose to translate the Hebrew *ethalec*, previously rendered as “*I shall walk with*”, as “*I shall please*” – placebo in Psalm 114:9. By the eighth century, this psalm was an integral part of the *Office of the Dead*, and verse 9 was the first response from the congregation: “*Placebo Domino in regione vivorum*” – “*I will please the Lord in the land of the living*”.

In France, it was the custom for the mourning family to distribute largesse to the congregation immediately following the ritual. Often, distant relatives and even total strangers would attend the ceremony, singing the placebo response while feigning great anguish, in the expectation of receiving a satisfying repast. These ‘placebo singers’ were thus fakers and by the eighteenth century had given their name to fake remedies designed to fool the patient.<sup>2</sup>

At that time, and well into the nineteenth century, placebo remedies were described as ‘commonplace methods or medicine’, perhaps reflecting the relative lack of effective pharmacological agents. The term was not always pejorative. Placebos were used by even the most eminent practitioners. In his 1998 review of the subject, Kaptchuk quotes an 1811 definition as “*any medicine adapted more to please than to benefit the patient, sometimes with a derogatory implication but not with the implication of no effect*” (my emphasis).<sup>3</sup> By the early twentieth century, the practice of deliberately administering therapies known to be inactive was becoming more questionable, with the famous US physician, Richard Cabot, saying that while he had been trained to use placebos, he had concluded “*I have not yet found any case in which a lie does not do more harm than good*”.<sup>4</sup>

That placebos could have salutary effects was clear to practitioners from the start. The first ‘proof’ was published

in 1799 by the British physician Haygarth, when he gave an account of the effectiveness of wooden sham devices designed to mimic the popular (and expensive) metal device called a ‘Perkins tractor’ at ‘drawing out’ rheumatism and inflammation in the head and face.<sup>5</sup>

Despite continuing to be the shady resort of charlatan practitioners, placebos have, however, found an enduring place in human clinical research. By the 1960s, placebo-controlled trials became the norm for trials designed to test new pharmaceuticals where no effective alternative was available, and in many jurisdictions such trials are required for the approval of new medications. In contrast to the placebo effect, inert substances may also produce unpleasant or harmful effects. The term ‘nocebo’ was coined by Walter Kennedy in 1961 to describe this phenomenon.<sup>6</sup> Kennedy chose the Latin word *nocebo* (“*I shall harm*”) because it was the opposite of the Latin word *placebo*, and used it to denote the counterpart of the placebo response.

One might expect from a phenomenon with such a venerable lineage that we would now know a great deal about the mechanisms by which placebos can produce apparently beneficial effects. In fact, surprisingly little is known about what has become a fascinating area of study for some. Indeed, in 2011, the Harvard Medical School formally declared their ongoing interest with the establishment of the *Program in Placebo Studies*.<sup>7</sup>

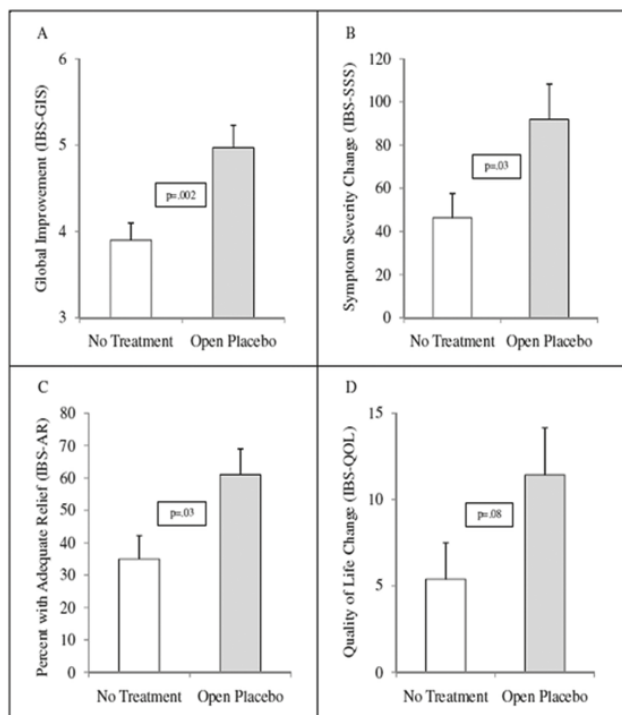
## EVIDENCE OF THE PLACEBO EFFECT

Common placebos include inert tablets, vehicle infusions, sham surgery and other procedures based on false information.<sup>8</sup> Whether we choose to call it a ‘sham compression’ or a placebo, an exposure to a hyperbaric chamber environment that is designed to mimic a true session of hyperbaric oxygen treatment (HBOT) clearly falls within this definition. A problem arises, however, if such a ‘sham’ actually has a therapeutic effect. Put simply, if a placebo actually has demonstrable and reproducible efficacy with clinically important effects, then it would cease to be a placebo and become an effective treatment. It seems a simple distinction, but herein lies the nub of a modern hyperbaric controversy.

Commonly, trials are designed to compare a putatively active therapy against a well-designed placebo or sham; well designed in the sense that the patient cannot distinguish one from the other. The purpose is to demonstrate whether or not the trial treatment can demonstrate effects over and above those produced by an inactive substance. Universally accepted placebos can have a surprisingly positive effect on a patient, and the degree to which a placebo may demonstrate benefit is discussed more fully below. However effective, the principle is that, if an ‘active’ therapy is no more effective than a ‘placebo’ therapy, then there is no ethical justification for using the ‘active’ agent. The most common rationale

**Figure 1**

Improvement in four subjective measures of bowel health and quality of life in patients with irritable bowel syndrome (IBS); in all cases an openly declared placebo was superior to a no-treatment control group in this randomised trial (from Kaptchuck, 2010<sup>10</sup>)

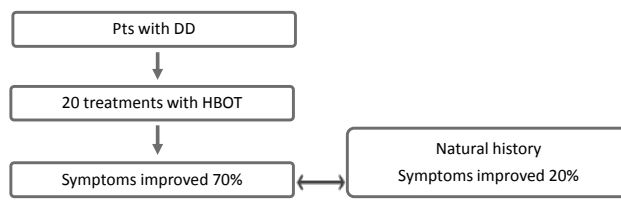


behind this is that the ‘active’ agent is less safe or more expensive and inconvenient than the placebo alternative.

This concept is not novel, and the medical literature is full of such examples. One such example relevant to hyperbaric practice for its physical nature is the well-known sham surgery trial of Dimond.<sup>9</sup> In this randomised study, an experienced cardiac surgeon performed either an internal mammary artery ligation for angina pectoris or a sham procedure through a similar incision with exposure of the vessels but no ligation. The patient and the cardiologist measuring the outcomes were blinded to the allocation. Both groups of patients reported statistically significant improvements in chest pain and used less nitroglycerine for pain relief, but there were no clinically significant differences between the groups. Electrocardiographic signs of ischaemia on exercise were unchanged before and after the procedures in either group. Although one could conclude that both the sham and the ‘real’ operative procedure were truly efficacious, Dimond preferred the interpretation that this was evidence of a participation effect. As he remarked in this paper *“The frightened, poorly informed man with angina, winding himself tighter and tighter, sensitizing himself to every twinge of chest discomfort, who then comes into the environment of a great medical center and a powerful positive personality and sees and hears the results to be anticipated from the suggested therapy is not the same total patient who leaves the institution with the trademark scar.”*

**Figure 2**

A case series describing the use of HBOT to treat hypothetical Davis Disease (DD)



What is less well known is that placebos can have such effects even when the patient knows the given ‘treatment’ is without any active drug, as compared with a control group who knowingly did *not* get a placebo.<sup>10</sup> In this randomised trial, Kaptchuk tested placebo (with reinforcement) against a no-treatment control, with no attempt at deception or concealed administration. Patients were randomized to three weeks of either open-label placebo pills presented as *“placebo pills made of an inert substance, like sugar pills, that have been shown in clinical studies to produce significant improvement in IBS symptoms through mind-body self-healing processes”* or no-treatment controls with the same quality of interaction with providers. There were widespread improvements in placebo over no treatment (Figure 1).

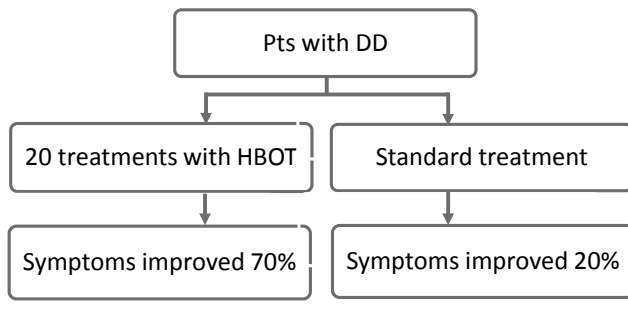
It has been known for some time that placebo effects can be exhibited through specific physiological pathways. In 1978, Levine published a fascinating example using the relief of dental pain with opiates as a model.<sup>11</sup> In a blinded, randomised, controlled trial using pain assessed with a visual analogue scale as the primary outcome, patients were given either naloxone or a placebo at three and four hours after dental extractions. Some given the placebo reported an improvement in pain scores and were identified as ‘placebo responders’. The relevant finding for us is that on subsequent injection of naloxone, the placebo responders reported an increase in pain. The conclusion is that this particular placebo response is mediated through opiate receptors; placebo responders in this model produced endorphins that could subsequently be antagonised by naloxone.

**Hyperbaric oxygen and the placebo effect**

Imagine we are reviewing clinical work designed to demonstrate the effect of a course of HBOT for the hypothetical, chronic, incurable neurological condition ‘Davis Disease’ (DD), named for the first patient in whom it was described. The first piece of evidence we locate is a simple case series as represented in Figure 2. Case series are regarded as poorly reliable clinical proof because of the many potential sources of bias that may be present. For example, these patients may all have a mild form of the disease where symptoms wax and wane over time, or may not all truly have DD because of improperly applied diagnostic testing. One

**Figure 3**

A non-random comparative trial of HBOT versus 'standard treatment' for hypothetical Davis Disease (DD)



further source of bias is that they were all highly selected and motivated, and the improvement seen is a participation effect rather than a true pharmacological effect of HBOT. Whatever the 'truth', there are three potential conclusions: HBOT improved the symptoms of DD in this group of patients; these patients are different in some way from the usual patient with DD and this is the true expected rate of improvement for such a group; or the improvement is due to a placebo or participation effect. On the information given we simply do not know which of these options is the most likely.

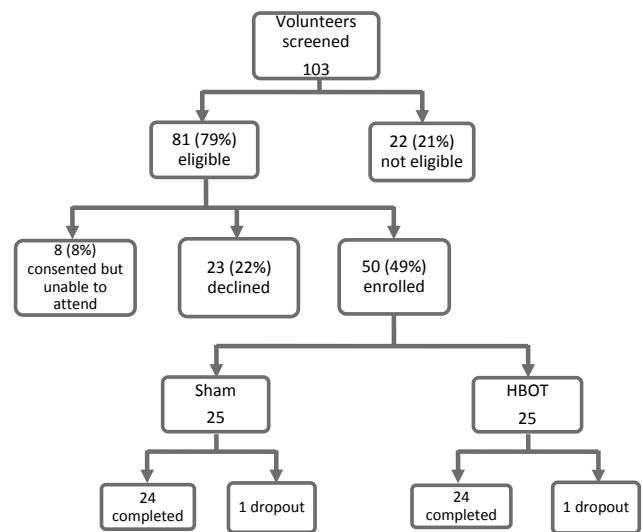
We continue our review of the evidence and find the non-random, cohort study represented in Figure 3. Here a group of patients have been studied, some of whom were selected to have HBOT and some of whom continued to have the standard treatment available. Although the method and circumstances of this selection is of great importance in determining what biases may be more likely in this trial (e.g., those getting HBOT are willing to pay for it, or they are those mobile enough to attend the chamber), the fact is that any non-random selection method is subject to potential bias. Put simply, we cannot guarantee the two groups are exactly comparable in all respects except that one group received HBOT. In fact, our interpretation as to the 'true' effect of HBOT is almost unchanged. HBOT may improve the symptoms of DD, the patients who got HBOT may be different in some way that makes them more responsive, or a participation effect is operating. How likely the second option is to be true will depend on how truly comparable the two groups are; close examination of the methods used, the size of the cohort and the results of any subgroup or propensity analyses may influence our estimation of this likelihood. We still need more reliable information.

The next trial we look at is represented in Figure 4. Now we have found a randomised, blinded, controlled trial where HBOT is compared to a sham therapy involving compression to 131 kPa, breathing air. Importantly, neither the patients nor the investigators were aware of the group to which any individual had been allocated.

The results of the major outcome are reproduced in Figure 5. Both groups have improved in their 'badness' score

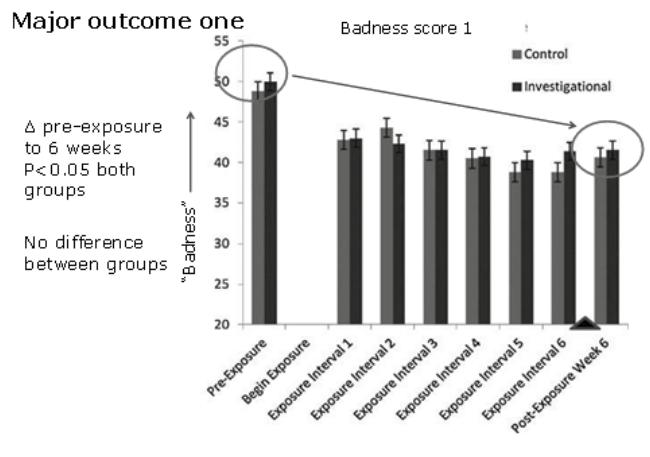
**Figure 4**

A randomised controlled trial of HBOT versus a sham therapy for hypothetical Davis Disease (DD); the sham was 131 kPa breathing air



**Figure 5**

Results of the major outcome in the two randomised groups from the trial shown in Figure 4; the circles highlight the reduction in 'badness' scores in both groups; there are no between-group differences that reach statistical or clinical significance (figure modified from 12)



for this outcome, but there are no important differences between the groups at any time. The difference now is that we have effectively eliminated the potential conclusion that the observed effects are due to differences between the groups. These patients have been randomised, and we rely on this process to evenly distribute all important patient characteristics. Often, authors will publish the proportion of patients in each group who have known potential confounders for the outcome (or the mean value of such a factor), in order to demonstrate there are no important differences between groups, at least for those factors. This is a form of reassurance that the random schedule has performed as expected.



There are now only two potential conclusions – either both therapies work equally well, or there is no true efficacy for HBOT because it performs no better than sham. In the latter case, the improvement must be due to a placebo or participation effect. Which option you prefer will depend on your willingness to accept that the sham therapy is actually an effective treatment in its own right.

#### HBOT AND MILD TRAUMATIC BRAIN INJURY

In fact, these results come from a recent paper investigating the use of HBOT for the treatment of mild traumatic brain injury with ongoing symptoms of post-traumatic stress disorder or post-concussion symptoms.<sup>12</sup> The authors chose to accept the conclusion that a placebo effect was at work: “Given that HBO<sub>2</sub>, in this controlled study, demonstrates no therapeutic value, requires long treatment series, is expensive, exposes patients to potential side effects, and has limited availability, clinical usage is not warranted...”

While this is the position accepted by the majority of practitioners in the field, there are a small number of practitioners and scientists who prosecute the alternative hypothesis.<sup>13</sup> Suggesting the sham here has a ‘real’ therapeutic effect invokes one (or more) of three mechanisms. Breathing air at 131 kPa may be therapeutic because of the pressure exposure or minor increases in the inspired partial pressure of oxygen or of the nitrogen in air. One disturbing consequence of this position is that it may not be possible to truly sham a hyperbaric oxygen session at all. Any convincing ‘pretend’ treatment will inevitably involve some positive pressure above ambient in order to seal the doors of the chamber and produce the need to equalize the middle ear. Efrati has suggested this leaves us with no alternative but to use open-label, randomised evidence as the best possible design in hyperbaric medicine (Efrati SB-JE, personal communication, 2014).

For the majority, the lack of evidence for a therapeutic effect of either the small amount of increase in inspired oxygen (equivalent to about 27% oxygen at 101.3 kPa) or the small increase in environmental pressure and inspired nitrogen means the ‘participation effect’ alternative is simply the much more likely proposition. This assumption is often referred to as *Occam’s razor* or *lex parsimoniae* after William of Occam who popularised this approach in the fourteenth century. Put simply in modern English, Occam suggested that among competing explanations, the one with the fewest assumptions should be selected. While other, more complicated solutions may ultimately prove correct, the fewer assumptions that are made, the better.

#### Conclusions

The placebo effect is under active study and has proved to be both widespread and surprising in scope. Clinical trialists need to be wary of participation effects and in particular are

urged to adopt RCTs with sham controls in order to tease out the true benefit of therapies above those that could be ascribed to placebo.

Once again we find ourselves at a fascinating point in the history of hyperbaric medicine. The long-running arguments within the field concerning the efficacy or otherwise of HBOT for a range of chronic neurological conditions have been hampered until recently by a lack of methodologically rigorous human trials. Sham-controlled trials in multiple sclerosis,<sup>14–16</sup> cerebral palsy,<sup>17</sup> post-concussion syndrome<sup>12,18,19</sup> and autism spectrum disorder (ASD)<sup>20,21</sup> have somehow moved this debate from ‘does HBOT work?’ to ‘do both low-level compression breathing air and HBOT work?’. Of particular methodological interest in this regard is the small trial of Granpesheeh et al, who found no evidence in children with ASD of a difference in outcome between ‘active’ HBO at 131 kPa breathing 24% to 28% oxygen and ambient air using airflow noise to simulate compression.<sup>21</sup>

It is my opinion this is not a helpful debate and may be difficult to resolve. I have no certainty to offer here. The repeated demonstration that we can expect the same results with HBOT and trivial exposures while breathing air (and a number of other versions of sham therapy) seems much more likely owing to the placebo effect than an as yet unexplained mechanism. But it remains possible (if unlikely) that time will prove me wrong. At present, I cannot see how those on the other side of this debate can prove their assertions, given that shamming HBOT is not possible in their interpretation of the world. Interestingly, most protagonists of this interpretation of the evidence still advocate 100% oxygen breathing at 152 kPa rather than the safer, cheaper alternative of 131 kPa air.<sup>22,23</sup> This suggests they still believe in the benefits of HBOT over air-breathing despite the results of the trials referred to above. The impression given is that the goal posts are being moved.

Perhaps the best those of us who have taken Occam’s approach can do is proceed with caution and await some form of convincing evidence that confinement in a chamber at minimal pressure really does have significant healing potential for the human brain. It is a fascinating possibility with great ramifications for the future of hyperbaric medicine. It is also very unlikely.

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**Conflict of interest:** nil

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*Michael H Bennett*

*University of New South Wales and Prince of Wales Hospital, Sydney Australia*

**Address for Correspondence:**

*Associate Professor Michael H Bennett, MD*

*Department of Diving and Hyperbaric Medicine*

*Prince of Wales Hospital*

*Barker St, Randwick*

*NSW 2031, Australia*

**Mobile:** +64-(0)411-483-491

**E-mail:** <m.bennett@unsw.edu.au>

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The database of randomised controlled trials in hyperbaric medicine maintained by Michael Bennett and his colleagues at the Prince of Wales Hospital Diving and Hyperbaric Medicine Unit, Sydney is at:  
<<http://hboevidence.unsw.wikispaces.net/>>

Assistance from interested physicians in preparing critical appraisals is welcomed, indeed needed, as there is a considerable backlog. Guidance on completing a CAT is provided.

Contact Associate Professor Michael Bennett: <m.bennett@unsw.edu.au>

# Short communication

## Blood lead levels in scuba divers: a pilot study

Thorsten Janisch and Rüdger Kopp

### Abstract

(Janisch T, Kopp R. Blood lead levels in scuba divers: a pilot study). *Diving and Hyperbaric Medicine*. 2014 December;44(4):241-242.)

**Introduction:** Lead is a toxic element which is known to accumulate in the body. Nevertheless, it is very widely used as a diving weight.

**Methods:** Blood samples were taken from 20 recreational scuba divers to assess blood lead concentrations.

**Results:** The last dive before blood sampling was an average of 4.8 weeks previously (range 1–18 weeks). All the samples were within the normal background range, the highest lead concentration being  $44.8 \mu\text{g}\cdot\text{L}^{-1}$  with an average concentration of  $26.5 \mu\text{g}\cdot\text{L}^{-1}$  (range  $11.7\text{--}44.8 \mu\text{g}\cdot\text{L}^{-1}$ ).

**Conclusion:** The results show no elevated blood lead concentrations in this group of divers compared to background levels. However, owing to the small number of divers studied and the variable, often long interval between the last dive and blood sampling, the results cannot be generalized.

### Key words

Scuba diving, toxicity, clinical toxicology

### Introduction

Lead is a metallic element found in nature mainly as lead sulphide in combination with other elements (so-called 'galena'). All lead compounds are harmful. Examples of everyday lead-containing products are batteries, insulation, solder for electronic work, X-ray shielding, potable water pipes, ammunition, paints, wood preservatives, as well as its use during glass production.<sup>1,2</sup> The uptake of lead and lead compounds in the human body takes place orally or by inhalation. Transdermal, elemental lead is hardly ever absorbed but organic lead compounds could very well be.

Despite its toxicity, lead is very widely used as a diving weight because of its high density, low price and resistance to corrosion in sea water (see Figure 1 for examples). In this pilot study, we measured the blood lead levels of recreational scuba divers who use lead as diving weights.

### Methods

The study was approved by the RWTH Aachen University ethics committee (EK number 020/14) and was carried out in accordance with the Declaration of Helsinki (2013 revision). Subjects were recruited for study through an invitation to participate using the e-mail distribution list of three local diving clubs in Aachen, Germany in April 2014. Divers who used uncoated lead or lead bags as weights were asked for a blood sample. The first 20 recreational scuba divers who met these criteria were included in the study after formal written consent.

The following data were collected: age, gender, profession, smoking habits, total number of dives, and number of

dives in the past 12 months, date of last dive, type of diving weights used, age of these weights and any history of possible occupational lead exposure.

Following cleansing of the puncture site with an ethanol solution, a blood sample was collected from a median antecubital vein of each subject into an EDTA tube (Sarstedt S-Monovette EDTA K, 2.7 mL). The blood samples were collected over a period of six weeks in April and May 2014 and were stored at room temperature. Lead concentration was assessed on a single run by a high-resolution, continuum source atomic absorption spectrometer (HRCS-AAS; ContrAA 700, Analytik Jena, Germany) at the Institute of Occupational Medicine at the University Hospital Aachen, Germany in May 2014.

The data were anonymised and statistical analysis was conducted using Microsoft Excel® (2003) and IBM SPSS® version 22.

### Results

Mean age of the 20 divers was 44 years (range 32–59 years). Fourteen of the divers were men and six women. Mean number of dives was 861 (range 80–5,600). Mean number of dives in the last year was 62 (range: 10–200). The last dive before the blood sample was drawn was an average of 4.8 weeks previously (range 1–18 weeks).

The highest serum lead concentration was  $44.8 \mu\text{g}\cdot\text{L}^{-1}$  with the average lead concentration being  $26.5 \mu\text{g}\cdot\text{L}^{-1}$  (median  $25.4 \mu\text{g}\cdot\text{L}^{-1}$ ; range  $11.7\text{--}44.8 \mu\text{g}\cdot\text{L}^{-1}$ ). All values were below the reported upper limits of background levels for adults (up to  $70 \mu\text{g}\cdot\text{L}^{-1}$  in women and  $90 \mu\text{g}\cdot\text{L}^{-1}$  in men). There was

**Figure 1**

Top left: V-weight from solid lead; top right: soft weight with lead;  
below: solid lead pieces on a weight belt



no relationship between the number of dives and the lead level (Pearson product-moment correlation coefficient 0.363,  $P = 0.127$ ) nor between the time interval from the last dive to when the blood sample was taken (Pearson product-moment correlation coefficient -0.290,  $P = 0.229$ ).

### Discussion

Upon absorption, lead interacts with the thiol-group of several enzymes, like delta-aminolevulinic acid dehydratase and ferrochelatase, both of which are important for heme biosynthesis. Lead can also modify DNA-methylation.<sup>3</sup> Acute lead poisoning can cause non-specific symptoms such as abdominal, muscle and joint pain, headaches and dizziness, anaemia, nephropathy, and encephalopathy. Mild symptoms may present even at blood lead concentrations below  $100 \mu\text{g}\cdot\text{L}^{-1}$ ; severe symptoms can be expected from a concentration of  $800 \mu\text{g}\cdot\text{L}^{-1}$  upwards.<sup>1</sup> Lead is excreted directly via the kidneys, with a half-life of about 30 days, or stored in the bones.<sup>4</sup> Lead can be released into the bloodstream from bone even after a prolonged period.<sup>2</sup> Chronic or repeated exposure to lead can be asymptomatic but, in addition to the symptoms described above, may lead to cognitive performance degradation, arterial hypertension and foetal damage.<sup>1,5</sup>

To our knowledge, this is the first study to investigate blood lead concentration in recreational divers. Values up to  $70 \mu\text{g}\cdot\text{L}^{-1}$  in women and  $90 \mu\text{g}\cdot\text{L}^{-1}$  in men are the upper limits quoted for lead background levels in adults.<sup>6</sup> Therefore, with a maximum value of less than  $50 \mu\text{g}\cdot\text{L}^{-1}$ , there is no evidence of elevated lead levels in this group of 20 recreational scuba divers.

Our study has several limitations. It is not a random sample, but rather the first 20 volunteers. The average time between the last dive and taking the blood sample was 4.8 weeks (range 1–18 weeks). Since the half-life of incorporated lead is about 30 days, this has an appreciable influence on our results, with possibly higher blood lead levels if blood samples had been taken directly after the dive. As a pilot study, the statistical power was not determined. Therefore, further studies are merited.

### Conclusion

No elevated blood lead levels were measured in a group of 20 divers. Because of study limitations, it cannot be assumed and generalised that divers do not have elevated lead levels from using lead as a diving weight.

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**Conflict of interest:** nil

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*Thorsten Janisch and Rüdger Kopp are specialist anaesthetists in the Department of Intensive Care, University Hospital RWTH Aachen, Germany*

#### Address for correspondence:

*Thorsten Janisch, MD  
Department of Intensive Care  
University Hospital RWTH Aachen  
Pauwelsstrasse 30  
52074 Aachen, Germany  
Phone: +49-(0)241-80-35385  
Fax: +49-(0)241-80-3380444  
E-mail: <tjanisch@ukaachen.de>*

# Case report

## Severe lower limb crush injury and the role of hyperbaric oxygen treatment: a case report

Styliani Stefanidou, Maria Kotsiou and Theodore Mesimeris

### Abstract

(Stefanidou S, Kotsiou M, Mesimeris T. Severe lower limb crush injury and the role of hyperbaric oxygen treatment: a case report. *Diving and Hyperbaric Medicine*. 2014 December;44(4):243-245.)

Open fractures with severe soft-tissue injury and critical local ischaemia of the lower limbs are usually difficult to treat and require a multidisciplinary approach. A 33-year-old Caucasian female with crush injury of the right foot (Gustilo IIIC) was admitted to hospital after a car accident. Despite surgical interventions, a persistent state of hypoxia was present because of the severe vessel injury, and amputation was suggested. Seventy-two hours after admission she was referred to the hyperbaric medicine unit for hyperbaric oxygen treatment (HBOT) to define the limits of viable tissues prior to amputation. After six sessions, clinical improvement was so obvious that the decision to amputate was rejected and she underwent a total of 32 HBOT in addition to frequent debridement and administration of antibiotics. After the HBOT course, she underwent successful surgical reconstruction with a vascularised cutaneous flap. Full healing was achieved. Given the fact that hyperbaric oxygen mechanisms of action target the pathophysiology of crush injuries it should be considered not only for the definition of viable tissue limits but also to enhance viability, even in the most serious situations. HBOT may prove a valuable supplement in the therapeutic armamentarium of these patients.

### Key words

Hyperbaric oxygen therapy, trauma and stress, wounds, case reports

### Introduction

Open fractures with severe soft-tissue injury and critical local ischaemia of the lower limbs are often difficult to treat and require a multidisciplinary approach. When crush injuries are severe, the rate of complications, including infection and non-healing of fractures and tissues, range up to 50 to 60% with a high amputation rate.<sup>1,2</sup> In these cases, adjuvant treatment with hyperbaric oxygen (HBOT) may be of crucial importance, based on sound pathophysiological rationale and growing experimental and clinical evidence. We present a case of lower limb crush injury that highlights many of these issues.

### Case report

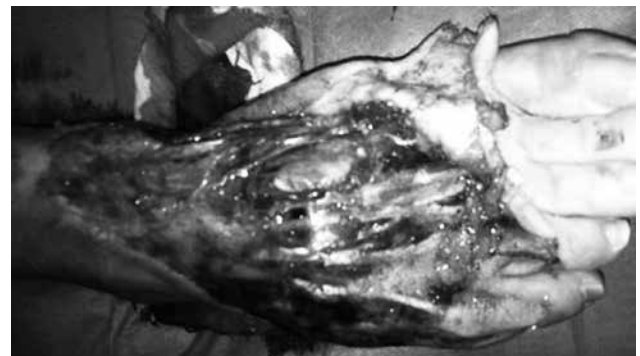
A 33-year-old Caucasian female was admitted to the emergency department with a crush injury of the right foot received in a car accident. She had open fractures with dislocation of the tarsal and all the metatarsal bones (rupture of the ligaments – Lisfranc) and a severe crush injury of the dorsal surface of the right foot. (Gustilo IIIC; Figure 1) The dorsalis pedis artery had been dissected and angiography revealed blockage of the posterior tibial artery with little collateral circulation. Emergency surgery with fixation and stabilization using Kirschner wires was performed, but there was no rheological improvement, as shown in repeat angiography (Figure 2). Transcutaneous oximetry (TCOM) of the right foot showed an intense hypoxic state (almost zero).

Due to the progressive deterioration of the injured foot and the persistent hypoxia, amputation was proposed. Therefore, she was referred to the hyperbaric medicine unit 72 hours after admission in order to improve the local metabolic processes and define the limits of necrotic versus viable tissues. HBOT was administered twice daily for the next three days at a pressure of 243 kPa. Each HBO session consisted of two 40-minute periods of 100% oxygen via an oronasal mask, with a 5-minute air break.

Despite the initial poor prognosis, after these six HBOT, clinical improvement was obvious. There was also a

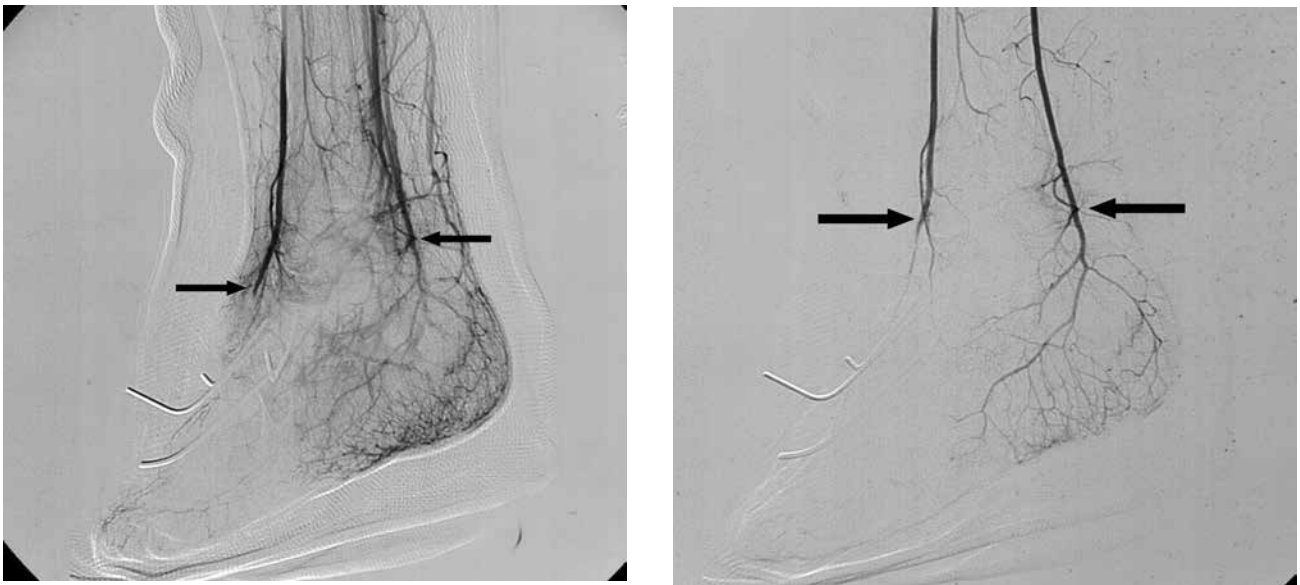
**Figure 1**

Photo of the injured foot on admission to the emergency department; despite the poorly-focused image the severe extent of the injury can be clearly seen



**Figure 2**

Angiography performed after emergency surgical interventions; the dorsalis pedis artery is dissected (left arrow) and there is blockage of the posterior tibial artery (right arrow) and little collateral circulation can be seen

**Figure 3**

The foot at 2-months follow up after surgical reconstruction with a vascularised cutaneous flap



pronounced rise in the TCOM values measured adjacent to the injury to 300 mmHg on oxygen at 243 kPa. Oedema reduction, the decrease in the quantity and improved quality of exudates and the reduction in the inflammatory response were so apparent that the decision to amputate was postponed and an extended HBOT course proposed instead. The patient underwent a total of 32 HBOT, 26 on a daily

basis, without complications, plus frequent debridement to remove necrotic tissue and exudates and to promote tissue granulation. Antibiotic treatment was modified to colistin as *Pseudomonas aeruginosa* was isolated from the wound.

A month later, the patient was submitted to removal of Kirschner wires. At six weeks post injury, a week after the end of the HBOT course, she underwent successful surgical restoration of the foot with a vascular skin flap. (Figure 3). At three months follow up, healing remained complete and with intensive physiotherapy, she had started walking again without the aid of crutches. At seven months follow up, healing remains intact, the patient walks without crutches and has returned to work (as a teacher) and everyday activities.

### Discussion

Crush injury is characterized by a vicious cycle of ischaemia, hypoxia, oedema, disturbed microcirculation and secondary ischaemia in the area bordering the primary trauma.<sup>2</sup> HBOT ameliorates the effects of acute traumatic ischaemia by interrupting this cycle.<sup>1</sup> The therapeutic effects of HBOT are well recognized through experimental and controlled clinical trials in different kinds of ulcers.<sup>1-4</sup> However, clinical experience for its potential efficacy in crush injuries is sparse and there are few cases in the literature where scheduled amputations have been prevented. Current evidence suggests it should be started as soon as possible, preferably in the immediate postoperative period.<sup>4</sup>

In this case, the intensive hypoxia of the injured area despite the surgical interventions was reversed as soon

as HBOT commenced. The progressive increase in the diffusion of oxygen in the injured tissues overcame the “*circulus vitiosus*” of oedema and hypoxia and led to such an apparent clinical improvement that amputation was rejected. The multidisciplinary approach with HBOT, antibiotics, debridements and surgical reconstruction with a vascularised cutaneous flap resulted in the salvage of the foot with good functional recovery.

The oxygen gradient and thus diffusion in plasma and tissues is markedly raised during HBOT and can overcome a decreased but not obliterated perfusion. Injured but viable cells in the penumbra have increased oxygen needs. At a time when oxygen delivery is decreased by impairment of the microcirculation, survival of the cells is directly dependent on oxygen tension.<sup>2</sup> Secondary mechanisms via which HBO may help salvage severely ischaemic tissue include perturbation of ischaemia-reperfusion injury, rheological improvement, elimination of anaerobic bacteria, enhancement of antibiotic action and of the intracellular killing mechanisms of polymorphonuclear leukocytes and an anti-inflammatory effect by inhibition of specific cytokines.<sup>1,5,6</sup>

A distinct feature of hyperoxygenation, particularly with regards to crush injuries, is the pronounced reduction in oedema provoked by vasoconstriction and reduction of blood flow; the latter more than compensated by hyperoxia.<sup>2</sup> Furthermore, HBOT has been demonstrated to promote advancing angiogenesis in the margins of a lesion and this may be relevant in cases such as the one presented here.<sup>5-8</sup> Although each case is unique, we believe that even when further vascular surgery has been excluded, HBOT should be considered not only to define viable tissue limits but also to enhance viability even in the most serious situations. HBOT may prove a valuable supplement in the treatment algorithm of crush injuries.

TCOM is considered to be the gold standard in the evaluation of the response of the tissues to HBOT, and is an important tool both in the selection of patients suitable for treatment and in the prediction of the response to treatment.<sup>3</sup>

## Conclusion

Given the fact that the mechanisms of action of HBOT target the pathophysiology of crush injuries, further evaluation by means of high-quality, randomized controlled trials is needed to determine the role of HBOT as an adjunctive treatment in the therapeutic armamentarium of these patients.

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*Styliani Stefanidou, Maria Kotsiou and Theodore Mesimeris  
Hyperbaric and Diving Medicine Department, St Paul General  
Hospital, Thessaloniki, Greece*

## Address for correspondence:

*Theodore Mesimeris, MD*

*Mitropoleos 99*

*54622, Thessaloniki, Greece*

**Phone:** +30-(0)6937-392662

**Fax:** +30-(0)2310-451727

**E-mail:** <hyperbaricunit@yahoo.gr>

# Continuing professional development

## Inner ear barotrauma

Ian Gawthrop

### Accreditation statement

#### INTENDED AUDIENCE

The intended audience consists of all physicians subscribing to *Diving and Hyperbaric Medicine* (DHM), including anaesthetists and other specialists who are members of the Australia and New Zealand College of Anaesthetists (ANZCA) Diving and Hyperbaric Medicine Special Interest Group (DHM SIG). However, all subscribers to DHM may apply to their respective CPD programme coordinator or specialty college for approval of participation.

This activity, published in association with DHM, is accredited by the ANZCA Continuing Professional Development Programme for members of the ANZCA DHM SIG under Learning Projects: Category 2 / Level 2: 2 credits per hour.

#### OBJECTIVES

The questions are designed to affirm the takers' knowledge of the topics covered, and participants should be able to evaluate the appropriateness of the clinical information as it applies to the provision of patient care.

#### FACULTY DISCLOSURE

Authors of these activities are required to disclose activities and relationships that, if known to others, might be viewed as a conflict of interest. Any such author disclosures will be published with each relevant CPD activity.

#### DO I HAVE TO PAY?

All activities are free to subscribers.

#### Key words

Inner ear, barotrauma, MOPS (maintenance of professional standards),

#### Recommended background reading

Practitioners are referred to the following background references and reading.

- 1 Elliot EJ, Smart DR. A literature review of the assessment and management of inner ear barotrauma in divers and recommendations for returning to dive. *Diving Hyperb Med.* 2014;44:209-23. (this issue)

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- 5 Wong R, Walker M. Diagnostic dilemmas in inner ear decompression sickness. *SPUMS Journal.* 2004;34:5-10.
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#### How to answer the questions

Please answer all responses (A to E) as True or False. Answers should be posted by email to the nominated CPD coordinator.

EUBS members should send their answers to Lesley Blogg. **E-mail:** <lesley.blogg@eubs.org>.

ANZCA DHM SIG and other SPUMS members should send their answers to Neil Banham.

**E-mail:** <neil.banham@health.wa.gov.au>.

If you would like to discuss any aspects with the author, contact him at: <ian.gawthrop@health.wa.gov.au>.

On submission of your answers, you will receive a set of correct answers with a brief explanation of why each response is correct or incorrect. A correct response rate of 80% or more is required to successfully undertake the activity. Each task will expire within 24 months of its publication to ensure that additional, more recent data has not superseded the activity.

#### Question 1. Regarding the anatomy of the inner ear:

- A. Reissner's membrane divides the scala vestibule and the scala media;
- B. the oval window lies inferior to the round window in the vestibule of the membranous labyrinth;
- C. endolymph is contained in the scala media;
- D. the round window receives sound vibrations directly from the stapes footplate;
- E. perilymph and endolymph have similar ionic composition.

#### Question 2. Inner ear barotrauma (IEBt) in divers:

- A. classically causes a conductive hearing loss;
- B. is usually due to rupture of the oval window;
- C. can be due to an anatomical predisposition;
- D. is associated with acute ENT pathology approximately 30% of the time;
- E. can present some days post diving after lifting or straining.



*Question 3. The major differential diagnosis of IEBt is inner ear decompression sickness (IEDCS). These two conditions pose a diagnostic dilemma; however, divers with IEBt:*

- A. have a history of provocative deep diving on mixed gases;
- B. must not be recompressed if the diagnosis remains unclear;
- C. always have signs of middle ear barotrauma;
- D. may possibly improve with recompression;
- E. commonly give a history of problems equalising during the dive.

*Question 4. In the investigation of IEBt:*

- A. comparison with a dive medical baseline pure tone audiogram is helpful;
- B. sensorineural hearing loss involving lower frequencies is associated with round window rupture;
- C. high resolution computed tomography is recommended to identify an anatomical predisposition;
- D. the Tullio phenomenon has a high sensitivity for identifying a perilymph fistula;
- E. the diagnosis is supported by a positive fistula test.

*Question 5. The management of IEBt involves:*

- A. bed rest with head elevation to 30–40 degrees;
- B. steroids;
- C. clear guidelines that exist on which patients need surgical exploration;
- D. avoiding activities that may raise ICP;
- E. the possibility of returning to diving if appropriate criteria are fulfilled.

---

## SPUMS 44th Annual Scientific Meeting 2015

Palau Royal Resort, Malakai, Palau, Micronesia  
16–23 May 2015

### Guest Speaker

Neal Pollock, PhD, Duke University and Director of Research DAN International

### Topics

Diabetes and diving; the older diver; breath-hold diving

**Convenor:** Dr Catherine Meehan, Cairns

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The link to the conference booking site is now open at: <[www.spums.org.au](http://www.spums.org.au)>

Register now

**For further information e-mail:** <[cmeehan@mcleodstmed.com.au](mailto:cmeehan@mcleodstmed.com.au)>

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## Back articles from DHM

After a one-year embargo, articles from *Diving and Hyperbaric Medicine* are placed on the Rubicon Foundation website <<http://www.rubicon-foundation.org/>>, an open-access database, available free of charge and containing many other publications, some otherwise unobtainable. At present, this task is not fully up to date for DHM but articles to the September 2012 issue are now available. Rubicon seeks donations to continue its work to document the hyperbaric scientific literature.

More recent articles or other enquiries about articles should be sent to: <[editorialassist@dhmjournal.com](mailto:editorialassist@dhmjournal.com)>  
Embargoed articles will be charged for; details on application.

# Letter to the Editor

## Australia's model work health and safety regulations and medical fitness requirements for professional divers

In my recent roles as Education Officer for SPUMS and also SPUMS representative on Standards Australia, there were frequent queries regarding the requirements for professional diving medicals in Australia. The requirements for Australia have been set by Australian Federal Government Legislation: *Australian model work health and safety regulations* (4 November 2011).<sup>1</sup>

The legislation requires the medical practitioner providing certification of divers to be registered in Australia. In keeping with this legislation, the 2014 version of Australian/New Zealand Standard 2299.1 will separate the medical requirements for divers depending in which country they are working. New Zealand has a centralised registry and health review system for its professional diver medicals, whereas this is not the case in Australia.

In the new Australian model work, health and safety regulations, the section on *Diving work* commences on page 177, section 4.8. The legislation requires that all occupational divers receive a "current certificate of medical fitness to dive by a doctor with appropriate training in underwater medicine". By the legislated reference to AS2299.1:2007,<sup>2</sup> the South Pacific Underwater Medicine Society is referred to as the appropriate body to provide information on training courses in diving medicine for medical practitioners.

The following is offered for guidance, and the linkages for this mandate are as follows: (The page numbers referred to are in the model work, health and safety regulations)

Definition of "appropriate training in underwater medicine" (Page 4):

*Appropriate training in underwater medicine means training that results in knowledge of the matters specified in clause M3 of Appendix M to AS/NZS 2299.1:2007 (Occupational diving operations—Standard operational practice).*

The requirement for workers to hold a "current certificate of medical fitness" (Page 177, clause 168)

Division 2 General diving work – Fitness and competence of worker

168 Person conducting business or undertaking must ensure fitness of workers

*A person conducting a business or undertaking at a workplace must not direct or allow a worker to carry out general diving work or undergo training for general diving work unless the worker holds a current certificate of medical fitness.*

Definition of "fitness criteria" (Page 19):

Fitness criteria, in relation to diving work, means the fitness criteria specified in clause M4 of Appendix M to AS/NZS 2299.1:2007 (Occupational diving operations— standard operational practice)

M 4.1 General:

*The following bodily systems (Paragraphs M 4.2 to M 4.14) should be evaluated from the diver's history and the medical examination. Where relevant, numerical values are given for certain medical fitness requirements.*

The paragraphs M 4.2 to M 4.14 then cover a comprehensive assessment of body systems that can only be carried out with a medical assessment which includes a physical examination.

Definition of "current" (Page 15):

*Current certificate of medical fitness means a certificate of medical fitness that:*

*(a) was issued within the past 12 months;*

*And (b) has not expired or been revoked.*

Requirement that the certificate is issued by a registered medical practitioner with "appropriate training in underwater medicine" (Page 178, clause 169);

169 Certificate of medical fitness

A certificate of medical fitness must:

*be issued by a registered medical practitioner with appropriate training in underwater medicine.*

and (E) Definition of "registered medical practitioner" (Page 39):

*Registered medical practitioner means a person registered under the Health Practitioner Regulation National Law to practise in the medical profession (other than as a student).*

## References

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- 2 Standards Australia and Standards New Zealand. Australian/New Zealand Standard 2299.1. Occupational diving operations Part 1: Standard operational practice. 2007. ISBN 0 7337 8326 0.

*Associate Professor David Smart, MD  
Department of Diving and Hyperbaric Medicine,  
Royal Hobart Hospital  
Hobart, Tasmania 7000, Australia  
E-mail: <david.smart@dhhs.tas.gov.au>*

## Key words

Diving at work, occupational diving, occupational health, fitness to dive, letter (to the Editor)

## SPUMS Diploma in Diving and Hyperbaric Medicine

### Requirements for candidates (May 2014)

In order for the Diploma of Diving and Hyperbaric Medicine to be awarded by the Society, the candidate must comply with the following conditions:

- 1 (S)he must be medically qualified, and remain a current financial member of the Society at least until they have completed all requirements of the Diploma.
- 2 (S)he must supply evidence of satisfactory completion of an examined two-week full-time course in diving and hyperbaric medicine at an approved facility. The list of such approved facilities may be found on the SPUMS website.
- 3 (S)he must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
- 4 (S)he must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, in a standard format, for approval before commencing their research project.
- 5 (S)he must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication. Accompanying this report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of other documentation, it will be assumed that the paper is to be submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' available on the SPUMS website <www.spums.org.au> or at <www.dhmjournal.com>.

The paper may be submitted to journals other than *Diving and Hyperbaric Medicine*; however, even if published in another journal, the completed paper must be submitted to the Education Officer for assessment as a diploma paper. If the paper has been accepted for publication or published in another journal, then evidence of this should be provided.

The diploma paper will be assessed, and changes may be requested, before it is regarded to be of the standard required for award of the Diploma. Once completed to the reviewers' satisfaction, papers not already submitted to, or accepted by, other journals should be forwarded to the Editor of *Diving and Hyperbaric Medicine* for consideration. At this point the Diploma will be awarded, provided all other requirements are satisfied. Diploma projects submitted to *Diving and Hyperbaric Medicine* for consideration of publication will be subject to the Journal's own peer review process.

#### **Additional information – prospective approval of projects is required**

The candidate must contact the Education Officer in writing (or email) to advise of their intended candidacy and to discuss the proposed topic of their research. A written research proposal must be submitted before commencement of the research project.

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis and if the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed

and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered. It is expected that the research project and the written report will be primarily the work of the candidate, and that the candidate is the first author where there are more than one.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and guidelines on research practice, available at: <www.nhmrc.gov.au/\_files\_nhmrc/publications/attachments/r39.pdf>, or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documentary evidence of approval by an appropriate research ethics committee. Human studies must comply with the Declaration of Helsinki (1975, revised 2013). Clinical trials commenced after 2011 must have been registered at a recognised trial registry site such as the Australia and New Zealand Clinical Trials Registry <http://www.anzctr.org.au/> and details of the registration provided in the accompanying letter. Studies using animals must comply with National Health and Medical Research Council Guidelines or their equivalent in the country in which the work was conducted.

The SPUMS Diploma will not be awarded until all requirements are completed. The individual components do not necessarily need to be completed in the order outlined above. However, it is mandatory that the research project is approved prior to commencing research.

As of 01 June 2014, projects will be deemed to have lapsed if

- 1 The project is inactive for a period of three years, or
- 2 The candidate fails to renew SPUMS Membership in any year after their Diploma project is registered (but not completed).

With respect to 1 above, for unforeseen delays where the project will exceed three years, candidates must advise the Education Officer in writing if they wish their diploma project to remain active, and an additional three-year extension will be granted.

With respect to 2 above, if there are extenuating circumstances that a candidate is unable to maintain financial membership, then these must be advised in writing to the Education Officer for consideration by the SPUMS Executive.

If a project has lapsed, and the candidate wishes to continue with their DipDHM, then they must submit a new application as per these guidelines.

The Academic Board reserves the right to modify any of these requirements from time to time.

As of June 2014, the SPUMS Academic Board consists of:

- Dr David Wilkinson, Education Officer;
- Associate Professor Simon Mitchell;
- Associate Professor (retired) Mike Davis;
- Dr Denise Blake.

**All enquiries and applications should be addressed to:**

David Wilkinson

**Fax:** +61-(0)8-8232-4207

**E-mail:** <education@spums.org.au>

#### **Key words**

Qualifications, underwater medicine, hyperbaric oxygen, research, medical society



## Notices and news



EUBS and SPUMS notices and news and all other society information is now to be found on the respective society websites: <[www.eubs.org](http://www.eubs.org)> and <[www.spums.org.au](http://www.spums.org.au)>

### 41st EUBS Annual Scientific Meeting 2015 First Announcement

Dates: 19–22 August  
Venue: The Academic Medical Center (AMC), Amsterdam

The AMC was one of the founders of hyperbaric medicine in the last century owing to the work of Professor Boerema and his colleagues. His work, in close cooperation with the Royal Netherlands Navy, is often quoted in textbooks on diving and hyperbaric medicine. AMC continues to be highly active.

There will be an excellent mixture of science in diving and hyperbaric medicine.

The annual EUBS meeting coincides with SAIL 2015 – the world-famous, 5-yearly event with Tall Ships and other sailing ships referring to the maritime history and heritage of The Netherlands. The maritime sail event and the numerous cultural aspects of Amsterdam, combined with the renewal of scientific ideas and social contacts, will inspire you!

Looking forward to seeing you in Amsterdam, on behalf of the organizing committee:  
**Albert van den Brink, General Secretary**

**For more information:** <[www.eubs2015.org](http://www.eubs2015.org)>



website is at  
<[www.eubs.org](http://www.eubs.org)>

Members are encouraged to log in and to keep their personal details up to date

#### The 5th Arthur-Bornstein Workshop Diving in offshore wind farms

Unfortunately this meeting had to be postponed as a satellite meeting of the 40th EUBS ASM 2014 in Wiesbaden. It is intended to hold the meeting during 2015 in Germany.

**For more information** contact Dr. Karl-Peter Faesecke:  
<[faesecke@schlaichpartner.de](mailto:faesecke@schlaichpartner.de)>



website is at  
<[www.spums.org.au](http://www.spums.org.au)>

Members are encouraged to log in and to keep their personal details up to date

#### German Society for Diving and Hyperbaric Medicine

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by the German Society for Diving and Hyperbaric Medicine (GTÜeM) according to EDTC/ECHM curricula, can be found on the website:

<[http://www.gtuem.org/212/Kurse/\\_Termin/Kurse.html](http://www.gtuem.org/212/Kurse/_Termin/Kurse.html)>

## Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists

Eligible candidates are invited to present for the examination for the Certificate in Diving and Hyperbaric Medicine of the Australian and New Zealand College of Anaesthetists.

**All details are available on the ANZCA website at:**  
<<http://anzca.edu.au/edutrain/DHM/index.htm>>

*Suzu Szekely, FANZCA, Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine.*

**E-mail:** <[Suzu.Szekely@health.sa.gov.au](mailto:Suzu.Szekely@health.sa.gov.au)>

## Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2015

### Medical Officers' Course

Dates yet to be finalised

### First DMT Course for 2015

Full: 16–27 March

Further information tba in March 2015 issue

### All enquiries to:

Lorna Mirabelli, Course Administrator

**Phone:** +61-(0)8-8222-5116

**Fax:** +61-(0)8-8232-4207

**E-mail:** <[Lorna.Mirabelli@health.sa.gov.au](mailto:Lorna.Mirabelli@health.sa.gov.au)>

## Capita Selecta Duikgeneeskunde The metabolic gases O<sub>2</sub> and CO<sub>2</sub> in diving medicine

14th University of Amsterdam advanced refresher course

**Date:** 07 February 2015

**Venue:** Academic Medical Centre, Amsterdam

Jan Willem Bech, Jean-Claude Le Péchon and Wouter Sterk will discuss safety management (chambers, filling stations), O<sub>2</sub> free radicals, diving (patho)physiology of O<sub>2</sub> and CO<sub>2</sub>, mechanisms of intoxication, O<sub>2</sub> in high altitude diving, working in compressed air, HBOT of DCI, etc. The course is intended for diving physicians, paramedics and high-level diving instructors.

**For further information:** <[www.diverresearch.org](http://www.diverresearch.org)>

The  
**Diving and Hyperbaric Medicine Journal**  
website is at

<[www.dhmjournal.com](http://www.dhmjournal.com)>

## Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organized more than 180 courses over the past 20 years. In 2015 SHF will organize more courses than ever, targeting an international audience.



The courses Medical Examiner of Diver (parts I and II) and the modules of the Diving Medicine Physician course comply fully with the ECHM/EDTC curriculum for Level 1 and 2d respectively and are accredited by the European College of Baromedicine.

### SHF courses for 2015

**24 January:** ENT and diving refresher course; Leeuwarden, Netherlands

**7–14 March:** In-depth course decompression/recompression/HBOT; Manado, Indonesia

**11 & 17 April:** Basic course diving medicine (level 1 part 1); Loosdrecht, Netherlands

**18, 24 & 25 April:** Basic course diving medicine (level 1 part 2); Amsterdam, Netherlands

**13, 14 May:** Basic course diving medicine (level 1 part 1); Oman

**16–23 May:** Basic course diving medicine (level 1 part 2); Oman

**12 & 13 June:** In-depth course diving medicine in case studies; Loosdrecht, Netherlands

**3 October:** ENT and diving refresher course; Rotterdam, Netherlands

**7–14 November:** Basic course diving medicine (level 1 part 1); Palau

**14–21 November:** 23rd SHF In-depth course diving medicine; Palau

**21–28 November:** 23rd SHF In-depth course diving medicine; Palau

**tba:** Basic course diving medicine (level 1 part 1); Antwerpen, Belgium

**For further information:** <[www.scotthaldane.org](http://www.scotthaldane.org)>

## DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.

**Go to the website:** <<http://www.daneurope.org/web/guest/>>

## UHMS Annual Scientific Meeting 2015

**Dates:** 04–06 June 2015 (pre-course 03 June)

**Venue:** Hilton Bonaventure, Montreal, Canada

More information coming soon: <[www.uhms.org](http://www.uhms.org)>

## The ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2015

**Dates:** 23 February–06 March

**Venue:** Prince of Wales Hospital, Sydney, Australia

Course content includes:

- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen
- Wound assessment including transcutaneous oximetry
- Visit to HMAS Penguin
- Visit to the NSW Water Police
- Marine envenomation
- Practical sessions including assessment of fitness to dive

Approved for the ANZCA CPD programme (knowledge and skills category):

56 hours for attendance at lectures/presentations for one credit per hour.

24 hours for workshops/PBLDs/small group discussions for two credits per hour

### Contact for information:

Ms Gabrielle Janik, Course Administrator

**Phone:** +61-(0)2-9382-3880

**Fax:** +61-(0)2-9382-3882

**E-mail:** <Gabrielle.Janik@sesiahs.health.nsw.gov.au>



### DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village  
Victoria, 3172, Australia

**E-mail:** <hdsaustraliapacific@hotmail.com.au>

**Website:**  
<www.classicdiver.org>

## International Meeting on Ultrasound for Diving Research – Ultrasound 2015

**Dates:** 25–26 August

**Venue:** The Swedish Armed Forces Diving and Naval Medical Centre (DNC), Karlskrona, Sweden

This inaugural meeting will bring together experts in diving and decompression physiology to discuss and educate on the use of ultrasound in assessing the stress caused by decompression and the associated risks of decompression sickness. The meeting will include a methodology consensus discussion and hands-on workshops.

**Speakers include:** Ron Nishi, Alf Brubakk, Neal Pollock, Jay Buckley and Mikael Gennser

**Convenors:** Lesley Blogg (SLB Consulting) and Andreas Møllerlökken (NTNU Norway)

**For more information,** please visit:

<ultrasound2015.wix.com/ultrasound2015>

**Facebook:** <www.facebook.com/Ultrasound2015>

**E-mail:** <ultrasound2015@yahoo.co.uk>

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## Hyperbaric Oxygen, Karolinska

Welcome to: <<http://www.hyperbaricoxygen.se/>>.

This site, supported by the Karolinska University Hospital, Stockholm, Sweden, offers publications and free, high-quality video lectures from leading authorities and principal investigators in the field of hyperbaric medicine.

You need to register to obtain a password via e-mail. Once registered, watch the lectures online, or download them to your iPhone, iPad or computer for later viewing.

**For further information contact:**

Folke Lind, MD PhD

**E-mail:** <folke.lind@karolinska.se>

**Website:** <www.hyperbaricoxygen.se>

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## Advertising in *Diving and Hyperbaric Medicine*

Companies and organisations within the diving, hyperbaric medicine and wound-care communities wishing to advertise their goods and services in *Diving and Hyperbaric Medicine* are welcome. The advertising policy of the parent societies – EUBS and SPUMS – appears on the journal website: <www.dhmjournal.com>

Details of advertising rates and formatting requirements are available on request from:

**E-mail:** <editorialassist@dhmjournal.com>

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## Instructions to authors

The 'short' *Instructions to Authors* will no longer be printed in each issue of the Journal. Please refer to the *Diving and Hyperbaric Medicine* website: <www.dhmjournal.com> for a downloadable pdf of the full instructions (revised June 2014).

## DIVER EMERGENCY SERVICES PHONE NUMBERS

### AUSTRALIA

1800-088200 (in Australia, toll-free)  
+61-8-8212-9242 (International)

### SOUTHERN AFRICA

0800-020111 (in South Africa, toll-free)  
+27-10-209-8112 (International, call collect)

### NEW ZEALAND

0800-4DES-111 (in New Zealand, toll-free)  
+64-9-445-8454 (International)

### EUROPE

+39-6-4211-8685 (24-hour hotline)

### ASIA

+10-4500-9113 (Korea)  
+81-3-3812-4999 (Japan)

### UNITED KINGDOM

+44-7740-251-635

### USA

+1-919-684-9111

**The DES numbers (except UK) are generously supported by DAN**

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### DAN ASIA-PACIFIC DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. All information is treated confidentially with regard to identifying details when utilised in reports on fatal and non-fatal cases. Such reports may be used by interested parties to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence unless otherwise agreed) to:

DAN Research  
Divers Alert Network Asia Pacific  
PO Box 384, Ashburton VIC 3147, Australia  
Enquiries to: <research@danasiapacific.org>

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### DAN Asia-Pacific NON-FATAL DIVING INCIDENTS REPORTING (NFDIR)

NFDIR is an ongoing study of diving incidents, formerly known as the Diving Incident Monitoring Study (DIMS). An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.

**The NFDIR reporting form can be accessed on line at the DAN AP website:**  
<[www.danasiapacific.org/main/accident/nfdir.php](http://www.danasiapacific.org/main/accident/nfdir.php)>

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### DISCLAIMER

All opinions expressed in this publication are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policies or views of the SPUMS, EUBS or the Editor and Board.

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