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Association Réunionnaise de Médecine Subaquatique et Hyperbare

Scott Haldane Diving Medicine

SOUTHERN AFRICAN UNDERSEA AND HYPERBARIC MEDICAL ASSOCIATION

Cardiovascular responses to breath-hold diving

Does HBOT alter the biochemical and histological responses to burns?

Can HBOT patients at risk of middle ear barotrauma be identified?

Oxygen, scuba diving and the autonomic nervous system

UHMS diver rescue and resuscitation recommendations

Oxygen and critical flicker fusion frequency

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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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The Editor's offering

Modern non-invasive instrumentation has greatly enhanced our ability to study environmental physiological phenomena such as during immersion and diving. The study by Marabotti et al on the effects of a long breath-hold during horizontal whole-body immersion is an example of the wide range of physiological parameters that can be measured, thus providing new challenges for their interpretation.[1] That human breath-hold diving holds such intense interest for several research groups internationally is not surprising when one considers the complexities of the conflicting physiological processes that accompany it. It seems that knowledge and the breaking of physiological barriers have already moved on since Professor Schagatay's recent three review articles.[2]

Critical flicker fusion frequency (CFFF), which can be defined as the frequency at which a flickering light of constant intensity is seen as a steady light source, has been used in the study of vision, fatigue, workload and vigilance, and the effect of psychotropic drugs, and has been reported previously for diving studies.[3] It has been claimed that CFFF measures brain "*executive function*" (e.g., dual-tasking, inhibition, planning ability, etc.), but this has been disputed recently by two of our reviewers. Nevertheless, CFFF could be a practical substitute for various psychological measures in assessing 'cerebral arousal' in different environmental conditions. J Leach (personal communication, 2013) points out that there are two components to consider, firstly whether CFFF changes occur under different environmental conditions, and secondly to determine what these changes mean with respect to cognitive (brain) function. The present study, looking at differences between air and oxygen breathing at normobaria, is one of an intended series using CFFF to study the diving/hyperbaric environment.[4] It remains unclear to this writer what the eventual role of CFFF testing will be in the study of diver performance.

English as a second language

One of the difficulties our reviewers and I face quite commonly is the submission of manuscripts from authors for whom English is a second (often third or fourth!) language (ESL). As someone who has not dared use his schoolboy French and German for half a century, I admire such efforts. However, sometimes authors' command of English is such that it is difficult to understand what is written and unpleasant to read. I spend a great deal of my time helping to rewrite papers submitted from continental Europe, and my experiences over the past six years of our joint publication convince me that it is essential that the Editor-in-Chief of *Diving and Hyperbaric Medicine* must be a native English speaker. It would assist the journal office greatly if ESL authors would take advantage of the services that are readily available to improve their manuscripts before submission. However, this problem of poor English writing skills is not confined to ESL authors, and some papers submitted by native English speakers are also poorly written. Poor use of language and/or lack of attention to the correct structure of a submission can make the difference between a positive, constructive review and, therefore, the likelihood of successful publication, and one which recommends rejection, simply because the reviewer lost patience with the quality of the presentation. I see my role as Editor as one of facilitating publication if at all possible, but this is sometimes an uphill, ultimately futile battle. Researchers need to remember that journals are for the readers and the message needs to be clear and interesting.

The European Association of Science Editors (EASE <http://www.ease.org.uk/>) is "*an internationally oriented community of individuals from diverse backgrounds, linguistic traditions and professional experience who share an interest in science communication and editing*". EASE provides a freely available guide in more than 20 languages, *The EASE Guidelines for Authors and Translators of Scientific Articles to be Published in English,* that provides simple, clear advice aimed at making international scientific communication more efficient.[5] Another useful tool for authors is the *EASE Toolkit for Authors*.[6] The Toolkit was prepared particularly for junior researchers from non-English or developing countries and aims at increasing authors' confidence in writing and submitting articles. The EASE website has several links to other editorial services that provide English editing for ESL authors. In addition, one member of the EUBS Executive, Lesley Blogg, provides assistance for ESL authors (<SLBConsulting@chapelclose20.fsnet.co.uk>).

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Michael Davis

The Presidents' page

Living in a Bayesian world: can we "Bayes" our approach to decompression?

Costantino Balestra, President EUBS and Michael Bennett, President SPUMS

Ernst Straus said not too long ago (1948) "*this is so simple, God could not have passed it up*". This appealing idea seems apt when considering a very old formula developed by an English presbyterian pastor, Thomas Bayes, in 1748 and refined by Pierre-Simon Laplace thirty years later: Bayes' formula. Bayes formula is so simple that anyone (even the most mathematically inept doctor) can grasp the fundamentals behind it. More than 30,000 publications are available nowadays based on this fantastic equation. Many scientific papers have been published using this phenomenal mathematical idea within the medical field. According to a PubMed search using *Bayes* and *Bayesian* key words, we found that the first thousand papers had been achieved in 2005, the second in 2009, and the third in 2013; every four years about 1,000 papers, meaning that slightly more than one paper per working day is published using this approach!

The power of the formula lies behind three probabilistic functions that permit the calculation of a fourth. It all comes down to the chance of a true positive result divided by the chance of any positive result. We can simplify the equation to:

$$\Pr(A|X) = \frac{\Pr(X|A)\Pr(A)}{\Pr(X)} \quad (1)$$

Pr(X) is a normalizing constant and helps scale our equation. This simple approach allows a large number of applications in the medical field.

Recent collaborative work between three universities raised an interesting question: can we predict medical conditions?[1] In the era of increasingly large diving-related databases, be they military, commercial or recreational, one might reasonably expect many papers published in the diving field to be based on Bayesian principles. Applying the Bayesian key words and adding "*scuba diving*" to the search, we were rather disappointed to find just two papers![2,3] However, at its simplest, the Bayesian model can be expressed as:

Condition 1 and condition 2 → condition 3 (2)

These 'conditions' are factors for which the subject (diver or patient) is positive and are identified from a large set of candidate parameters. When applied to large databases, this approach has the advantage of determining probability of particular associations using very large numbers of subjects, even when relatively little is known about the individual subject. These association rules are designed to generate a hierarchical approach such as:

Dyspepsia and epigastric pain → heartburn (3)

This is a conditional statement indicating that dyspepsia and epigastric pain are commonly followed by heartburn. This concept is of considerable interest in our field; the association could be :

Scuba diving and bubble → DCS (4)

If only it were so simple! It might work that way if our sole purpose was simply to identify cases where DCS (decompression sickness) could be definitively diagnosed. In the real world, it has been repeatedly demonstrated that we cannot define a clear-cut relationship between bubbles and DCS occurrence. Individuals appear to react in very different ways to decompression stress and some cope with it very successfully, others less so.

Applying a Bayesian approach, we must consider the amount (be it in terms of number of circulating gas emboli, arterial or venous, or volume of gas) of bubbles associated with other parameters. For example, we might propose:

Dive profile and individual's parameters → decompression stress (eventually leading to DCS) (5)

This approach might be successful. Can we formulate a successful way of facing this dilemma of decompression stress or DCS as an outcome? According to Einstein, yes, a simple equation can mathematise it. This can be considered as a mathematical counsel to youngsters and researchers :

$A + B \rightarrow C$ (6)

Where C stands for a successful approach to the problem; A stands for enthusiasm and B the capability of knowing when to keep one's mouth shut. Even if this seems a bit puerile, it is a pearl of wisdom, since enthusiasm is generally in opposition to a strategy of keeping tight-lipped.

The take-home message is: Balance. We should not be too exclusive in our approaches to decompression. The Bayes formula shows us that both approaches – one favouring the study of DCS outcome and the other studying decompression stress (with or without DCS) – have value. In this Bayesian world ... two probabilistic approaches have a greater chance to lead to a third final one. That's all we wish!

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Key words

Decompression sickness, research, editorials, general interest

Original articles

Cardiac function and oxygen saturation during maximal breath-holding in air and during whole-body surface immersion

Claudio Marabotti, Paolo Piaggi, Danilo Menicucci, Mirko Passera, Antonio Benassi, Remo Bedini and Antonio L'Abbate

Abstract

(Marabotti C, Piaggi P, Menicucci D, Passera M, Benassi A, Bedini R, L'Abbate A. Cardiac function and oxygen saturation during maximal breath-holding in air and during whole-body surface immersion. *Diving and Hyperbaric Medicine*. 2013 September;43(3):131-137.)

Introduction: The magnitude of the oxygen-sparing effect induced by the diving response in humans is still under debate. We wished to compare cardiovascular changes during maximal breath-holding (BH) in air and during whole-body immersion at the surface in a group of BH divers.

Methods: Twenty-one divers performed a maximal static apnea in air or during whole-body immersion. Doppler-echocardiography, arterial blood pressure and haemoglobin saturation (S_aO_2) were obtained at the beginning of, and at 1/3, 2/3 and maximal BH time.

Results: BH time was on the average 3.6 ± 0.4 min, with no differences between the two conditions. S_aO_2 significantly decreased during BH in both conditions, but was significantly higher during immersion as compared to the dry ($P = 0.04$). In both conditions, BH induced a significant linear increase in right ventricular diameter ($P < 0.001$), left ventricular (LV) volumes ($P < 0.001$) and LV stroke volume ($P < 0.001$) but a significant linear decrease in LV ejection fraction ($P = 0.033$). In both conditions, Doppler diastolic parameters showed changes suggesting a constrictive/restrictive left ventricular filling pattern (i.e., an increase of early diastolic left ventricular filling velocity, $P = 0.005$, and a decrease in the deceleration time of early diastolic left ventricular filling. $P < 0.001$).

Conclusion: BH induces progressive LV enlargement both in air and whole-body immersion, associated with reduced LV ejection fraction and progressive hindrance to diastolic filling. For a similar apnea duration, S_aO_2 decreased less during immersed BH, indicating an O_2-sparing effect of diving, suggesting that interruption of apnea was not triggered by a threshold critical value of blood O_2 desaturation.

Key words

Breath-hold diving, physiology, diving reflex, cardiovascular, echocardiography, Doppler, hypoxia

Introduction

Prolonged interruption of respiration enables mammals to dive. Maximal duration of voluntary breath-holding (BH) is conditioned by the amount of oxygen carried from the surface, by its rate of consumption during diving and by hypoxaemic tolerance, and varies enormously, from hours in some marine mammals to seconds in typical terrestrial animals.[1] Cardiac effects of prolonged BH in humans, evaluated in a magnetic resonance (MRI) study, showed that prolonged BH in air produced no changes in heart rate and peripheral vascular resistance, but rather a progressive depression in cardiac contractility paralleled by left ventricular dilatation with maintained stroke volume and cardiac output.[2]

The diving response is a well-known phenomenon that has an oxygen-sparing effect and may prolong BH duration, as demonstrated in instrumented aquatic mammals and in diving birds.[3,4] The diving response has also been documented in humans during whole-body immersion at surface with face immersion and during in-air experiments.[5,6] Elicitation of the diving reflex by face immersion during in-air apneic dynamic exercise reduced lung oxygen depletion and increased anaerobic metabolism, thus suggesting an oxygen-sparing effect.[7,8] Body immersion entails environmental changes that significantly affect the cardiovascular system. During head-out immersion, a blood shift from the peripheries to the thorax has been observed, leading to an increase in central blood volume.[9] As a consequence of the increased ventricular preload, an increase in both stroke volume and cardiac output has been reported in head-out immersed humans.[10]

The overall haemodynamic changes induced by whole-body immersion and diving response elicitation are an increase in central blood volume and a reduction and redistribution (in favour of heart and brain) of cardiac output. Therefore, immersion, because of haemodynamic changes and diving response activation, might modulate the cardiovascular response to maximal BH and, due to the oxygen-sparing effect of the diving response, could also favorably influence the time-course of hypoxic depression of cardiac function. The aim of this study was to evaluate the influence of whole-

body immersion on haemodynamics and arterial oxygen saturation (S_aO_2) in humans during maximal BH in air and during total-body immersion.

Materials and methods

SUBJECTS

A group of 21 subjects (18 male, 3 female; age 35 ± 4 years, range 26–51 years) was studied. All subjects were high-level recreational free divers with at least three years of apnea diving experience, performing at least 3 h per week of BH diving training; no subject was engaged in regular physical activity besides underwater training. All subjects were able to reach a depth of at least 30 metres' sea water (msw) under constant weight (i.e., with no ballast aid for descent). No subject had a history or clinical evidence of hypertension or other cardiac or pulmonary disease. All subjects were non-smokers and had been fasting for at least 2 h before the study. The local University Hospital Ethics Committee approved the study protocol (approval number 2085). All participants received information about the aims and procedures of the study and gave their written consent.

BREATH-HOLD MANOEUVRE

The study was performed during a free diving meeting at Sharm El Sheikh, Egypt, in a swimming pool (air temperature 27°C; water temperature 29°C) in a single session. Each subject, lying in left lateral decubitus, performed a maximal duration ('peak') apnea the day before the study (mean apnea time 3.7 ± 0.5 min, range 3.0–5.1 min). For each athlete, we designed a personalised schedule for Doppler-echocardiographic acquisition, dividing the pre-determined BH duration into thirds (1 min or more each) and acquiring physiological signals at four epochs:
I – early BH;
II – end of the first third;
III – end of the second third;
IV – 'peak' (near end) of apnea.

In this way, the four epochs were equally spaced within the apnea period in all the athletes. At peak epoch, recording was continued up to the end of apnea.

On the day of the study, each athlete performed two apneas, once in air and once during whole-body immersion. Both tests were performed with subjects lying in left lateral decubitus during maximal inspiration. Before both tests, athletes had a period of 2–4 minutes of relaxation and preparation. During the immersion test, subjects entered the water and spent this preparation period in the upright position beside a metal stretcher held by two assistants. When they were ready to start the test, they lay on the stretcher, made a maximal inspiration and immersed their face. The comfortable water temperature and the period of preparation before immersed apnea minimized the possible acute influence of immersion on the data acquired at the first epoch. The two tests were done 2 h apart; between tests, subjects were allowed drinking water or soft drinks but no food or exercise or breath-hold training. The order of the two tests was assigned randomly.

DOPPLER-ECHOCARDIOGRAPHY

Doppler-echocardiography was performed using a commercially available instrument (MyLab 30, Esaote SPA, Florence, Italy). This technique has proved to be reliable for dynamic and non-invasive assessment of cardiac anatomy and systolic and diastolic function of the left ventricle during diving.[11,12] During immersed apnea, the instrument was placed beside the pool while the operator stood in the water on the left side of the subject being studied.

An apical four-chamber view loop (4 s duration) and a pulsed-wave Doppler tracing of trans-mitral blood flow were recorded. Analysis was made offline, according to the American Society of Echocardiography recommendations, by an expert in Doppler-echocardiography, unaware of the identity of the subject or the experimental conditions.[13] From the four-chamber view, the following parameters were obtained: end-systolic and end-diastolic left ventricular volumes (ESV and EDV respectively) by the area-length method, and right ventricular internal dimension (RV, i.e., maximal diastolic distance from the right side of the interventricular septum to the right ventricular free wall).[14] From the same view, maximum transverse (from inter-atrial septum to the opposite atrial wall, LALL) and supero-inferior (from the mitral valve plane to the opposite wall, LASI) dimensions were calculated for the left atrium during ventricular systole. Early (E) and late (A) peak trans-mitral diastolic flow velocities, as well as deceleration time of E velocity (DTE), were obtained from pulsed-wave Doppler tracings, by sampling blood velocities at the level of the mitral valve tips; E-to-A ratio (E/A) was then calculated.

Duration of cardiac cycle (R–R interval) was measured as the time interval between two consecutive mitral A-peaks; heart rate (HR) was then calculated (60/R–R interval expressed in seconds). The mean value of three consecutive cardiac cycles was considered. Left ventricular stroke volume (SV) was calculated as the difference between diastolic and systolic left ventricular volumes. Cardiac output (CO) was obtained as the product of SV and HR. Finally, left ventricular ejection fraction (EF) was calculated as 100*(SV/EDV).

ARTERIAL BLOOD PRESSURE AND S_aO_2

In a subgroup of six subjects, systolic and diastolic blood pressure (SBP and DBP) were measured at the same time as echocardiography acquisition by a submersible sphygmomanometer; mean arterial pressure (MAP) was calculated as DBP+(SBP-DBP)/3.[15] Total peripheral resistance (TPR) and the rate-pressure product (RPP) were

Figure 1

Changes in end-diastolic volume (EDV, upper-left panel), end-systolic volume (ESV, upper-right), ejection fraction (EF, middle-left), stroke volume (SV, middle-right), maximal transverse dimension (LA LL, lower-left) and right ventricle dimension (RV, lower-right) during the four epochs of maximal breath-hold in air (solid dots) and during surface immersion (open dots). See text for detailed explanations. * $P < 0.05$ (wet vs. dry at each epoch)

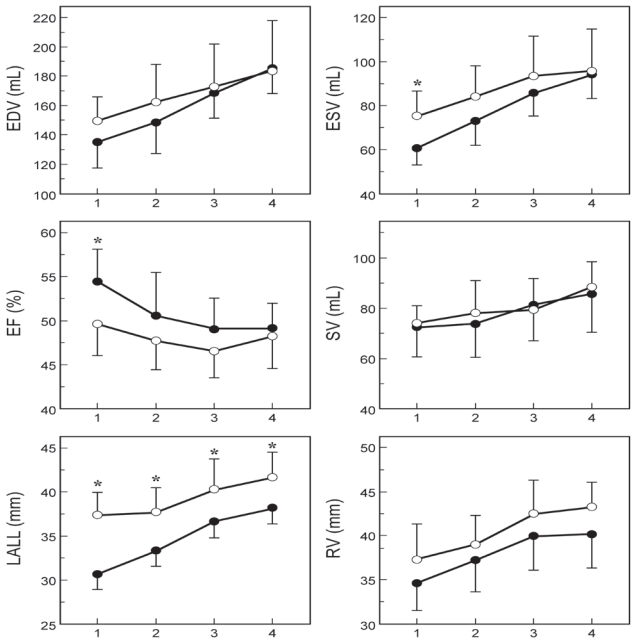
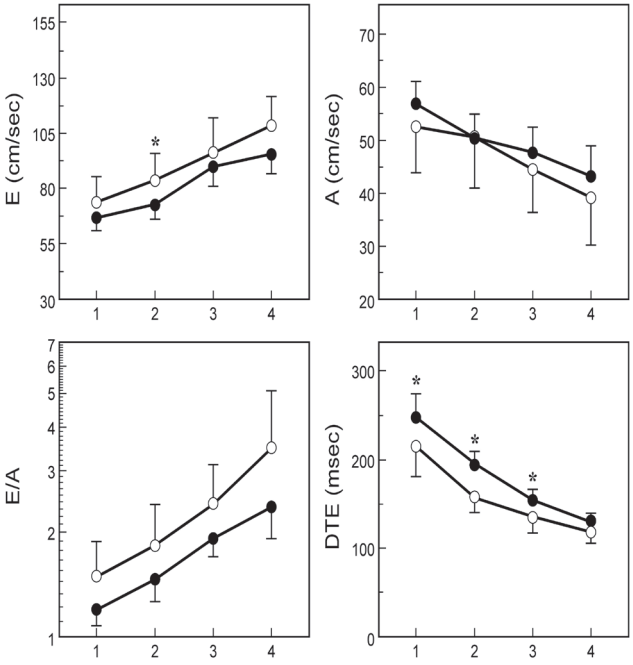


Figure 2

Changes in early (E, upper-left panel) and late (A, upper-right) peak trans-mitral diastolic flow velocities, E-to-A ratio (E/A, lower-left) and deceleration time of E velocity (DTE, lower-right) during the four epochs of maximal breath-hold in air (solid dots) and during surface immersion (open dots). See text for detailed explanations.

* $P < 0.05$ (wet vs. dry at each epoch)



also calculated as MAP/CO and SBP*HR respectively. In the same subgroup, percentage of saturated haemoglobin (S_aO_2) was measured simultaneously with echocardiography by means of an oximeter OEM III (NONIN Medical, Inc., Plymouth, MN, USA) placed over the right temporal artery.

STATISTICAL ANALYSIS

Student's paired t-tests were used to evaluate changes in selected parameters at the different times during apnea and to assess differences between apnea conditions (i.e., in air or immersed).

A mixed model analysis was conducted to explore:

- impact of apnea duration ('effect of time');
- impact of apnea condition (dry or immersed; 'effect of condition');
- impact of interaction between time and apnea condition after taking into account the repeated measures (i.e., intra-subject variability).

Logarithmic transformations were applied for skewed variables having a non-Gaussian distribution according to the Kolmogorov-Smirnov test; post-hoc comparisons were performed using the Bonferroni correction in case of a significant result. *P*-values < 0.05 were considered statistically significant. Data are presented as mean ± standard deviation (SD).

Results

APNEA DURATION

Apnea lasted 3.6 ± 0.4 min (range 2.9–5.3 min). No difference was detected between the two BH conditions.

CARDIAC CHANGES DURING BH (EFFECT OF TIME AND COMPARISON BETWEEN CONDITIONS)

Parameter values (mean and SD) obtained at each epoch during dry and wet BHs are reported in Table 1. No significant interaction effect between apnea duration and apnea condition (immersed or dry) was found for any of the analyzed parameters, meaning that changes relative to each parameter were concordant (i.e., showed the same pattern in the two conditions).

Doppler-echocardiographic parameters

Both EDV and ESV linearly increased from epoch I to IV in both conditions ($P < 0.001$). Immersed apnea, as compared to dry, showed higher ESV at epoch I ($P = 0.015$; Figure 1). EF showed a linear as well as a quadratic negative correlation with time ($P = 0.033$ and 0.039 respectively) in both conditions (Figure 1) in epoch I, being lower in immersed as compared to dry apnea ($P = 0.003$). SV linearly increased from epoch I to IV in both conditions ($P < 0.001$;

Table 1

Parameters (mean (SD) shown) at each epoch during dry and total-body immersion during a maximal breath-hold (see text for detailed explanations); *n* – number of subjects; * – $P < 0.05$ (immersed vs. dry at each epoch); † – $P < 0.05$ (effect of time during apnea)

Parameter	*n*	Condition	Epoch I	Epoch II	Epoch III	Epoch IV
Left ventricular end-diastolic volume (ml)	21	Immersed †	145.5 (31.9)	162.5 (46.3)	168.5 (48.3)	183.9 (62.0)
		Dry †	135.8 (40.1)	150.0 (45.8)	169.5 (45.2)	185.2 (32.9)
Left ventricular end-systolic volume (ml)	21	Immersed †	74.5 (19.6) *	84.3 (22.5)	90.5 (29.7)	95.5 (34.7)
		Dry †	60.3 (16.5) *	73.2 (23.1)	85.0 (25.8)	94.6 (22.0)
Ejection fraction (%)	21	Immersed †	48.7 (5.9) *	47.5 (6.3)	46.7 (5.3)	48.3 (6.7)
		Dry †	55.1 (6.9) *	51.0 (8.7)	49.9 (6.8)	49.2 (4.7)
Stroke volume (ml)	21	Immersed †	70.9 (17.7)	78.2 (27.6)	78.0 (21.5)	88.4 (32.2)
		Dry †	75.5 (27.8)	76.8 (28.4)	84.5 (24.7)	85.6 (25.8)
Heart rate (bpm)	21	Immersed	57.1 (10.0)	54.9 (10.1)	57.0 (10.8)	55.5 (10.6)
		Dry	62.0 (8.2)	58.8 (8.5)	57.5 (13.1)	56.5 (18.1)
Cardiac output (L min^{-1})	21	Immersed	4.04 (1.21)	4.17 (1.38)	4.30 (1.04)	4.42 (1.15)
		Dry	4.61 (1.51)	4.46 (1.56)	4.68 (1.45)	5.14 (2.05)
Left atrial latero-lateral dimension (mm)	21	Immersed †	37.2 (5.1) *	37.2 (5.0) *	40.5 (5.2) *	41.9 (4.9)*
		Dry †	31.0 (4.7) *	33.0 (3.7) *	36.4 (3.5) *	38.2 (3.6)*
Left atrial supero-inferior dimension (mm)	21	Immersed †	40.3 (4.6)	40.8 (4.4)	43.3 (3.8)	45.0 (4.5)
		Dry †	39.2 (4.4)	39.6 (3.5)	41.3 (4.1)	43.4 (4.6)
Right ventricular dimension (mm)	21	Immersed †	36.9 (6.5)	38.7 (5.1)	39.9 (11.4)	43.3 (4.5)
		Dry †	34.6 (5.1)	36.9 (6.0)	39.9 (6.5)	40.1 (6.7)
Early trans-mitral flow velocity (cm sec^{-1}) (ET-MV)	21	Immersed †	72.4 (17.2)	83.3 (17.2) *	94.8 (20.5)	108.4 (21.2)
		Dry †	66.4 (11.6)	71.6 (11.7) *	89.6 (12.8)	95.8 (17.6)
Late trans-mitral flow velocity (cm sec^{-1})	21	Immersed †	53.6 (13.1)	50.1 (12.7)	45.7 (11.5)	38.9 (13.9)
		Dry †	56.3 (11.4)	50.8 (9.6)	48.0 (7.2)	43.1 (10.7)
Early/late ratio	21	Immersed †	1.4 (0.5)	1.8 (0.8)	2.3 (1.0)	3.5 (2.5)
		Dry †	1.2 (0.2)	1.5 (0.3)	1.9 (0.4)	2.4 (0.8)
Deceleration time of ET-MV (msec)	21	Immersed †	196.5 (53.6) *	155.1 (31.4) *	126.4 (25.9) *	119.0 (22.0)
		Dry †	240.7 (51.5) *	191.8 (32.4) *	154.9 (22.0) *	130.8 (13.0)
Systolic BP (mmHg)	6	Immersed	138.1 (22.4)	138.3 (14.5)	150.7 (21.1)	158.8 (31.9)
		Dry	120.5 (18.2)	124.7 (18.0)	122.9 (47.6)	143.3 (11.7)
Diastolic BP (mmHg)	6	Immersed	78.6 (16.6)	86.4 (6.8) *	90.1 (9.5)	100.2 (14.1)*
		Dry	68.8 (16.8)	72.8 (9.2) *	83.1 (11.7)	81.7 (11.3)*
Mean BP (mmHg)	6	Immersed †	97.0 (10.6)	108.2 (6.1) *	112.0 (14.2)	111.2 (7.7)*
		Dry †	76.9 (11.1)	83.3 (10.5) *	86.6 (16.0)	104.3 (4.3)*
Total peripheral resistance (mmHg*min L^{-1})	6	Immersed	26.6 (8.5)	31.4 (8.4)	26.1 (4.7)	28.6 (1.2)
		Dry	20.5 (7.0)	24.8 (9.7)	24.9 (12.0)	21.3 (5.3)
Rate-pressure product (bpm*mmHg)	6	Immersed	7,660 (1,882)	7,283 (1,475)	8,683 (1,676)	8,249 (1,669)
		Dry	7,315 (1,122)	7,086 (1,037)	6,783 (2,933)	8,433 (2,235)
Haemoglobin saturation (%)	6	Immersed †	98.2 (2.1)	96.3 (3.3)	91.7 (4.8)	91.5 (5.2)
		Dry †	98.2 (1.6)	96.9 (1.8)	90.0 (4.2)	84.6 (7.4)

Figure 1). No significant difference was present between the two conditions at each epoch.

Left atrial dimensions, as assessed by latero-lateral and supero-inferior diameters in the four-chamber view, increased linearly with time in both conditions ($P < 0.001$ and $P = 0.001$ respectively). Latero-lateral diameter was greater in immersed compared with dry apnea at all epochs ($P = 0.001$; Figure 1).

Right ventricular transverse diameter increased linearly with time in both conditions ($P < 0.001$) with no significant difference between conditions (Figure 1). The E-wave peak velocity of the left ventricular filling rate increased with time in both conditions ($P = 0.005$) without any difference between the two conditions except at epoch II (Figure 2). The A-wave peak velocity decreased linearly with time in both conditions ($P < 0.001$) without any difference between the two conditions (Figure 2). The E/A ratio linearly increased with time in both conditions ($P < 0.001$) without any difference between the two conditions (Figure 2). Finally, DTE showed significant linear and quadratic decrease with time in both conditions ($P < 0.001$ for both) with significantly lower values during immersed apnea at epochs I, II and III as compared to dry apnea ($P = 0.012$, Figure 2).

Haemodynamic parameters

HR and cardiac output did not change with time in either condition and no differences were found between conditions. MAP increased with the duration of apnea during both conditions, while a similar trend in systolic and diastolic arterial pressures did not reach statistical significance. Increase in both diastolic and mean arterial pressure during immersed compared with dry apnea reached statistical significance at epoch II (DBP $P = 0.003$; MAP $P = 0.011$) and at epoch IV (DBP $P = 0.038$; MAP $P = 0.015$). Systemic vascular resistance tended to increase with time ($P = 0.06$). Moreover, TPR showed higher values at peak apnea than at the onset of apnea (26.7 ± 8.7 vs. 21.0 ± 5.7 mmHg*min L^{-1}; $P = 0.005$) during both conditions but no significant difference between conditions. RPP, an index of external cardiac work, did not change with time.

Oxygen saturation

In both conditions, S_aO_2 remained constant at epochs I and II, but progressively decreased at epochs III and IV (Table 1). Significantly higher values of S_aO_2 were observed during immersed compared with dry apnea ($P = 0.040$); at peak apnea, mean S_aO_2 was higher in water than in air, although the difference did not reach statistical significance ($P = 0.13$).

Discussion

CARDIAC ANATOMY AND SYSTOLIC FUNCTION

In the current study, prolonged apnea (whether dry or immersed) induced a progressive increase in left ventricular systolic and diastolic volumes with reduced left ventricular EF but increased stroke volume and no changes in cardiac output. These findings substantially confirm the pattern previously documented with MRI during maximal dry apnea in elite apnea athletes.[2] In that study, progressive increases in left ventricular diastolic and systolic volumes, impairment of LV systolic function as shown by reduced maximal left ventricular elastance and ejection fraction, but maintained stroke volume and cardiac output, likely through the activation of the Frank-Starling mechanism, were seen.[2] The finding of progressive LV enlargement is also in agreement with previous studies on animals with either mechanical airway obstruction or with respiratory paralysis and artificial ventilation, showing that prolonged hypoxia impairs left ventricular systolic function and induces left ventricular dilatation.[16,17]

In contrast, comparison of the present findings with previous ones at depth is made difficult by the fact that available observations at depth are limited to the early period of apnea.[11,12,18] Nevertheless, if comparison is confined to the early phase of apnea, enlargement of LV volume and increase in SV observed during both dry and shallow immersion apnea, contrast with LV volumes and SV reduction at depth. As compared to diving at depth, shallow immersion is characterized by mildly increased environmental pressure (approximately 40 cm of water). Although sufficient to exert compression on the venous system, this hydrostatic pressure is unable to reduce pulmonary gas volume, thus preventing the significant pulmonary blood shift and chest volume reduction occurring at greater depth that could affect central haemodynamics and, therefore, explain the different changes in LV volumes observed during shallow and at-depth immersion.[9,19]

LEFT VENTRICULAR DIASTOLIC FUNCTION

A progressive increase in early diastolic trans-mitral velocities and reduction in deceleration time of E velocity was observed during BH in both conditions. These changes resemble a constrictive/restrictive left ventricular diastolic pattern.[20] A similar diastolic pattern was previously described during breath-hold diving at depth.[11] This pattern could be reversed to normal after chest re-expansion at depth by means of a single maximal inspiration from a scuba device.[18] However, at variance with the present study, the above pattern was not observed during shallow immersion.[12] This discrepancy might be explained by the spot nature of previous observations limited to the early phase of apnea rather than to the entire period of apnea including peak maximal apnea, when the additional effects of hypoxia become apparent. The diastolic dysfunction observed with shallow immersion, in the absence of hydrostatic chest compression, might be explained by the combined effect of the positive transthoracic pressure induced by breath-holding at total lung capacity (with relaxed respiratory muscles) and of the reduced compliance of the dilated left

ventricle. Alternatively, or in combination, the progressive increase in right ventricular volume during maximal BH may also support the hypothesis that right ventricular dilatation hampered left ventricular filling (ventricular interdependence).[21,22] The more pronounced constriction observed during immersed apnea might be related to the shift of blood from the peripheral venous pool to the thorax, increasing the amount of uncompressible blood in the thorax. Whatever the case, left ventricular diastolic impairment may explain the observed progressive left atrial enlargement during apnea.

HAEMODYNAMICS

As previously observed during dry maximal apnea, MAP increased slightly during breath-hold, both in dry and immersed conditions.[2] Previous papers reported large increases of arterial pressure during prolonged BH in man, sitting in air with face immersed in cold water.[23] This discrepancy may be due to differences in BH protocols between studies, in particular to the different environment of evaluation (medical laboratory vs. swimming pool, the latter being likely perceived as familiar by divers) and to the different water temperature (6–8^{O}C vs. 29^{O}C).

Recent data have shown that divers have a greater sympathetic and pressor response to apnea as compared to matched non-diver controls, suggesting that BH dive training may affect the autonomic response to hypoxia.[24] In that series, the vasoconstrictor response correlated with the duration of apnea, indicating a possible contribution of sympathetic tone to the oxygen-sparing effect. The observation in our study of significantly higher MAP values in the late stages of immersed BH, as compared to the dry, suggests that, in diving-trained subjects, immersion may drive the sympathetic flow contributing to coping with prolonged apnea.

OXYGEN SATURATION

In previous studies of human BH diving, elicitation of the diving response decreased the rate of oxygen desaturation, and face immersion attenuated cardiovascular depression.[7,25–27] On this basis, we expected higher S_aO_2 during immersed apnea compared to dry. Whilst S_aO_2 decreased during both conditions it was significantly higher during immersed apnea compared with dry. In particular, at peak apnea, S_aO_2 was on average 83% in dry as opposed to 92% in immersed apnea. Interestingly, duration of maximal apnea was similar in wet and dry conditions suggesting that interruption of apnea was not triggered by a threshold critical value of blood O_2 desaturation.

DIVING RESPONSE

Heart rate did not significantly change with time during either condition, nor was it different in immersed as compared to dry apnea. Similarly, no significant difference was found in total vascular resistance between BH conditions. This behaviour was in line with recent results showing that cardiovascular autonomic control in breath-holding humans does not show significant changes until the hypoxic BH phase.[28] In spite of the lack of the main hallmarks of the diving reflex (possibly due to the warm water), a higher S_aO_2 was observed in immersed apnea compared to dry. This finding could indicate that, in trained subjects, immersion by itself has an oxygen sparing effect, possibly owing to a higher level of relaxation and, hence, to a reduction of metabolic requirements.

Conclusions

In conclusion, the present study confirms that in-air maximal voluntary BH leads to progressive left ventricular dilatation with increased stroke volume and maintained cardiac output. Whole-body immersion induced similar haemodynamic changes but significantly less oxygen desaturation, suggesting an oxygen-sparing effect of immersion even in the absence of increased hydrostatic pressure and thus of significant pulmonary blood shift. The similar apnea duration in dry and immersed BH, in spite of different S_aO_2 levels, suggests that apnea interruption is not driven by blood oxygen desaturation. Finally, hindrance to left ventricular filling was observed in both dry and immersed apnea, resembling that described at depth and compatible with a constrictive-restrictive effect of chest squeezing. The mechanism underlying this pattern in maximal apnea may be related to increased right ventricular volume (ventricular interdependence), in turn linked to increased venous return to the heart.

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Functional comparison between critical flicker fusion frequency and simple cognitive tests in subjects breathing air or oxygen in normobaria

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Abstract

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Introduction: Measurement of inert gas narcosis and its degree is difficult during operational circumstances, hence the need for a reliable, reproducible and adaptable tool. Although being an indirect measure of brain function, if reliable, critical flicker fusion frequency (CFFF) could address this need and be used for longitudinal studies on cortical arousal in humans.

Methods: To test the reliability of this method, the comparison between CFFF and three tests (Math-Processing Task, Trail-Making Task, and Perceptual Vigilance Task) from the Psychology Experiment Building Language battery (PEBL) were used to evaluate the effect of 10 minutes of 100% normobaric oxygen breathing on mental performance in 20 healthy male volunteers.

Results: Breathing normobaric oxygen significantly improved all but one of the measured parameters, with an increase of CFFF (117.3 ± 10.04% of baseline, $P < 0.0001$) and a significant reduction of time to complete in both the math-processing (2,103 ± 432.1 ms to 1,879 ± 417.5 ms, $P = 0.0091$) and trail-making tasks (1,992 ± 715.3 to 1,524 ± 527.8 ms, $P = 0.0241$). The magnitude of CFFF change and time to completion of both tests were inversely correlated (Pearson r = -0.9695 and -0.8731 respectively, $P < 0.0001$). The perceptual vigilance task did not show a difference between air and O_2 ($P > 0.4$).

Conclusions: The CFFF test provides an assessment of cognitive function that is similar to some tests from PEBL, but requires a less complicated set up and could be used under various environmental conditions including diving. Further research is needed to assess the combined effects of increased pressure and variations in inspired gas mixtures during diving.

Key words

Air, oxygen, narcosis, performance, psychology, research

Introduction

For the diver, the probability of being victim of a narcotic event is higher than that of having a decompression accident. Yet fundamental studies about the mechanisms of inert gas narcosis (IGN), conducted mainly in the context of air diving, saw a rapid decline linked to the introduction of helium diving.[1] However, the popularity of recreational diving and, in particular, technical diving, calls for further study.

Nitrogen narcosis occurs in man at around 0.4 MPa and includes spatial and temporal disorientation, euphoria, hallucinations, disruption in motor and locomotor coordination, mood changes and cognitive impairment.[2] The behavioural approach to its study claims that the majority of the cognitive deficits are caused by a single fundamental deficit: the slowing of information processing due to decreased arousal, which is controlled within the reticular formation.[3,4] This hypothesis seems to be confirmed by neurochemical studies on rats.[5]

One of the most compelling questions within the field of neuroscience is how the complexity of human behaviour emerges from activities at the synaptic and cellular level and, in spite of the continuing advancements in technology, the current and dominant emphasis within this field largely fails to address questions and applications outside the laboratory.[6] This is especially true in the case of IGN as neurophysiologic or neurochemical measurements could be difficult to assess directly underwater, undoubtedly because of the lack of available technical and methodological means.

The re-emergence of the critical flicker fusion frequency (CFFF) test in experimental diving medicine may address this need. Historically a correlation between change in the mental state of divers, CFFF and electro-encephalogram (EEG) has been reported.[7] Also, changes in CFFF during a helium-oxygen dive to 62 ATA (6.28 MPa) showed systematic variations and a relationship between compression and pressure.[8] These variations were grossly parallel to EEG modifications. Since EEG reflects a range of complex brain activities, CFFF, which appears to correlate with EEG, might usefully reflect changes in brain function.[9] Unfortunately, subsequent investigators could not replicate these earlier results, and the use of CFFF was abandoned, it being considered unreliable and non-specific.[10,11]

Outside of the field of diving, despite some limitations, several authors have emphasized the advantages of CFFF assessment as a simple, objective, quantitative method for measuring alertness and arousal in humans.[12–17] In studies during anaesthesia, CFFF revealed brain impairment earlier than some behavioural tests or subjective symptom

appearance.[18, 19] Under standard conditions, the CFFF test may be used for longitudinal studies on cortical arousal in humans.[13]

The purpose of the present study was to assess brain performance using CFFF and behavioural (psychometric) tests to validate the possibility of detecting changes while breathing 100% oxygen (O_2) or air under normobaric conditions. The choice of O_2 was made because previous studies of IGN showed that the changes observed were also related to the oxygen partial pressure.[20–22] Should the results be favourable, the method could then be extended to the evaluation of performance under various environmental diving conditions.

Materials and methods

Experimental procedures were conducted in accordance with the Declaration of Helsinki and were approved by the Academic Ethical Committee of Brussels (CE-B200-2011-5). All methods and potential risks were explained to 20 male volunteers who gave their written, informed consent prior to the experiment. All subjects were healthy non-smokers, undertook regular physical activity (aerobic exercise one to three times a week), were on no medications and had no history of migraine. The participants were instructed not to take any alcoholic beverages for 72 hours and no caffeine-containing beverages for 4 hours before the experiments.

PROCEDURES

All tests were carried out in a quiet room at a constant temperature of 22°C to avoid any disturbance of concentration. All subjects were asked to wear a Tru-fit Mask with a demand valve (Life Support Products, USA) and breathe either air or O_2 for 10 minutes in a randomized order. After 10 minutes for each breathing condition, three tests from the Psychology Experiment Building Language (PEBL) battery were performed and immediately after this the CFFF test was undertaken. The total procedure time was 16.5 ± 1.55 min.

CRITICAL FLICKER FUSION FREQUENCY TEST (CFFF)

In the present study, CFFF was assessed with a specific watertight device (Human Breathing Technology, Trieste, Italy).[10,11] The device consists of a rotating ring, surrounding a short cylindrical waterproof plastic housing of 8 cm diameter containing the numeric (digital) frequency indicator covered by an acrylic transparent window. Attached to this housing a flexible cable is connected to a single blue light emitting diode (LED, colour temperature 8000 Kelvin), inserted in a small, waterproof, cylindrical container (to shield it from stray light and reflections). During the test the subject is looking straight at the LED at a distance individually adapted to their personal vision, generally around 50 cm. The investigator increases or decreases the flickering frequency of the LED by operating the rotating ring in the appropriate direction.

Thanks to the design of the device, subjects are not aware of the starting and actual flicker frequency before, during and after the test. When the subject sees the light change from fusion to flicker (or flicker to fusion), the subject signals the investigator and the test is stopped. The frequency reached is recorded. Each subject performed the test three times and the average was calculated for statistical analysis.

THE PSYCHOLOGY EXPERIMENT BUILDING LANGUAGE BATTERY TESTS (PEBL)

PEBL tests were specifically chosen to track deterioration in visual-perceptual organization, visual-motor coordination as well as integration, and visual memory.[23] Three PEBL tests were used: math processing, trail making and perceptual vigilance.

Math-processing task (MathProc)
The subject is asked to subtract and/or add one- or two-digit numbers presented on a screen and to assess whether the result is more or less than five in a maximum 4-second time frame; this procedure is limited to 2 minutes. Time to complete the task within the 4-sec time frame, and correct, incorrect and timed-out answers are used for analysis.

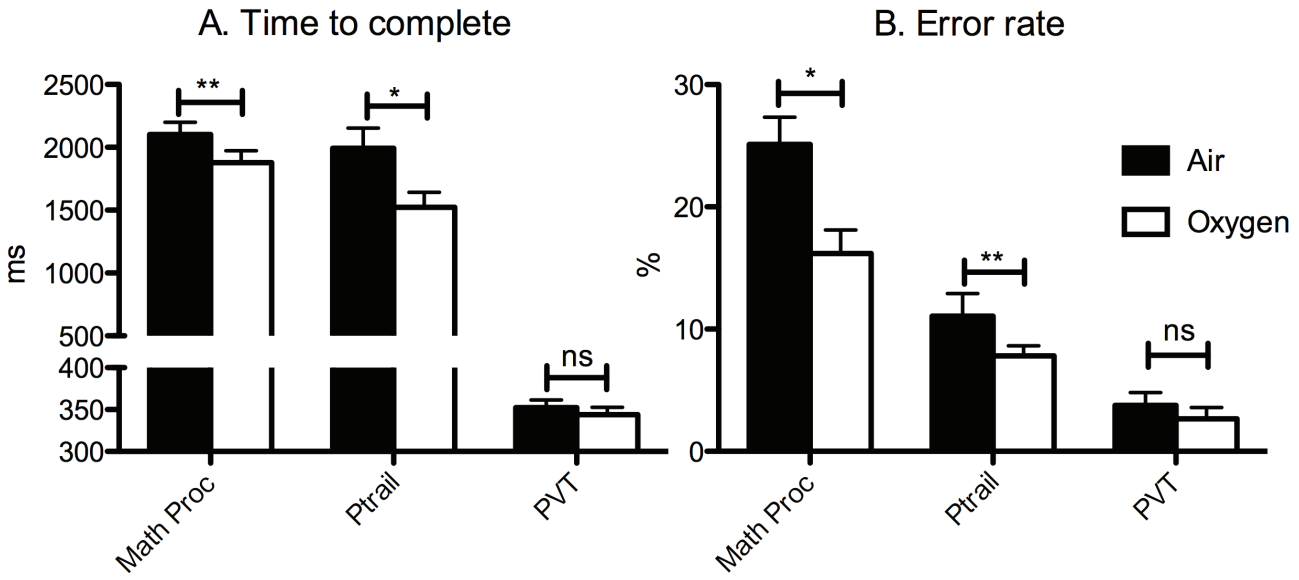
Trail-making task (Ptrail)
This test is used to assess brain injury, hand-eye coordination and general intelligence. The test is divided into 2 parts. In the first part, the circles are numbered (1, 2, 3, etc.) and the subject has to connect them in numerical order (1, 2, 3, etc.). In the second part, the circles have both numbers and letters and the subject has to click on them in alternating order (1-A, 2-B, 3-C, etc.). The trial continues until the test person has connected all the circles in the correct order. The number of clicks to finish the test and erroneous clicks are stored for analysis.

Perceptual vigilance task (PVT)
This test is commonly used to measure simple response time. Using a computer screen and a keyboard, the participant has to press the spacebar as quickly as possible when a red circle stimulus appears randomly at delays between 2 and 12 seconds for 16 times for a maximum of 2 minutes. The reaction times are captured for analysis.

STATISTICS

As they are time-limited tasks, each subject performed a different number of calculations for MathProc or simple response times for PVT in the defined time periods. Before analysis, we calculated the mean for each test and participant in order to obtain a unique set of 20 measures for each breathing condition. Since all data passed the Kolmogorov-

Figure 1
A. Time to complete MathProc and Ptrail tests and reaction time (PVT)
B. Error rate (%) in the three PEBL tests used during air or oxygen breathing (ns = not significant; * $P < 0.05$; ** $P < 0.01$)



Smirnov and Shapiro-Wilk tests, allowing us to assume a Gaussian distribution, they were analysed with a Student's paired test and two-way ANOVA.

Taking the initial (pre-breathing) values as 100%, percentage changes were calculated for each parameter, allowing an appreciation of the magnitude of change rather than the absolute values. Then, a possible correlation was assessed through a Pearson test and linear regression. All tests were performed using a standard computer statistical package, GraphPad Prism version 5.00 for Windows (GraphPad Software, San Diego, California, USA). A threshold of $P < 0.05$ was considered statistically significant. All data are presented as mean ± standard deviation (SD).

Results

Mean age of the 20 subjects was 25 ± 6.6 years; weight 71.4 ± 9.5 kg; height 1.77 ± 0.06 m; BMI 22.8 ± 2.0 kg m^{-2}.

CRITICAL FLICKER FUSION FREQUENCY TEST

The mean CFFF while breathing 100% normobaric O_2 was significantly higher (117.3 ± 10.04 %, $P < 0.0001$) than when the subjects breathed air (baseline, 100%).

PEBL TESTS

In general, breathing normobaric O_2 significantly improved all measured parameters. The mean time to complete the math-processing task while breathing air was 2,103 ± 432 ms and, for trail-making, 1,992 ± 715 ms. After O_2 breathing, these times significantly decreased to 1,879 ± 418 ms ($P = 0.0091$) and 1,524 ± 528 ms ($P = 0.0241$) respectively (Figure 1A). A comparable effect was observed in the math-processing error rate from 25.1 ± 10% to 16.2 ± 8.6% ($P = 0.0263$) and, for trail-making, from 11.06 ± 8.29% to 7.8 ± 3.7% ($P = 0.0066$) (Figure 1B). The perceptual vigilance task did not show a significant difference between air and O_2 ($P > 0.4$). All values have been rounded up or down.

Two-way ANOVA showed a 6.8% chance of randomly observing as much interaction between the PEBL tests and O_2 breathing. Therefore, results of each test are not influenced by other tests (F = 2.75, DFn = 2, DFd = 114, $P = 0.068$).

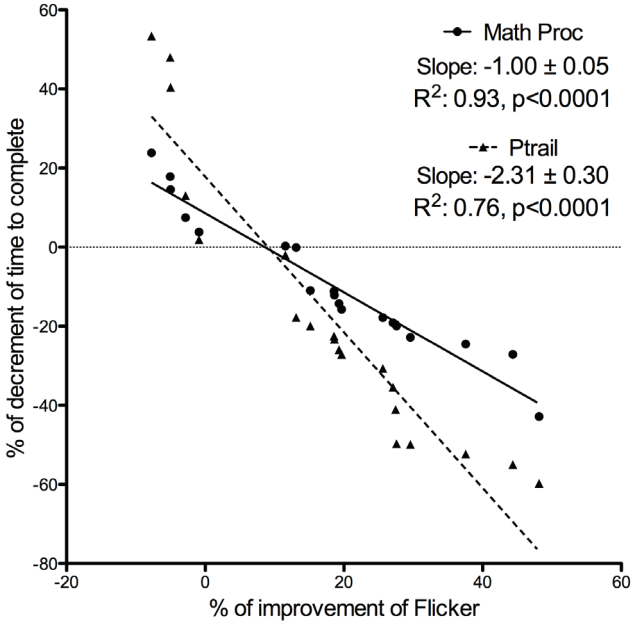
Oxygen breathing affects both time to complete (1.86% of the total variance) and error rate of MathPoc and PTrail (4.58% of the total variance) as there is respectively a 0.43% (F = 8.51, DFn = 1, DFd = 114, $P = 0.0043$) and a 0.008% (F = 11.87, DFn = 1, DFd = 114, $P = 0.0008$) chance of randomly observing an effect this big in an experiment of this size.

The kind of test accounted for 72% of the variance. This suggests that all tests are not equal in detecting a modification of brain performance (F = 164.29, DFn = 2, DFd = 114, $P < 0.0001$). This assumption is logical since we did not observe a significant change in the perceptual vigilance task.

COMPARISON BETWEEN CFFF AND PEBL TEST

The magnitude of CFFF change and the time to complete both the math-processing and trail-making tasks are inversely correlated (Pearson r = -0.9695 and -0.8731 respectively; Figure 2). Since $P < 0.0001$, we can reject the idea that the correlation is due to random sampling. This relation was further confirmed by linear regression.

Figure 2
Correlation calculation and linear regression of the magnitude of CFFF change and time to complete the MathProc and Ptrail tests



Discussion

The environmental characteristics of diving include pressure and breathing various gas mixtures adapted to the planned depth. According to Dalton's and Boyle's Laws, inert gas partial pressure increases with depth, and may generate significant nervous system dysfunction with disturbances of all memory, intellectual operation and locomotor activity.[2] As a consequence, the diver's safety may be impaired. Therefore, there is a need for an objective, valid and reliable measurement tool to evaluate brain performance in divers. Ideally, these indices should be reproducible, less subject- or investigator-dependent than a psychometric behavioural approach, based on observing a change in neurological parameters like EEG measurement, but also easy to implement underwater. Unfortunately this tool does not seem to exist unless we consider the use of CFFF, as has been done in several studies.[7,8,10,11] However, CFFF has never been validated or correlated to an independent, reliable set of brain-performance evaluations. Since there are two effects to consider (pressure and the nature of the gas breathed), the present study focuses on the easiest condition to test first – the effect of the mixture breathed under normobaric conditions – in order to later differentiate this effect from those of pressure or environment.

Normobaric hyperoxia has been shown to accelerate nerve conduction and although the exact mechanism is still debated, we would expect to see an improvement in brain performance. The results of the MathProc and Ptrail tests and the changes in the CFFF support this. Also, the regression graph shows a significant inverse correlation between CFFF and the time to completion of these tasks, suggesting that these tests might be considered comparable in providing assessment of cortical functions.

This may be because of the neural pathways involved in these processes. When experiments combine neural and mental chronometry, the contribution of perceptual and motor processes to the duration and variability of behavioural reaction time must be taken into account. Whereas perceptual processing accounts for a relatively constant amount of time for a given stimulus condition, the processes of mapping particular stimuli onto the appropriate behaviour and preparing the motor response provide flexibility but introduce delay and variability in reaction time.[24] Therefore, one of the causes for the difference in the reaction time could obviously be the nature of the task itself.[25] There are probably fewer processing stages for automatic attention to act upon in simple tasks (PVT) than in complex ones or in mechanisms less sensitive to automatic attention (MathProc and Ptrail). The idea that very different mechanisms mediate simple and complex tasks is certainly not new.[26] Also, automatic attention mechanisms could be set to operate at a lower gain level in simple tasks than in complex tasks as a consequence of the usual particular demands of these tasks. There is evidence for an adjustment of automatic attention mechanisms to task demands.[27,28]

Finally, there could be less room for automatic attention to reduce reaction time in simple tasks than in complex tasks because of the greater previous preparation to process the target stimulus in the former tasks than in the latter ones.[29] In simple terms, any increase in the number of synapses involved in a certain task would account for increased response time – each synaptic connection adding a delay of approximately 1 ms. The neural pathway for the PVT tests might involve fewer neural connections. Therefore, even if the conduction between neurons is increased because of the increased partial pressure of oxygen, this effect will not be noticeable since the number of synaptic connections involved is too small. Conversely, if many neural connections are involved, the integration over the whole path will yield a shorter time for the test, as shown for Ptrail and MathProc or a higher CFFF.

Conclusion

We conclude that the CFFF test provides an assessment of cognitive function that is similar to some tests from PEBL but requires a less complicated set up and could be used under various environmental conditions. Using CFFF, it would thus be possible to conveniently measure cognitive performance underwater. Further research is needed to assess the combined effects of increased pressure and variations in inspired gas mixtures during diving.

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A prospective analysis of independent patient risk factors for middle ear barotrauma in a multiplace hyperbaric chamber

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Abstract

(Commons KH, Blake DF and Brown LH. A prospective analysis of independent patient risk factors for middle ear barotrauma in a multiplace hyperbaric chamber. *Diving and Hyperbaric Medicine*. 2013 September;43(3):143-147.)

Introduction: Middle ear barotrauma (MEBT) is the most common complication of hyperbaric oxygen therapy (HBOT). We wished to determine whether independent risk factors could predict which patients will require tympanostomy tubes in order to continue HBOT.

Methods: Data regarding demographics, medical history and physical examination were collected prospectively over one year. Multivariate logistic regression was used to analyse the data.

Results: One hundred and six patients were included. The cumulative risk of MEBT over the first five treatments was 35.8% and that for needing tympanostomy tubes was 10.3%, while that for needing tubes at any time was 13.2%. Risk factors for MEBT on bivariate analysis were older age, history of ENT radiation and anticoagulant use. Risk factors for requiring tympanostomy tubes included a history of cardiovascular disease and patients being treated for an infective condition. The adjusted multivariate logistic model identified history of difficulty equalising as the only characteristic significantly associated with MEBT during the first five treatments, adjusted odds ratio (AOR) (95%CI): 11.0 (1.1 – 111.7). Being female, AOR (95%CI): 24.7 (1.8 – 339.7), and having a history of cardiovascular disease, AOR (95%CI): 20.7 (2.0 – 215.3), were significantly associated with the need for tympanostomy tubes during the first five HBOT, but there was no significant association between any other characteristics and the need for tubes at any point.

Conclusion: Despite some significant risk factors for MEBT being identified, we were unable to predict accurately enough which patients needed tympanostomy tubes during their HBOT to recommend these being placed prophylactically in selected patients.

Key words

Ear barotrauma, hyperbaric oxygen therapy, risk factors, hyperbaric research, morbidity

Introduction

The most common and easily identifiable complication of hyperbaric oxygen therapy (HBOT) is middle ear barotrauma (MEBT).[1] MEBT occurs when there is a change in the ambient atmospheric pressure accompanied by an inability to equalise the pressure in the middle ear with the new atmospheric pressure. This process of equalisation occurs by the active opening of the Eustachian tube linking the nasopharynx to the middle ear, allowing the passage of air and therefore equalisation of pressure. It is abnormalities of anatomy and function along the Eustachian tube and poor equalization technique that lead to MEBT.

Pain is the primary symptom of MEBT. Complications of MEBT include otalgia, haemorrhage into or rupture of the tympanic membrane (TM), ossicular chain disruption and potentially conductive and/or sensorineural hearing deficit. More serious complications occur rarely.[2,3]

Previous studies have reported incidences of HBOT-related MEBT ranging from 8% up to 94% in specific populations.[4–14] This inconsistency in the reported rate of MEBT is thought to be multi-factorial and may include differing inter-observer experience and interpretation of grading and the use of different grading scales between centres.[15] Reported risk factors for HBOT-related MEBT include age over 61 years or less than 40 years, female gender, prior evidence of Eustachian tube dysfunction, patients with artificial airways and patients undergoing HBOT for delayed radiation injury of the nasopharynx.[5,8–12,16]

The Hyperbaric Medicine Unit (HMU) at the Townsville Hospital (TTH) receives referrals from throughout North Queensland, covering a population of greater than 650,000 people over an area of more than 600,000 km^2. Approximately 200 patients, with a wide variety of conditions, are referred each year. On average, each patient receives 30 daily treatments of HBOT. The current practice at TTH at the time of this study was that patients who were unable to successfully equalise pressure in the middle ear had placement of tympanostomy tubes performed under a general anaesthetic. Although a prioritized procedure, the process of referral for tympanostomy tubes to the Ear, Nose and Throat (ENT) Clinic and then awaiting access to theatre time can sometimes take several weeks or even months.

The aim of this prospective study was to determine whether a number of independent risk factors could predict which patients would require tympanostomy tubes. If possible, this would allow for prophylactic tube insertion, and could significantly reduce interruption and delay to HBOT and inconvenience to patients.

Methods

Approval for the study was granted by The Townsville Health Service District Human Research Ethics Committee. Every patient who received HBOT at TTH between 01 June 2009 and 31 May 2010 was enrolled into the study. The exclusion criteria were: age < 18 yrs; non-English speaking (or no interpreter available); and patients who already had tympanostomy tubes.

Demographic data were collected prospectively from the documented past medical history and physical examination findings recorded by the admitting medical officer for all patients prior to commencing HBOT. These data included: age; gender; condition requiring HBOT; use of analgesics; a history of diabetes, cardiovascular or respiratory disease; psychiatric illness; other significant illness; smoking history; history of facial/ENT surgery; history of facial/ENT radiation; previous equalisation problems; previous scuba diving experience and previous HBOT problems.

Otoscopic examination of the TM was performed prior to commencing HBOT, following each of the first five treatments and as required after that. The Valsalva manoeuvre was described to all patients prior to commencing treatments and otoscopic examination was performed whilst the patient attempted to Valsalva to determine whether TM movement was visible or not. Dynamic assessment of TMs was not performed as it was not part of the standard pre-HBOT assessment at TTH at the time of the study.

TTH uses a triple-lock hyperbaric chamber (Fink Engineering Pty Ltd, Warana, Queensland, Australia) pressurised with compressed air. The majority of patients were compressed to 243 kPa at a rate of 14 kPa min^{-1}. Treatment at this pressure runs for 80 minutes with patients breathing 100% oxygen via a closed-circuit head hood. Two 5-minute air breaks occur during the treatment, and decompression occurs at a rate of 9.5 kPa min^{-1}. Other treatment tables used (e.g., for divers with decompression illness) included RN62 and Comex 30.

During treatments, patients were asked to report any symptoms of MEBT, and additional otoscopic examinations were performed during pressurisation by the inside chamber attendant, as indicated. If symptoms or signs of MEBT were found during pressurisation, they were managed as per current practice, including slowing the rate of compression, a trial of topical decongestants or cessation of treatment. MEBT was graded on a scale of 0 to 5 using the Edmonds classification (Table 1), which is interchangeable with the modified Teed scale, and is standard in most hyperbaric units in Australasia.[17,18]

The primary analysis was multivariate logistic regression, with barotrauma (yes/no) as the dependent variable and the demographic characteristics, history and physical findings as independent variables. Secondary endpoints included the need for tympanostomy tube placement at any point during the first five HBOT sessions, and the need for tube placement at any point during therapy.

Table 1
The Edmonds scale for middle ear barotrauma

Grade	Criteria
0	Symptoms with no signs
1	Injection of the tympanic membrane (TM)
2	Injection of the TM plus slight haemorrhage within the substance of the TM
3	Gross haemorrhage within the TM
4	Free blood in the middle ear, as evidenced by blueness and bulging
5	Perforation of the TM

Results

During the study period, 108 adult patients underwent HBOT at TTH; after excluding two subjects who already had tympanostomy tubes in place, 106 subjects were included in our analysis. The subjects were mostly males (67%), with a median (range) age of 62.0 (18–86) years. The incidence of MEBT after the first treatment was 22.6%, with a cumulative risk of 35.8% over the first five treatments, and 43.4% at any point during treatment.

The cumulative risk of needing tympanostomy tubes during the first five HBOT treatments was 10.3%. The cumulative risk of needing tubes at any time during the HBOT treatment regimen was 13.2%. Table 2 shows the demographic characteristics and indications for HBOT of subjects with and without MEBT, as well as those requiring tube placement within the first five treatments or at any time during their treatment.

On bivariate analysis, there was a positive association of developing MEBT with advancing age, history of ENT radiation therapy and anticoagulant use. Bubble-related indications, documented TM movement with Valsalva and scuba experience appeared protective against MEBT. Patients with a history of cardiovascular or psychiatric disease, patients with a tracheostomy tube in place and patients being treated for an infective condition were more likely than others to require tube placement at some point during their treatment.

In the unadjusted model, having a bubble-related indication was associated with a decreased risk of MEBT during the first five treatments (odds ratio (OR) = 0.07). However, after controlling for confounding effects of other variables, this association was negated, (Adjusted odds ratio (AOR) (95% confidence intervals, CI): 0.13 (0.01–2.7); and difficulty with equalising ears was the only patient characteristic significantly associated with MEBT during the first five

Table 2
Association between patient demographics, indication for HBOT and middle ear barotrauma (MEBT); numbers of patients (%) experiencing MEBT or requiring tympanostomy tubes

	All subjects Day 1–5	No MEBT	MEBT		Tympanostomy tubes
			Rx 1–5	At any time	
	(*n* = 106)	(*n* = 68)	(*n* = 38)	(*n* = 10)	(*n* = 14)
Characteristic					
Mean age	57.2	53.0	64.5	62.4	62.6
95% CI	53.8–60.7	48.4–57.5	59.6–69.4	52.7–72.2	55.3–70.0
Male	71 (67.0)	47	24	5	7
Female	35 (33.0)	21	14	5	7
Indication for HBOT (some patients have multiple indications)					
Bubble injury	20 (18.9)	19	1	0	0
Infective condition	10 (9.4)	5	5	3	4
Radiation tissue damage	48 (45.3)	26	22	5	8
Wound problem	21 (19.8)	14	7	2	2
Other	7 (6.6)	4	3	0	0

Table 3
Multivariate logistic regression models for patients having HBOT who experienced middle ear barotrauma (MEBT) and those requiring tympanostomy tubes (within the first five treatments or at any time); only variables that reached or approached statistical significance are shown

Dependent variable	Independent variables	Odds ratio	95% CI odds ratio		*P*–value
			Lower	Upper	
MEBT	Difficulty equalising	11.0	1.1	111.7	0.042
Tympanostomy tube (Rx 1–5)					
	Female	24.7	1.8	339.7	0.016
	Cardiac history	20.7	2.0	215.3	0.011
	Documented TM movement	0.1	0.01	1.1	0.063
	Difficulty equalising	21.9	0.7	724.1	0.084
Tympanostomy tube (at any point)					
	Infectious indication	6.7	0.97	45.6	0.053

treatments, AOR (95% CI): 11.0 (1.1–111.7). Although intuitive, this finding should be viewed with some caution given the small number of patients who had difficulty with equalisation (*n* = 9). Table 3 shows the results of the multivariate logistic regression modelling.

Being female (OR = 9.9), a history of cardiovascular disease (OR = 25.2) and a documented immobile TM (OR = 0.07) were associated with the tube placement during the first five treatments in the unadjusted model. After adjusting for confounding variables, only being female, AOR (95%CI): 24.7 (1.8–339.7) and having a history of cardiovascular disease, AOR (95%CI): 20.7 (2.0–215.3) remained significantly associated with the need for tympanostomy tube placement during the first five treatments. There was no interaction effect between these two variables.

Only an infection-related indication was associated with the need for tube placement at any time during the course of treatment in the unadjusted model (OR = 5.7); however, after adjusting for confounding variables, this association was no longer significant, AOR (95%CI): 6.7 (0.97–45.6).

Discussion

The prevention and management of MEBT during HBOT includes education regarding equalisation techniques, slowing of pressurisation, avoidance of further HBOT until symptoms have resolved and, although the supporting evidence is limited, the use of systemic or topical decongestants.[19,20] The insertion of temporary tympanostomy tubes to create an artificial passage for equalisation to occur is a more invasive management option. This treatment has a number of risks, including otorrhoea, otalgia, infection, decreased hearing, persistent TM perforations and tinnitus.[3] Whilst MEBT rarely results in the cessation of HBOT, anecdotal reports from our region are that all of these management approaches can result in interruptions and delays to HBOT regimes.[15]

In Australia, HBOT is mainly available in hospitals located in large cities and regional centres.[21] Many patients live too far away from the hospital to permit daily travel for HBOT, resulting in a large proportion of patients having to relocate themselves and family members and needing to arrange long breaks from their usual occupations. Delays during HBOT,

therefore, can be both socially and financially costly for some patients. In our study the average delay to treatments in those patients with MEBT was 10 days, with a maximum delay of 40 days waiting for tympanostomy tube placement. Sadly this happened to be a patient who had travelled from out of town for their course of treatments.

Given our large catchment area for patient referrals and sometimes delayed referral times for tympanostomy tube placement, we chose to collect and analyse a wide list of variables in order to attain the best outcome to assist with managing our patients more efficiently. The ability to predict which patients will require tympanostomy tubes would allow for prophylactic insertion and minimum disruption to treatment and inconvenience to patients. Unfortunately, we could not accurately predict which patients will go on to suffer MEBT and which will need tubes. However, some key points should be noted.

Our reported total cumulative incidence of MEBT of 43.4% is higher than many other published rates.[4–14] This may be because the Edmonds classification has a lower threshold for diagnosing MEBT since it grades symptoms without signs (Grade 0), which the original Teed scoring system does not.[21] The incidence of MEBT in our study population is also higher than the 13.6% reported in a study published just after our data collection had been completed.[4] This probably reflects the different population groups in the two studies: MEBT in the acute setting versus mainly non-acute patients with radiation tissue damage and wound problems in our population. This is also reflected in the different average ages between the two studies: 37.5 years versus 62.0 years in our study.

Patients being treated for bubble injury, those in whom TM movement can be visualised, and those with scuba experience appear less likely to suffer MEBT or to need tubes. 18.9% of the patients in this study were treated for bubble injury, all of which were diving-related and not iatrogenic. This suggests that the knowledge and experience of TM movement that comes with scuba diving is a protective factor.

In previous studies, patients with delayed radiation injury in the head and neck region were at increased risk of suffering MEBT or of requiring tympanostomy tubes.[16,22] Our study only partially supports this in that patients who had previously undergone ENT radiation treatments were more likely to suffer MEBT at some point during their first five HBOT, but they were not more likely to require tube placement.

When controlling for other factors, the best predictor of MEBT during the first five treatments was difficulty equalising ears (AOR = 11.0), but the small number of patients (nine) with such difficulty means this statement should be viewed with caution. This finding does, however, correlate with previous studies. MEBT correlates positively with an immobile TM on otoscopy during the Valsalva manoeuvre; patients who are unable to autoinflate the middle ear have been reported to have a higher incidence and greater severity of barotrauma than patients who are able to autoinflate.[7,10]

When controlling for all other factors, although women were at increased risk of requiring tube placement during their first five treatments compared to men (AOR = 24.7, five of 35 women), other similar-sized studies on gender as a risk factor for MEBT have been conflicting, suggesting that this is not a reliable predictor.[4,9] The most striking (and non-intuitive) finding, was the association between a history of cardiovascular disease and the need for tube placement during the first five treatments. Again, however, this increased risk is relative; only seven of 35 patients with a cardiovascular history required tubes and, when looking at whether patients ever needed a tube, including treatments beyond the first five, this association disappears. An interesting recurring theme was the relationship between MEBT and delayed radiation to the nasopharynx. Given the high numbers of these patients treated with HBOT, further studies could be aimed specifically at these patients to look at differences between total radiation doses, midline versus asymmetric radiation, and length of time since radiation.

All three of our patients with artificial airways required tubes. Having an artificial airway in place during HBOT has been identified previously as a risk factor for MEBT.[12] Of 28 patients with artificial airways in a study of 267 patients, 27 required tympanostomy tubes.[12] Our centre certainly does not treat sufficient patients with artificial airways to draw any useful conclusions, but it seems logical to prophylactically place tubes in these patients.

Since this study was completed, practice has changed at TTH and tympanostomy tubes are now being placed under local anaesthesia, a less time-consuming process, tolerated by patients. Staff confidence and ability to assess and grade MEBT has increased since completing the study. Audit of MEBT is part of the Australasian-wide documentation of clinical indicators by the Hyperbaric Technicians and Nurses Association. Another limitation to our study may have been the variability in accurately grading the degree of MEBT given that it is a subjective assessment and that we asked all chamber attendants and medical officers to participate. Pre-study education and reference material as well as ongoing supervision was provided by the authors.

Conclusions

Patients with a scuba diving history and those whose TMs could be visualized to move on otoscopic examination have decreased risks of MEBT and the need for insertion of tubes. Although these two factors appear to be protective, other risk factors for MEBT are more difficult to quantify. We cannot accurately predict which patients will require tubes

during their HBOT and, therefore, are unable to recommend prophylactic placement. On multivariate analysis, only difficulty equalising was a risk factor for MEBT and being female and having a history of cardiovascular disease were risk factors for early tube insertion. Infectious indications for HBOT may be a risk factor for tube placement at any time during HBOT. These groups of patients should be monitored closely during their course of HBOT to enable early diagnosis and intervention where appropriate.

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Review article

Effects of diving and oxygen on autonomic nervous system and cerebral blood flow

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Abstract

(Winklewski PJ, Kot J, Frydrychowski AF, Nuckowska MK and Tkachenko Y. Effects of diving and oxygen on autonomic nervous system and cerebral blood flow. *Diving and Hyperbaric Medicine*. 2013 September;43(3):148-156.)
Recreational scuba diving is a popular leisure activity with the number of divers reaching several millions worldwide. Scuba diving represents a huge challenge for integrative physiology. In mammalian evolution, physiological reflexes developed to deal with lack of oxygen, rather than with an excess, which makes adaptations to scuba diving more difficult to describe and understand than those associated with breath-hold diving. The underwater environment significantly limits the use of equipment to register the organism's functions, so, in most instances, scientific theories are built on experiments that model real diving to some extent, like hyperbaric exposures, dive reflexes or water immersion. The aim of this review is to summarise the current knowledge related to the influence exerted by physiological conditions specific to diving on the autonomic nervous system and cerebral blood flow. The main factors regulating cerebral blood flow during scuba diving are discussed as follows: 1) increased oxygen partial pressure; 2) immersion-related trigemino-cardiac reflexes and 3) exposure to cold, exercise and stress. Also discussed are the potential mechanisms associated with immersion pulmonary oedema.

Key words
Scuba diving, cerebral blood flow, oxygen, autonomic nervous system, neurogenic pulmonary oedema, immersion pulmonary oedema

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Introduction

Recreational self-contained underwater breathing apparatus (scuba) diving is a popular leisure activity. It is not easy to provide any estimates related to the number of scuba divers worldwide; however, some diving portals state 3–6 million; obviously, it is a large and growing population. When conducted within recreational limits (concerning depths, times, types of breathing mixtures, decompression burden, and environmental factors), the risk for injury or death is quite low. The annual per capita fatality rate ranges from 1.7 to 16.4 deaths per 100,000 persons per year depending on surveillance programmes, which is no higher than the rate for jogging or motor vehicle accidents (13 and 16 deaths per 100,000 persons per year, respectively).[1] Divers Alert Network (DAN) is a dive organization that publishes annual accident reports:
<http://www.diversalertnetwork.org/medical/report/>.

There are many reports describing the consequences of diving injuries, decompression illness and the high pressure neurological syndrome.[2,3] Some extreme environmental factors, like cold water and deep scuba diving, may have long-term negative effects on cerebral blood flow (CBF) and neurological function with consequent intellectual deterioration.[4] The possible neuropsychological effects of deep diving have been highlighted by other authors.[5,6] In this review, we will focus mainly on uneventful dives and their effect on the pathophysiology of the human body, concentrating on the autonomic nervous system (ANS) and CBF.

The main factors affecting the ANS and CBF during scuba diving include: increased oxygen partial pressure (PO_2), immersion-related trigemino-cardiac reflexes and exposure to cold, exercise and stress. All of these act through different physiological pathways, but they are cross-linked with variations and imbalances in ANS activity. The exact role that the ANS plays in the regulation of CBF remains a matter of scientific discussion, even outside the diving world. It is interesting to present how diving-related factors influence the ANS and consequently its contribution to the regulation of CBF during uneventful recreational diving. With the increasing popularity of scuba diving in populations with less than optimal health status (i.e., age, physical unfitness and mild cardiovascular disturbances) or autonomic imbalance (e.g., adolescents), such knowledge is of even greater importance. A better understanding of the role that the ANS and oxygen play during scuba diving may help to develop more efficient strategies in the treatment of several life-threatening diseases that are not related to diving activities, such as stroke, brain trauma or pulmonary hypertension.

ANS in the regulation of CBF

Before discussing the main factors affecting the ANS and CBF during scuba diving, we provide a brief summary of

the current knowledge with respect to the role of the ANS in CBF regulation in other conditions. This is a large subject and readers are referred to two review articles for more detailed information.[7,8]

The sympathetic (SNS) and parasympathetic (PNS) nervous systems function in opposition to each other. The SNS typically functions in actions requiring rapid responses, whilst PNS functions with actions that do not require immediate reaction. The SNS is often considered the "*fight or flight*" system, while the PNS is often considered the "*rest and digest*" or "*feed and breed*" system. Therefore, the SNS is activated by exercise, cold and anxiety to divert blood flow away from the gastro-intestinal tract and skin (via vasoconstriction) to brain, heart, skeletal muscles and the lungs. In addition, SNS activation increases heart rate and myocardial contractility, further enhancing blood flow to brain and skeletal muscles.

Brain blood vessels are richly innervated with parasympathetic fibres.[9,10] Acetylcholine, the postganglionic neurotransmitter of parasympathetic neurons, interacts with endothelial muscarinic receptors to facilitate vasodilation.[11,12] The release of acetylcholine from the animal cerebral cortex contributes to the exercise-induced increase in CBF.[13] Muscarinic receptors are present in human cerebral arteries, including the middle cerebral artery, and acetylcholine induces vasodilation in human cerebral arterioles by stimulating the brain subtype of these receptors.[11,14] Evidence for vasodilation of cerebral vessels by nerves releasing acetylcholine has been reported recently in humans, with an increase in CBF velocity during exercise being abolished with muscarinic receptor blockade.[15] Atropine, an acetylcholine antagonist, decreases pial artery pulsation in humans (unpublished observations).

The cerebral arteries, arterioles and, to a lesser extent, veins are richly innervated with sympathetic nerve fibres.[16,17] However, the role of the SNS in regulating cerebral circulation remains a matter of controversy.[18–20] Adrenergic receptors have been found in human cerebral vessels, including the middle cerebral artery, and their activation leads to contraction of human pial artery segments.[21,22] Only recently has it become possible to assess pial arteries in intact animals and humans. Infusion of noradrenalin (the SNS neurotransmitter) constricts pial arteries in animals, while static exercise exerts similar effects in humans.[23,24] There is accumulating evidence that the SNS might be an important element in brain protection against excessive increases in perfusion pressure and flow.[25–28] Moreover, recent studies employing techniques based on infrared light have suggested an increase in cerebral venous blood volume following sympathetic stimulation.[29]

Increased oxygen partial pressure (hyperoxia)

Oxygen partial pressure (PO_2) depends on the fraction of oxygen in the inspired gas mixture and ambient pressure (diving depth). In recreational diving, PO_2 is kept below 162.6 kPa (1.6 ATA) in order to avoid its toxic effect on the central nervous system (CNS). The ANS response to increasing PO_2 is biphasic, with the PNS predominating initially.[30] Massive sympathetic discharge originating from the CNS is seen only at very high PO_2, usually leading to acute intoxication and, while it has been described in animals, it has not been reported during conventional recreational scuba dives.[31–33]

It remains controversial whether or not, during the initial response to oxygen, the SNS is also activated. Interesting data comes from outside diving-related research, namely from a functional magnetic resonance imaging study in children aged from 8 to 15 years. Two minutes of hyperoxic ventilation at sea level (101.3 kPa inspired O_2) produced pronounced responses in the central autonomic and hormonal control areas, namely the posterior hypothalamus, insula, hippocampus, cerebellum, caudate, and thalamic regions, which are brain structures that are usually recognised as intertwined with the central SNS.[34] This clearly showed that even very short exposure to hyperoxia may affect several structures in the CNS. However, this study should be interpreted with caution because the CNS in children is less mature than that of adults and may respond differently to hyperoxia. Also, it is not known if activation of these regions resulted in any peripheral changes, i.e., SNS efferent firing, changes in hormone levels or cardiovascular alterations. Several studies in adults report a decrease in SNS activity in the PO_2 range below 162 kPa or lack of relevance of SNS activity on peripheral changes.[35–37]

Breathing oxygen-enriched gas mixtures under normobaric conditions (PO_2 less than 101.3 kPa) leads to peripheral vasoconstriction, and decreased heart rate, stroke volume and cardiac output, changed baroreflex sensitivity, reduced carotid artery diameter and CBF, and increased brain tissue oxygen saturation.[37–42] Peripheral blood pressure either does not change or increases.[31,37,42] Based on animal research, it is suggested that hyperbaric oxygen may exert different effects on the left and right ventricles.[42] While function of the left ventricle is depressed, the right ventricle may be less affected, which in turn may lead to pulmonary hypertension and increased pulmonary arterial wedge pressure.

Hyperoxia induces peripheral vasoconstriction. Several different explanations have been proposed for this, including inhibition of the local release of nitric oxide (NO) from cysteine-binding in the haemoglobin molecule (S-nitrosothiol) by increased venous PO_2 and superoxide anions and inactivation of endothelium-derived relaxing factor by hyperoxia.[43,44] Several other factors may be involved, but this vasoconstriction seems not to be associated with increased SNS activity.[36]

Decreases in heart rate, stroke volume and cardiac output occur as a result of two overlapping factors: 1) lowering of the heart rate, triggered by an increase in PNS activity;

and 2) impairment of left ventricular diastolic function.[42,45] Oxidative stress due to exposure to an oxygen-rich environment is well-documented in humans and animals.[46,47] On returning to normoxic conditions, the time course of recovery is different for various cardiovascular variables. Heart rate is the first to return to baseline, while the vascular resistance, cardiac output and stroke volume changes persist for longer. Such a sequence of events supports the above reasoning: 1) the recovery of cardiovascular function is not controlled solely by baroreflex activity but there are two distinct phenomena: central negative chronotropic action and local vasomotor control; and 2) cardiac output and stroke volume reduction could be strongly attributed to the impairment in left ventricle function.[30]

Hyperoxia-induced alterations in cardiac function need further consideration with respect to brain perfusion. The clinical and experimental literature suggest a close link between the function of the left ventricle and brain perfusion.[48–50] The CBF response to a rapid decline in systemic blood pressure in healthy volunteers was closely related to unloading of the arterial baroreceptors, which suggests cardiac output involvement in the regulation of CBF.[49] In that study, during full cardiac autonomic blockade, an attenuated tachycardia response exacerbated the cuff release-induced reductions in mean systemic blood pressure, and consequently evoked a greater transient decrease in mean CBF velocity. These findings indicate that the baroreflex-induced tachycardia response following acute hypotension regulates the reduction in systemic blood pressure, thus acting to minimise decreases in CBF.[49]

Data coming from our lab indicated direct interactions, independent of systemic blood pressure, between cardiac output and brain microcirculation, at least in animals.[50] To the best of our knowledge, there have been no studies assessing brain and heart interdependence during hyperoxia and the time course of recovery. If we hypothesise that such interdependence exists, the initial activation of the PNS may be seen as a regulatory mechanism to preserve brain homeostasis, i.e., it protects the CNS from O_2 intoxication.

Hyperoxia per se induces a decrease in CBF.[38,40] This was elegantly shown in a study in which healthy subjects were exposed to various ambient pressures (101.3 kPa, 202.6 kPa and 405 kPa) while breathing either air or 100% oxygen.[38] Oxygen inhalation reduced middle cerebral arterial blood flow velocity while ambient pressure per se did not seem to influence it. Unfortunately there was no comparison between lower and higher ambient pressures (101.3 kPa versus 405 kPa) while breathing the same and normoxic PO_2 (20 kPa). Therefore, PO_2 remains the main factor regulating CBF, but the effects of other confounders, including ambient pressure, need to be further elucidated.

The mechanism of oxygen-induced cerebral vasoconstriction is based on an interruption in NO-mediated basal relaxation of cerebral vessels.[51,52] Hyperoxia leads to the generation of reactive oxygen species like superoxide ($\cdot O_2$), which can react with NO to generate the strong oxidant peroxynitrite, resulting in transient reduction in its perivascular concentration.[51] The equilibrium between $\cdot O_2$ and NO is regulated by superoxide dismutase-3.[51] Superoxide dismutase-3 is present in high concentrations in vessels where NO is important for vascular relaxation. Inactivation by $\cdot O_2$ interferes with NO-dependent basal tone and vasorelaxation. Therefore, scavenging of $\cdot O_2$ by superoxide dismutase-3, plays a critical role in regulation of NO-dependent CBF.[53] As a result, the reduction in CBF caused by hyperoxia protects the brain against an excess of oxygen.

When hyperoxia persists after an initial fall in CBF, the production of NO appears to compensate and may result in an increase of CBF and, as a consequence, the delivery of toxic amounts of oxygen to the brain. Such secondary CBF increase always precedes O_2-induced manifestations of brain poisoning, both in animals and in humans.[51–55] However, the secondary increase in CBF and O_2-induced seizures were reported at very high oxygen doses (283 kPa and 506.5 kPa of inspired O_2 in humans and animals, respectively), therefore, significantly exceeding even the most liberal limits set for recreational scuba diving.

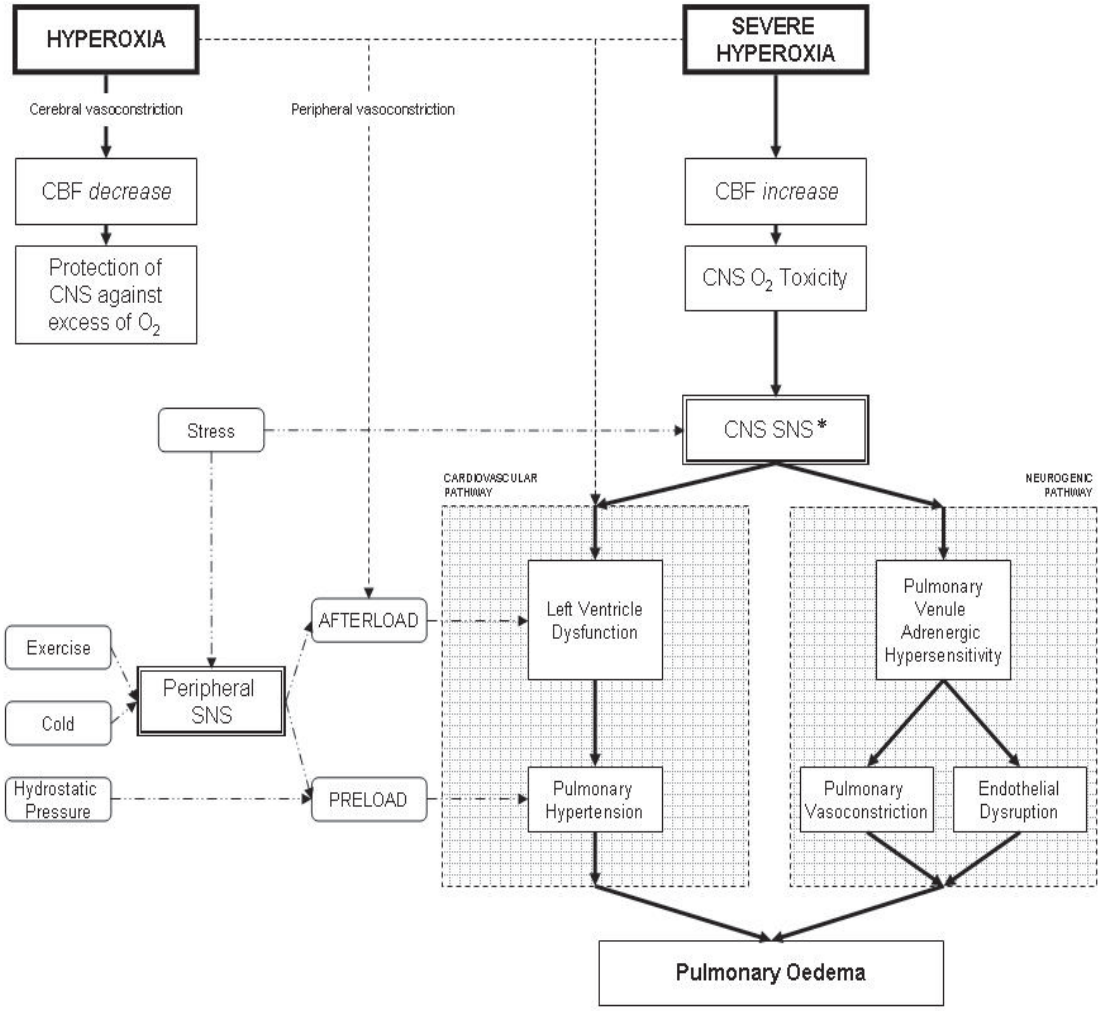
Nevertheless, doses of oxygen that induce CNS oxygen toxicity also activate the SNS. Intense SNS discharge and the release of catecholamines have been reported in brain insult irrespective of the causality, in particular, in neurologic conditions that cause abrupt, rapid and extreme elevations in intracranial pressure.[56,57] It is hypothesised that massive SNS discharge following CNS injury, for example, directly affects the pulmonary vascular bed via α- and β-adrenergic receptors, leading to isolated pulmonary venoconstriction and/or endothelial disruption. This theory of "*pulmonary venule adrenergic hypersensitivity*" can explain neurogenic pulmonary oedema.[58]

In diving, the CNS symptoms of oxygen toxicity (generalised convulsions) and pulmonary damage induced by hyperbaric O_2 have long been considered to be separate entities, with oxygen acting directly on the CNS in the first case and on the alveolar region of the lung in the second.[59] Involvement of the SNS, as a link to both events, was initially proposed in animal models, but has not been seen in human studies.[32,60,61] It remains unclear why oxygen brain poisoning leads to neurogenic pulmonary oedema in small animals, but not in humans.[31,32,60,61]

Immersion pulmonary oedema

Immersion pulmonary oedema (IPE) represents a different mechanism of lung injury than neurogenic pulmonary oedema caused by a significant CNS insult. Whilst the ANS is involved in both syndromes, in the case of CNS oxygen toxicity, lung inflammation and possibly Takotsubo cardiomyopathy (a form of acute left ventricular dysfunction

Figure 1
Pathways of oxygen and SNS involvement in development of pulmonary oedema;
* extrapolated data from animal studies



characterized by normal contraction of the apex, and dilatation of the remainder, with a fancied resemblance of the left ventricle to a Japanese octopus pot – 'takotsubo'), this is mediated by massive sympathetic discharges originating from the CNS.[58] On the other hand, in IPE, this is owing to an increase in central venous (preload), pulmonary arterial and pulmonary wedge pressures. This phenomenon is mediated by the cardio-endocrine-renal axis, with immediate translocation of blood to the heart and slower autotransfusion of fluid from the cells to the vascular compartment.[62] The question of whether central SNS activation may facilitate development of IPE during some dives with strenuous exercise in cold water remains unanswered. Figure 1 provides a graphical summary of the various pathways of oxygen and SNS involvement in the development of pulmonary oedema. Note that the SNS pathway leading directly to neurogenic pulmonary oedema has been confirmed only in small animals.

In 22 healthy divers who suffered from IPE after dives involving strenuous exercise, physiological and/or mental stress, the common feature was the occurrence of respiratory symptoms during the ascent.[63] Most of the dives were deep (37 metres' sea water, (msw) on average) and in cool water (15°C).[63] The average inspired PO_2 was 100 kPa, thus within the range that potentially may activate brain structures that are associated with the central SNS.[34] Some divers reported an unpleasant cold sensation, hard effort and/or anxiety. Anxiety is another potential trigger for central SNS activation.[64] It was proposed that the development of lung oedema was associated with decreased heart rate and ventricular contractility and peripheral vasoconstriction due to high oxygen exposure. In five divers, increased troponin levels were found within 24 hours after immersion and, in nine divers, elevated natriuretic peptide levels were considered to reflect congestive heart failure.[63] Both markers (troponin and natriuretic peptide) might have indicated left ventricle dysfunction due to central sympathetic hyperexcitation, but it is not possible to differentiate this from left ventricle dysfunction caused by the direct effect of hyperoxia.[64] IPE in scuba divers in cold waters, first described by Wilmshurst, is probably of haemodynamic

origin.[65–67] Immersion-induced increases in pulmonary blood volume and pulmonary hypertension due to exertion most likely have an additive effect.[68] Interestingly, breathing with hyperoxic gas attenuates high pulmonary pressure evoked by exercise in thermo-neutral water but not in cold water.[69] This suggests that SNS involvement triggered by cold-water immersion promotes pulmonary hypertension. Owing to the complexity of interrelated factors like temperature, exercise, anxiety, PO_2 and probably several other components, interaction between the peripheral and central SNS on the cardiovascular system during diving remains unclear.

Immersion-related trigemino-cardiac reflexes

The trigemino-cardiac reflex, or "*dive reflex*", represents the most powerful of the so-called oxygen-conserving autonomous reflexes in mammals.[70–74] Apnea with bradycardia is associated with a slightly smaller reduction in arterial oxygen saturation than apnea without bradycardia or with less pronounced bradycardia. The trigemino-cardiac reflex was first reported during surgery in the cerebellopontine angle.[75] It was observed that electrical, mechanical or chemical manipulation of the trigeminal nerve on its intra- and extra-cranial course provoked a drop in mean arterial blood pressure and bradycardia.[75,76] The exact mechanism of the trigemino-cardiac reflex is far from clear. In fact, it consists of many sub-reflexes, for example the dive reflex, nasopharyngeal reflex and oculo-cardiac reflexes, which are well described and frequently observed during diving. It seems that the trigemino-cardiac reflex is dominant and the efferent effects can strongly affect human physiology.[77,78]

During immersion, the trigemino-cardiac reflex is elicited by contact of the face with cold water and involves breath-holding, intense peripheral vasoconstriction, bradycardia and increased mean arterial pressure, thus maintaining adequate oxygenation of the heart and brain at the expense of organs less sensitive to hypoxia.[79] Within seconds of the initiation of such a reflex, there is a powerful and differentiated activation of the SNS and PNS with subsequent elevation in CBF. This increase in CBF is independent of the partial pressure of carbon dioxide.[80]

The increase in CBF, with no changes in the cerebral metabolic rate of oxygen or the cerebral metabolic rate of glucose evoked by the dive reflex, is most likely a neuroprotective adaptation or a type of preconditioning strategy.[78] Reticulospinal neurons of the rostral ventrolateral medulla oblongata are critical for detecting and initiating the vascular, cardiac and respiratory responses to hypoxia and ischaemia.[80] The systemic response to excitation of these neurons includes projections to spinal preganglionic sympathetic neurons and cardio-vagal motor medullary neurons, which results in blood redistribution from the viscera to the brain in response to a challenge to cerebral metabolism.[77,80] Excess blood flow to the brain allows diving mammals to survive for a relatively long time underwater.[81] Owing to their physiological function in diving mammals, the oxygen-conserving autonomous reflexes are gaining increasing attention as a potential preconditioning therapeutic strategy in various neurological conditions associated with neuronal death.[77,78]

Human bradycardia resulting from apneic face immersion is inversely proportional to water temperature within a range determined by the ambient air temperature. Face immersion in cold water after exposure to a high ambient air temperature induces the most pronounced bradycardia.[82] The question of whether face-only breath-hold immersion is a good model for investigating the changes that occur during scuba or even real breath-hold diving remains unanswered. The subject is usually requested to hold their breath, and then immerse their face in a tub of water, and to persist in such a position for a certain period of time.[83] The oxygen-conserving effect of the dive reflex in the immersed diver is the same as that observed in the dry, horizontal simulated diving model.[84] However, in scuba diving, the CBF increase seen in the dive reflex is most likely overridden by exposure to inhaled oxygen during the course of immersion and CBF is reduced to protect the brain from hyperbaric hyperoxia. Furthermore, the nasopharyngeal reflex and oculo-cardiac reflexes are supressed by the use of a diving mask. Nevertheless, simultaneous occurrence of trigemino-cardiac reflex and increased PO_2 in the breathing-gas mixture expose the diver to contradictory influences with respect to CBF and powerful, non-physiological and conflicting ANS stimulus. Moreover, the dive response might be partially involved in the mechanism described above for IPE.[85]

Exposure to cold, exercise and stress

When allowed to breathe, immersion in warm and cold water results in different responses with respect to cardiovascular parameters.[85] Warm-water, whole-body immersion (29–31^{o}C) produces increases in cardiac output, and pulmonary arterial, wedge and central venous pressures, while systemic arterial pressure and heart rate remain unaltered. Cold-water, whole-body immersion (18–20^{o}C) leads to systemic arterial pressure and heart rate increases, while cardiac output, and pulmonary arterial, wedge and central venous pressure increases were more pronounced in comparison to warm water.[85] Face-only immersion in cold water reduces apneic time and stimulates ventilation, predominating over the potential oxygen-conserving and apnea-prolonging effects of the diving response.[86] Sudden, whole-body immersion in ice water is linked to the so-called cold shock response consisting of respiratory gasps, hyperventilation, tachycardia, hypertension and decreased CBF during the first 2–3 minutes.[87] A significant reduction in CBF may be associated with signs of imminent syncope, such as drowsiness, blurred vision and loss of responsiveness, and may be a possible cause of drowning.[87] Adaptation to the response is possible and requires repeated cold immersion. However, even without

Table 1

Summary of changes in heart rate, cardiac output and systemic and pulmonary arterial, central venous and wedge pressures after immersion in warm (29–31OC) and cold (18–20OC) water and exposure to normoxia and hyperoxia at 476 kPa in warm water (29–31OC). * compared to baseline (before immersion); † compared to immersion in warm water; ‡ compared to immersion at 476 kPa (PO2 20 kPa); § at 476 kPa stroke volume was measured, not cardiac output; + increase, - decrease, +/- no change
(summary derived from references 40 and 60)

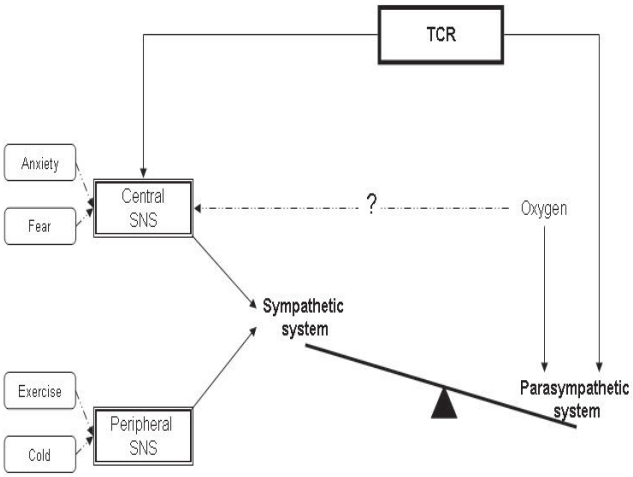
	Warm immersion at rest *	**Cold immersion** at rest†	**476 kPa** (PO_2 20 kPa) during exercise†	**476 kPa** (PO_2 172 kPa) during exercise‡
Heart rate	+/-	+	+	-
Cardiac output §	+	++	++	+/-
Systemic blood pressure	+/-	+	+	-
Pulmonary arterial pressure	+	++	not measured	not measured
Central venous pressure	+	++	++	-
Wedge pressure	+	++	not measured	not measured

prior cold-water experience, subjects, if properly educated, are able to suppress reflex hyperventilation following ice-water immersion and maintain their CBF at a level that is not associated with impaired consciousness.[88] Actually, in experienced scuba divers, noradrenaline and adrenaline blood concentrations may decrease after immersion, most likely because of orthostatic release caused by the external hydrostatic pressure on the peripheral vasculature.[89] This is in line with the fact that novice scuba divers tend to hyperventilate, particularly in stressful conditions, while more experienced divers are able to better control their underwater breathing depth and frequency. The cold-shock response is usually considered with respect to a change from a warm ambient temperature to immersion in cold water. However, a significant temperature change may also occur underwater, when a diver crosses a thermocline and/or swims into a cold current. In experimental conditions, temperature switches from 29–31^{O}C to 18–20^{O}C during immersion at sea level result in increased systemic and pulmonary arterial pressures, while heart rate, central venous pressure, stroke volume and systemic arterial resistance remain unchanged.[85] Unfortunately, there are no reports on the effect of temperature changes on CBF during diving, but it seems that, in such circumstances, the sudden, significant decrease in temperature may induce changes similar to cold shock with all of its consequences, including additional SNS activity.

Diving is usually associated with exercise. The typical response to exercise at the surface consists of tachycardia, hyperventilation, increased cardiac output and elevated CBF.[90] Exercise during immersion at sea level increases heart rate, cardiac output, and pulmonary arterial and wedge pressures, while central venous pressure remains unchanged, both in warm (29–31^{O}C) and cold (18–20^{O}C) water.[85] Responses to exercise at 476 kPa depend on the inspired PO_2. Exercise at 476 kPa with normoxic PO_2 (20 kPa inspired PO_2) in warm water increases heart rate, blood pressure, central venous pressure, stroke volume and systemic vascular resistance, while hyperoxia at 476 kPa (176 kPa inspired PO_2) in warm water decreases the heart rate, blood pressure and central venous pressure response

Figure 2

Autonomic conflict during uneventful scuba diving
TCR – trigemino-cardiac reflex



to exercise, whilst systemic vascular resistance and stroke volume are not significantly altered versus 20 kPa inspired PO_2.[68] As mentioned before, concomitant hyperoxia does not attenuate the increase in pulmonary vascular pressures associated with cold-water immersion.[69] A summary of changes in heart rate, cardiac output and systemic blood, pulmonary arterial, central venous and wedge pressures after immersion in warm and cold water and exposure to normoxia and hyperoxia at 476 kPa in warm water is provided in Table 1. Face cooling with mist water increases CBF during exercise.[91]

Concluding remarks

Taking into account all of the factors discussed, it is obvious that recreational scuba diving represents an effort for humans and activates both the sympathetic and parasympathetic components of the ANS. To further complicate the picture, there is a strong and conflicting relationship between the SNS and PNS. In fact, the phenomenon of 'autonomic conflict' described for cold-water immersion is most likely significantly exacerbated during scuba diving.[92] Figure 2

provides a graphic summary of this conflicted relationship. Furthermore, increased PO_2 diminishes while the trigemino-cardiac reflex and exercise elevate CBF, exposing the diver to contradictory or conflicting stimuli with respect to brain perfusion. PNS activation and related decreases in cardiac parameters seem to protect the brain from oxygen excess, but at the same time impair the ability of the left ventricle to cope with increased afterload and preload, and increases the probability of IPE. This could be of considerable importance for those individuals who are not physically trained or lack physical activity in their everyday life. Moreover, hyperoxia, exercise, cold and stress present during scuba diving may actually reveal or exacerbate existing but undiagnosed medical conditions.

While individual reflexes and responses to oxygen and immersion have been studied, the overall changes to CBF and the ANS during exposures to a combination of pressure, immersion and hyperoxia such as those seen during typical recreational scuba dives have not been well documented and require further study. Scuba diving represents a huge challenge for integrative physiology. During the evolutionary process, physiological reflexes that have developed to deal with the lack of oxygen underwater, rather than an excess make adaptations to scuba diving more difficult to describe and understand.

In this review, we have summarised the current knowledge related to the selected physiological factors exerted by scuba diving on CBF, with particular emphasis on the role of oxygen and the ANS. We have not addressed here one important issue: given the complex interactions between environment and ANS resulting in a myriad of effects (some contradictory) on CBF and cardiac performance, what will be the effects of many of the medications that our divers of increasing age are now taking, particularly those affecting the renin-angiotensin and cardiac systems?

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Work in progress

Characterization of early thermal burns and the effects of hyperbaric oxygen treatment: a pilot study

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Abstract

(Chong SJ, Kan EM, Song C, Soh CR, Lu J. Characterization of early thermal burns and the effects of hyperbaric oxygen treatment: a pilot study. *Diving and Hyperbaric Medicine*. 2013 September;43(3):157-161.)

Background and aims: Studies investigating hyperbaric oxygen treatment (HBOT) to improve outcome in burns have been inconclusive. In this study, we aimed to characterize early thermal burns injury in adult patients with < 40% total body surface area (TBSA) and to determine the effects of HBOT administered within 24 h to 48 h of a burn injury.

Methods: Seventeen subjects were randomized into control ($n = 9$) and HBOT treatment ($n = 8$) arms. Burn depth, measured by laser Doppler imaging (LDI) and histologically, white blood cell (WBC) count and plasma cytokine inflammatory markers were assessed at 24 h (pre HBOT) and 48 h (post HBOT) post burn, as were immunohistochemistry and microbiology of burns tissue samples at 48 h post burn.

Results: WBC count and serum interleukin (IL)-1β, IL-4, IL-6, IL-10 and interferon-γ were significantly elevated 24 h after burn, but no significant changes in any of these parameters were found with HBOT. HBOT had no significant effect on burn depth. Two HBOT patients and four control patients developed positive bacterial cultures.

Conclusions: Slower than anticipated recruitment resulted in considerably fewer patients than planned being studied. Inflammatory markers were significantly increased at 24 h in patients with < 40% TBSA burn. Early HBOT had no apparent effects on any of the parameters measured in this small pilot study. HBOT may possibly have a broad-spectrum antimicrobial effect worthy of further study. We report our methodology in detail as a possible model for future burns studies.

Key words

Burns, hyperbaric oxygen therapy, Doppler, inflammation, bacteriology

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Introduction

Burn injuries continue to result in long-term morbidity and mortality.[1] The depth of burns is not static and parts of the burn wound may 'convert' (progress to become deeper) over the first three to five days.[2] Three concentric zones of burn injury have been described: irreversibly injured tissue in the zone of coagulation, hypoperfused tissue in the zone of stasis and oedematous tissue in the zone of hyperaemia.[3] Although the zone of stasis is potentially salvageable, it is at risk of necrosis in the event of suboptimal treatment.[4] Burns wound conversion is clinically important but poorly understood.[5] Burns conversion directly affects morbidity and mortality since, as the extent of the burn increases, there is a greater need for burns excision, as well as more hypertrophic infections, sepsis, scarring, contracture and mortality.[6] Hence, a reduction in burns conversion should improve clinical outcomes and length of stay for burns patients. The mainstay of modern burns care currently involves a multidisciplinary approach, including urgent fluid resuscitation, early intensive care, early excision and coverage of the burn wound within the first three to five days.[7,8]

Hyperbaric oxygen therapy (HBOT) has been identified as a possible adjunct to burns management.[9,10] Burns have been associated with impaired microcirculation.[11] It has been reported that hyperbaric hyperoxia reduces microthrombi formation through the inhibition of leukocyte adhesion by inhibiting the activation of intracellular adhesion molecule-1, allowing the maintenance of the microvasculature and prevention of reperfusion injury.[11,12] A Cochrane review recommended that more reliable clinical data from large, randomised controlled trials (RCTs) was required before HBOT could be considered a routine treatment for thermal burns.[13] To our knowledge, there has been no RCT that studies the role of HBOT as an adjunct to modern burns care. Our group aimed to conduct a pilot RCT of HBOT in that context. Our hypothesis was that HBOT would reduce burns conversion through the mechanisms summarised above, the aim being to complete two HBOT sessions prior to early excision on day three post burn. To better understand the mechanism of action of HBOT, we also proposed to measure changes in systemic immunological markers and the immunohistochemistry and microbiology of the burn injury.

Materials and methods

STUDY DESIGN AND PATIENT RECRUITMENT

The study was an un-blinded, prospective randomized trial comparing standard modern burns wound care with the same level of care combined with HBOT given in the first 48 h post burn. The study was approved by the Singapore

General Hospital (SGH) Institutional Review Board (IRB) and registered at: https://register.clinicaltrials.gov (NCT00824551) and conducted according to the principles of the Helsinki Declaration. Following evaluation and informed consent, the subjects were randomized in blocks of 10 using a fixed sequential list generated by computer and sealed in opaque envelopes. The inclusion criteria were:

- age 21–60 years;
- thermal burns injury covering < 40% of total body surface area (TBSA) with areas of deep dermal/full thickness burns;
- admission < 24 h from time of injury.

Patients who had any co-morbidities or required endotracheal intubation were excluded. Between 2008 and 2010, 110 burns patients were admitted to the SGH Burns Centre of whom only 18 met these strict criteria. All patients received standard burns management at SGH, including exposure to ambient room temperature of 22–30^{O}C, use of bio-occlusive dressings after thorough cleansing, adequate resuscitation using the Parklands formula, provision of blankets and the administration of adequate analgesia but excluding non-steroidal anti-inflammatory drugs.[14] Any patients febrile above 38^{O}C had a full sepsis work up.

HBOT INTERVENTION

The HBOT protocol was chosen based on safety, research aims and practicality. Patients were treated on the routine HBOT runs, and completed two sessions within 22 hours of admission. Each HBOT session consisted of 90 min at 243 kPa breathing 100% oxygen, with an ambient room temperature of 22–30^{O}C. The minimum interval between the two sessions was 120 minutes. Sham hyperbaric treatments for control subjects were not performed.

BLOOD PROCESSING

Blood samples (15 ml) were collected into four containers before and after HBOT and at similar times in the control group via needle-stick venepuncture; one was sent for routine laboratory analyses, including white blood cell (WBC) count and differential. Three plastic serum separator tubes, each containing approximately 3 ml whole blood, were inverted five times and allowed to clot for 30 minutes at room temperature. These were then centrifuged at 1,200 rpm for 15 minutes at 4^{O}C and stored at -80^{O}C until analysis. Serum interleukin (IL)-1β, IL-2, IL-4, IL-5, IL-6, IL-10, IL-12 (p70), IL-13, interferon-gamma (IFN-γ) and tumour necrosis factor-alpha (TNF-α) levels were measured using a Bio-Plex system (Precision Pro Human Cytokine 10-plex panel, 171-A1001P, Bio-Rad Laboratories, Inc., USA). The choice of cytokines measured was based on the cytokines that had previously been reported to be elevated with burns.[15–17]

ASSESSMENT OF BURN DEPTH

Laser Doppler imaging

Laser Doppler imaging (LDI) was chosen as an objective, independent assessment tool.[12] A Moor Instruments LDI2-Burn Imager (BI) (Moor Instruments Ltd., Axminster, England, UK) was used. All patients underwent LDI scans before and after HBOT or at similar times in the control group. The LDI flux values were based on an improved colour palette used to interpret burn depth in previous studies: < 260 perfusion units (PU, unit for flux based on a range corresponding to the visible spectrum) for deep dermal and full thickness burns (blue and green); 260–800 PU for superficial dermal wounds (yellow, pink and red).[18] The areas of burns that fall into the pre-defined PU ranges can be marked out and measured using a touch screen on the LDI as regions of interest.

HISTOLOGICAL ASSESSMENT

A biopsy within the LDI-assessed deep dermal burn was taken from each patient 48 h post burn. Skin biopsy specimens were fixed by immersion in 4% formalin for 48 h and then dehydrated in alcohol, cleared with xylene, and embedded in paraffin wax. Sections of 4 μm thickness were cut and microwaved in citrate buffer for antigen retrieval and blocked with peroxidase blocking reagent (S2023, DAKO UK Ltd, UK). Sections were stained using hematoxylin and eosin (H&E) for general morphology. Skin samples were classified into five anatomical layers: epidermis (level 1), upper one-third of the dermis (level 2), middle third of the dermis (level 3), deepest third of the dermis (level 4) and subcutaneous fat (level 5). The epidermis was evaluated for burn artefacts (distortion of cell contour) and separation of epidermis from dermis (subepidermal blistering). The dermis was evaluated for the histological separation or destruction of different cell layers of hair follicles and vessel walls, microthrombi and infiltration of neutrophils. Burn depth at each evaluation was graded as 1–5 according to the depth of the deepest burn-related histological finding of each sample. In addition, sections were stained with Masson trichrome stain (HT15, Sigma-Aldrich), which stains denatured collagen red, to estimate full-thickness burn depth through disruption of the dermal layer.

Immunohistochemistry

For apoptosis staining, sections were stained according to the protocol provided in the ApopTag® Peroxidase in situ Apoptosis Detection Kit (S7100, Chemicon International, Inc MA, USA). For immunohistochemistry, sections were also incubated with monoclonal rabbit anti-CD11a (Ab52895, Abcam Inc MA, USA), diluted 1:100 in PBS; monoclonal mouse anti-CD68 (M0814, DAKO UK Ltd) diluted 1:150 in PBS; and polyclonal rabbit anti-vascular endothelial growth factor (VEGF) (Thermo Scientific, USA) diluted 1:100 in PBS for the detection of CD11a, CD68 and VEGF respectively. Subsequent antibody detection

Table 1

Summary of patient demographics, burn aetiology and total body surface area (TBSA) and treatment arm (n = 17); LDI – Laser Doppler imaging assessment of burn depth and burns conversion at 24 h and 48 h (n = 13), S - superficial (mean PU: 260 – 800), D – deep (mean PU: 0 – 260); H&E burn depth assessment at 48 h post-burn skin tissue (n = 15); M - male, F – female; N/A – not available

Age	Gender	Burn cause	TBSA (%)	LDI (24 h)	LDI (48 h)	H&E Score (48 h)
No HBO treatment						
24	M	Contact	2.5	N/A	N/A	N/A
24	M	Flash	17	N/A	N/A	4
28	M	Flame	35	N/A	N/A	3
59	F	Scald	3	S	S	0
38	M	Scald	7	D	D	4
47	F	Scald	8	D	D	4
30	M	Chemical	7	D	D	1
31	M	Scald	25	D	D	3
48	M	Scald	6.5	S	S	3
HBO treatment						
37	F	Flame	17.5	D	S	N/A
46	M	Scald	6.5	D	D	4
49	M	Flame	9.5	D	D	4
25	M	Scald	18	S	S	3
55	M	Flame	18	N/A	N/A	4
48	F	Scald	8	D	D	4
25	F	Flash	1	D	S	4
29	M	Flame	3	S	D	3

was carried out using either anti-mouse or anti-rabbit IgG (Envision + system-HRP, DAKO UK Ltd) and then visualised using Vector® VIP Peroxidase Substrate (SK4600, Vector Laboratories, Inc., USA). All samples were examined under light microscopy. The number of immuno-positive cells and apoptotic cells were scored semi-quantitatively by an independent observer who was blinded to allocation.

MICROBIOLOGY

Quantitative culture and histological identification of bacteria in tissue specimens of viable unburned tissue has long been considered the gold standard for determining burns wound infection.[13–14] Tissue biopsies were taken both from areas of deep dermal burn and likely viable margins from each subject 48 h after burning. All positive cultures were quantified based on counts per gram of tissue and tested for sensitivity. Gram-negative isolates were tested for susceptibility to a range of antibiotics using the Kirby-Bauer disk diffusion method. Results of tissue cultures from the HBOT group were compared with those in the control group.

STATISTICS

Pre-trial power analysis based on an expected improvement of at least 10% in burn depth conversion in the HBOT group compared with the control group required a sample size of 40 subjects from each treatment group to achieve the limits defined ($\alpha = 0.05$; $\beta = 0.9$). Differences between the control and HBOT groups were compared using the Mann-Whitney U test when there was evidence of kurtosis/skew in the distribution of the values obtained. A one-sample Student's t-test was used to compare the burn levels against normal physiological levels (either the specified clinical range employed at SGH or, where no such values existed, derived from the blood of ten healthy control subjects). Pearson chi-square test was used to compare the incidences of positive microbiological tissue cultures. Data are expressed as means ± SD, where appropriate. Significance was accepted at a P value of less than 0.05.

Results

DEMOGRAPHICS

One patient was excluded because of newly discovered diabetes mellitus. Of the remaining 17 patients, 5 were female and 12 were male of varying burn aetiology, mainly from scalds (eight patients) and flame burns (five patients) (Table 1). Nine subjects (mean age 36.6, range 24–59 years) were assigned as controls and eight (mean age 39.0, range 25–55 years) to HBOT. The TBSA at admission in the control and HBOT arms were 13% (range 2.5–35%) and 12% (range 1–18%) respectively. There were no statistical differences between the two groups. The anatomic location of the injuries varied widely. No problems were encountered by the patients during HBOT.

WBC COUNT AND CYTOKINES

The WBC count was significantly raised immediately after burn compared with the maximum normal range (14 ± 5

Table 2
Systemic levels of haematological and cytokine markers of burn subjects at 24 h (n = 17); mean ± SD

Marker	Non-burn control	Burn	*P*- value
White blood cells (10^9 L^{-1})	4–10	14 ± 5	0.011
Neutrophil (%)	40–75	76 ± 12	0.73
IL-1β (pg ml^{-1})	0.12 ± 0.16	1.13 ± 1.24	0.004
IL-4 (pg ml^{-1})	0.02 ± 0.05	0.16 ± 0.23	0.023
IL-6 (pg ml^{-1})	0.73 ± 1.73	127.68 ± 174.66	0.012
IL-10 (pg ml^{-1})	0.28 ± 0.51	14.4 ± 20.42	0.014
IFN-γ (pg ml^{-1})	0.05 ± 0.15	0.74 ± 1.29	0.003

vs. 10 x 10^9 L^{-1}, P = 0.011), but there was no significant increase in neutrophils beyond the physiological range (76 ± 12% vs. 75%, P = 0.73). No significant changes in WBC count were found between the control and HBOT groups at either assessment time. Serum cytokines IL-1β, IL-4, IL-6, IL-10, and IFN-γ levels were significantly elevated after burn compared with non-burn control values, but there were no differences in systemic cytokine levels between the two patient groups (Table 2).

BURN DEPTH AND IMMUNOHISTOCHEMICAL CHANGES

Using LDI, 9 out of 13 subjects (LDI data were lost for four patients) were identified with deep dermal burns at 24 h whilst eight were identified with deep burns at 48 h. One HBOT subject had burns conversion from superficial to deep whilst one subject in the non-HBOT group and two in the HBOT group had conversion from deep to superficial burn (Table 1). There were no significant changes between the first and second assessments in either the control (pre 251 vs. post 271 PU, P = 0.522) or HBOT (pre 238 vs. post 244 PU, P = 0.949) groups. There were no significant differences between the two patient groups at either assessment (P = 0.475 and P = 0.253 respectively).

Two patients did not require skin excision surgery whilst, of the remaining 15, superficial to partial thickness dermal injury was observed in six. No distinction in histological observations between control and HBOT groups could be made based on burn depth scoring and collagen alteration. Comparison of H&E scoring with LDI assessment of burn depth at day two showed a discrepancy in only 2 out of 12 assessments (Table 1). TUNEL staining revealed apoptotic cells mainly in the upper half of the dermis, whilst CD11a and CD68 immunostaining showed varying degrees of leukocyte and macrophage infiltration in the burned skin sections respectively. VEGF immunostaining for mononuclear and polymorphonuclear leukocytes, endothelial cells and fibroblast-like cells also varied. No differences in immunostaining could be seen between the HBOT and control groups.

MICROBIOLOGY

Four subjects from the control group had positive bacteriological cultures versus two in the HBOT group. Several different organisms were identified, including *Staph. aureus* and *Pseud. aeruginosa*.

Discussion

We wished to assess the role of HBOT as an adjuvant to modern burns care. While our power calculation suggested we required 40 subjects in each arm of this study in order to confirm or refute a clinically significant effect of HBOT, a decrease from the predicted numbers of burns admissions in Singapore over the study period, combined with the strict entry criteria for this study, resulted in a very slow recruitment rate, preventing completion of the study to the level planned. Therefore, this trial can provide only a limited understanding of the effects of HBOT on early burns. However, we considered that it would be useful to describe our methodology in detail. To better understand the mechanism of action of HBOT on burns conversion, we measured an extensive range of biochemical and haematological indices (of which only WBC counts are reported here) and inflammatory cytokine markers. No significant differences were reported for any of these parameters following HBOT. Thus, we did not see any discernible effects of HBOT in this limited number of patients.

Our findings corroborate previous reports on the accuracy and reliability of LDI for assessing burn depth.[19] In this study, H&E tissue staining and LDI were in agreement in the assessment of burn depth in 10 of 12 patients for whom complete data were available (Table 1). We were unable to demonstrate whether there were any positive effects of HBOT on burns conversion, again, probably because of the low power of the study.

The lower proportion of patients with positive tissue cultures after HBOT suggests that HBOT might have a broad-spectrum antimicrobial action, but numbers were too small to

demonstrate any real differences. Nevertheless, this may be a useful area for future study given the known antimicrobial effects of HBOT.[20]

Conclusions

The WBC count was significantly elevated at 24 h after burn, along with systemic cytokines. We found no statistically significant differences between the control and HBOT groups in WBC count, inflammatory cytokine levels or microbiology. There was no evidence of improved burn conversion with HBOT. Unfortunately the study was of low statistical power because of recruitment problems and larger RCTs are still needed to determine whether HBOT has a place in modern burns therapy. LDI correlated well with H&E staining for flame and scald burns; LDI should be an effective independent assessment tool for burn depth and burn tissue for future research. Immunohistochemical evidence of macrophage infiltration and increased VEGF expression features strongly within 48 h of burn.

Acknowledgements

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Conflict of interest
This study was supported by a research grant from Ministry of Defence, Singapore (20080110) and in-kind contribution by Singapore General Hospital, Singapore. No conflicting financial interests exist.

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Case reports

Pulmonary oedema in breath-hold diving: an unusual presentation and computed tomography findings

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Abstract

(Gempp E, Sbardella F, Cardinale M, Louge P. Pulmonary oedema in breath-hold diving: an unusual presentation and computed tomography findings. *Diving and Hyperbaric Medicine*. 2013 September;43:162-163.)
Haemoptysis and pulmonary oedema following deep breath-hold diving have been described in recent years. We describe the case of a 33-year-old healthy military diver who presented symptoms suggestive of pulmonary oedema after two breath-hold dives, the first lasting 0.5–1 min and the second 1–2 min, to 6 metres' depth in the sea. The diagnosis was promptly confirmed with chest computed tomography showing bilateral interstitial infiltrates in the upper regions of the lungs. To our knowledge, this is the first report to document pulmonary oedema in this setting of shallow breath-hold diving with atypical radiological presentation. A definite mechanism for this specific distribution of lung injury remains unclear.

Key words
Breath-hold diving, freediving, pulmonary oedema, radiological imaging, case reports

Introduction

In recent years, haemoptysis and pulmonary oedema have increasingly been observed in competitive breath-hold divers and underwater fishermen after deep dives.[1–3] These disorders have also been described in endurance swimmers and scuba divers but there is no case reported in very shallow diving depths except after experimental breath-hold dives preceded by full expiration.[4,5] This report, presented with the patient's consent, is the first to document computed tomography (CT) findings consistent with interstitial-alveolar damage due to capillary stress failure in this setting and is presented with the patient's consent.

Case report

A 33-year-old man was admitted complaining of cough and pink, frothy sputum immediately after two breath-hold dives to a depth of 6 metres' sea water, the first lasting 0.5–1 min and the second 1–2 min, with a surface interval of less than 1 minute. He noted wheezing but had no chest pain nor dyspnoea after exiting the water. The patient was a healthy military diver candidate on the first day of a naval ship diving course. He denied aspirating sea water and performing manoeuvres such as glossopharyngeal insufflation and voluntary diaphragmatic contractions. He was not taking any medications and was a non-smoker with no past medical or family history of cardiovascular disease or immersion pulmonary oedema. The water temperature was 10^{O}C and he was wearing a 5-mm neoprene wetsuit with 3 kg of weight. He was immersed at rest for about 10 minutes waiting his turn to dive. No significant strenuous fin swimming or physical exertion was performed during the hours preceeding the diving session.

On admission 90 minutes after initial symptoms, the diver was comfortable. Physical examination revealed mild, diffuse, bilateral 'crackles' on chest auscultation. His pulse was 72 bpm and blood pressure 130/70 mmHg. Arterial blood gases, laboratory investigations (including clotting tests, D-dimer and cardiac biomarkers) and electrocardiogram an (ECG) were normal. Chest CT scan showed bilateral interstitial and alveolar infiltrates in the upper lobes while the lower zones were remarkably normal (Figure 1). Follow-up investigations at one month, including ECG, exercise testing, spirometry with lung volumes and chest X-ray, were unremarkable.

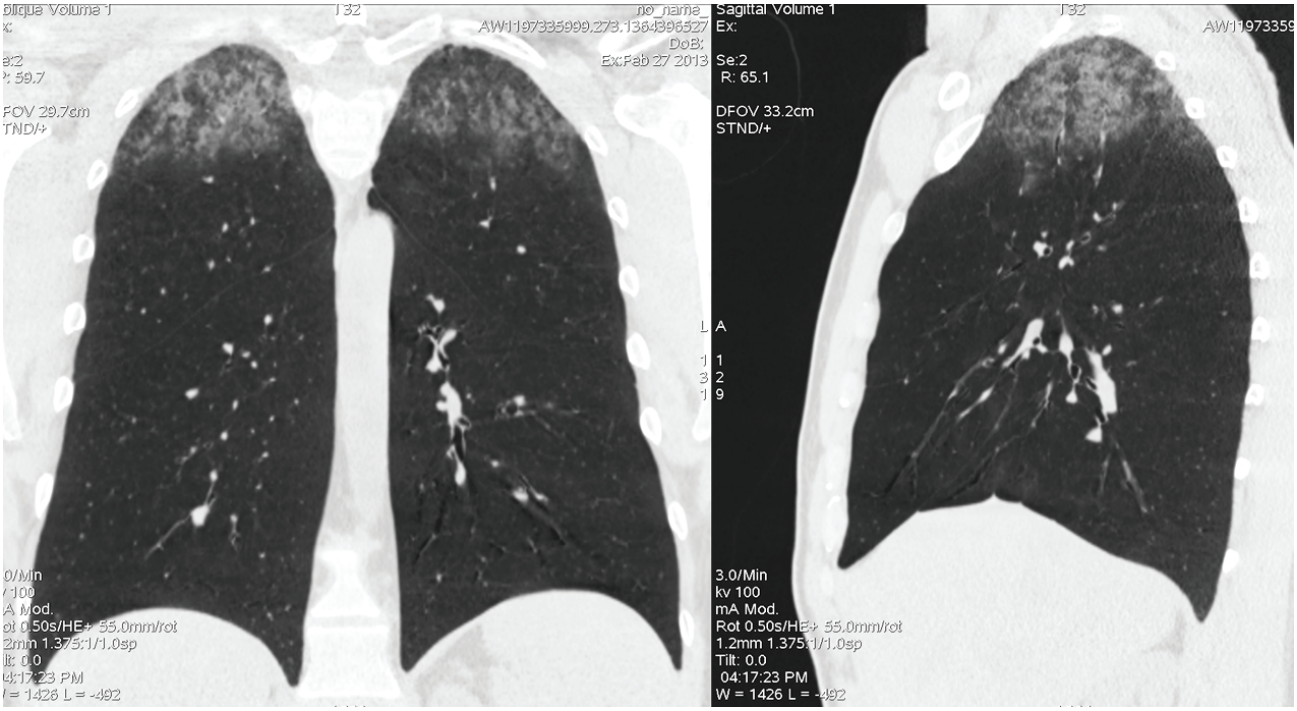
Discussion

Several factors contributing to pulmonary oedema in breath-hold diving have been identified, including cold water immersion leading to central pooling of blood and haemodynamic alterations, reduction of lung gas volume during descent ('lung squeeze'), exertion, hypoxia and diaphragmatic contractions.[3,6,7] Taken together, these mechanisms result in an increased trans-capillary pressure gradient that would disrupt the blood-gas barrier and cause alveolar haemorrhage. Some authors argue that aspirin or other non-steroidal anti-inflammatory drugs may also promote this condition.[8,9] It has also been postulated that neurohumoral stimulation resulting from emotional stress may be involved, as might be the case here.[10]

Although the radiographic features associated with pulmonary oedema in breath-hold divers have been well described in the literature, there are no data examining the distribution of these lesions within the lungs.[7,8] The striking preferential bilateral localisation of ground-glass opacities to the upper-zones in our case is rather

Figure 1

Chest CT scans with coronal and sagittal reconstruction (minimal intensity projection technique) that demonstrate areas of ground-glass opacities involving the apices of both lungs and sparing the rest of the parenchyma. Note the lack of Kerley lines or visibly enlarged vessels



unexpected. A proposed mechanism for the development of this pathological condition is the changes in alveolar pressure regimen between the lower and upper regions of the lungs that tends to be more negative in the apices when the diver has a vertical head-down posture during the descent. As a result, the transmural vascular pressure gradient and capillary stress should be maximal in those regions, thus leading to this specific pattern of interstitial oedema and subsequent alveolar injury.

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Efficacy of hyperbaric oxygen therapy in a young woman with idiopathic branch retinal artery occlusion

Mehmet Demir, Orhan Kara, Atakhan Yıldız and Dilek Guven

Abstract

(Demir M, Kara O, Yildiz A, Dilek G. Efficacy of hyperbaric oxygen therapy in a young woman with idiopathic branch retinal artery occlusion. *Diving and Hyperbaric Medicine*. 2013 September;43(3):164-165.)
We present a case of branch retinal artery occlusion (BRAO) in a healthy 20-year-old woman with no history of ocular or systemic diseases or drug use. She presented with a sudden decrease in visual acuity associated with a visual field defect of the right eye, which she had first noticed 4 hours earlier. Examination showed a BRAO with oedema at the upper part of the macula and surrounding area, and confirmed on fluorescein angiography. The left eye was normal. She was sent immediately for hyperbaric oxygen therapy (HBOT) and received 10 sessions (over 20 days) of 2 hours each at a pressure of 253 kPa. Follow up at four months showed a normal fundus, and visual acuity of 20/25. Visual field and fundoscopy were normal. Investigations for a cause of the BRAO proved negative. Retinal artery occlusion is rare in young people, and early application of HBOT in patients with RAO appears to improve outcome.

Key words
Retinal artery occlusion, hyperbaric oxygen therapy, outcome, case reports

Case report

A previously healthy, 20-year-old women presented 4 hours following the development of unilateral (right-sided) loss of vision. At presentation, visual acuity (VA) in the right eye was 20/200 with an inferior quadrant visual field (VF) defect. Fundoscopy showed oedema at the upper part of the macula and retinal quadrant (Figure 1). Central macular thickness (CMT) was 256 µm and intraocular pressure (IOP) was 12 mm Hg in the affected eye. Fluorescein angiography revealed ischaemia and a non-perfused branch retinal artery (BRAO) (Figure 2). The left eye was normal. She had no past medical or surgical history and was on no medications. Her social history was negative for alcohol, tobacco or illicit drug use. A complete evaluation regarding the aetiology of the BRAO was deferred so that HBOT could begin immediately. The patient commenced hyperbaric oxygen therapy (HBOT) within 5 hours of noting visual loss. She received 10 HBOT of 2 hours' duration each at a pressure of 253 kPa over 20 days.

Two weeks following HBOT, VA was 20/100 and CMT was 234 µm. Four months following therapy, VA was 20/25, CMT 208 µm and IOP 16 mmHg. The right fundus appeared normal (Figure 3) and angiography revealed a patent branch artery (Figure 4). Subsequent investigation into the aetiology of the patient's BRAO did not reveal a specific cause.

Discussion

Retinal artery occlusion (RAO) causes irreversible visual loss in all but 1–8% of patients.[1,2] HBOT has been found to be a safe and effective treatment for patients with RAO.[3] RAO is mostly seen in the elderly, with an estimated 0.85 per 10,000 patients over the age of 40 years affected.[4] RAO is rare in young people. The most common causes of RAO include hypertension, hyperlipidemia, hyperhomocysteinaemia, cardiac abnormalities, cardiac tumours, vasculitis, uveitis and infections.[5–7] Complications of RAO include optic atrophy, blindness, neovascular glaucoma and retinal atrophy. Although in this case no identifiable aetiology for her BRAO was identified, thorough investigation should always be attempted in cases of RAO.

Evidence for the efficacy of HBOT for RAO was reviewed in detail recently, suggesting a substantially improved rate of visual recovery with early HBOT compared to treatment without HBOT.[2] Based on this evidence, early HBOT therapy in patients with BRAO of no known aetiology may be a good therapeutic option, providing a favourable outcome with recovery of visual acuity, visual field defects and recovery of the retinal signs.

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Figure 1
Right fundus image showing retinal and macular oedema

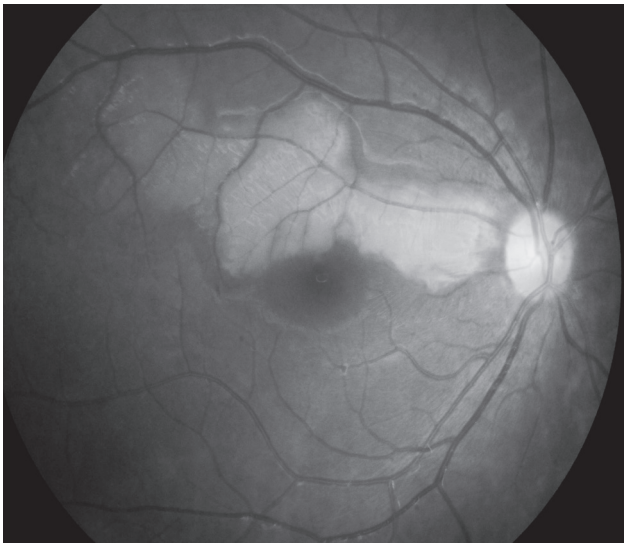


Figure 2
Fluorescein angiography showed ischaemic area and non-perfused right branch retinal artery

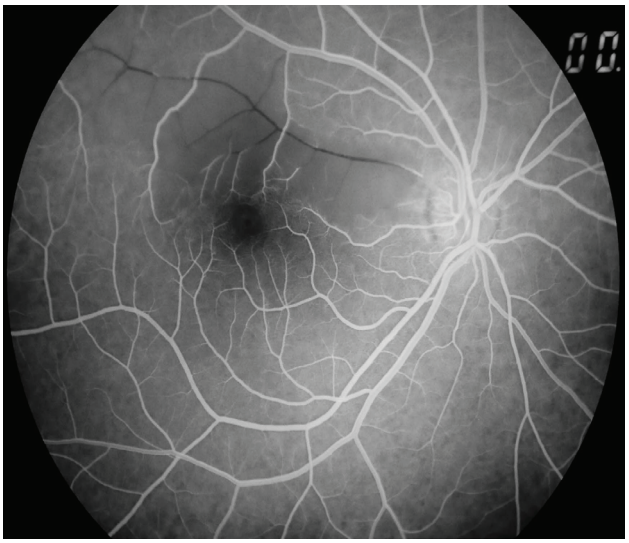


Figure 3
Normal right retinal fundus at four-month follow up

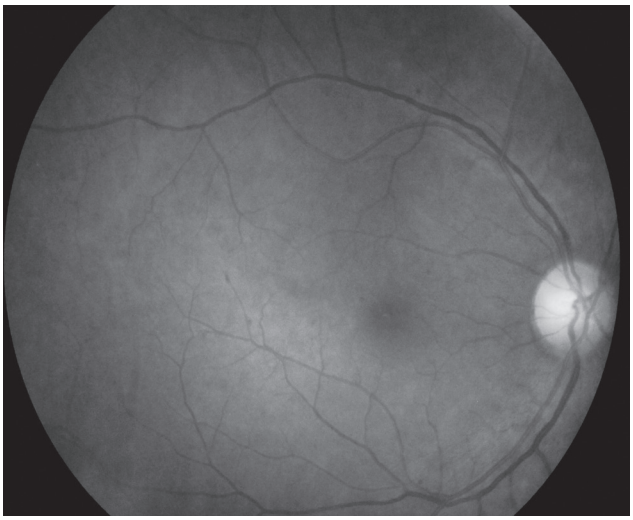
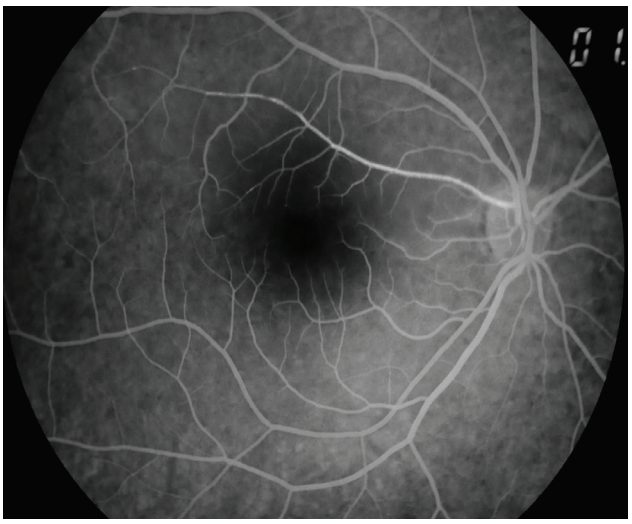


Figure 4
Fluorescein angiography showing re-canalised branch retinal artery



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Hyperbaric oxygen in the treatment of perichondritis of the pinna

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Abstract

(Fernandes FL, Lavor M, de Carvalho GM, Guimarães AC. Hyperbaric oxygen in the treatment of perichondritis of the pinna. *Diving and Hyperbaric Medicine*. 2013 September;43(3):166-167.)

Perichondritis is a condition characterized by pain, swelling and purulent discharge from the external ear, which may progress to a deformity of the pinna. The presence of co-morbidities such as diabetes mellitus may aggravate the situation. The main aetiological agent is *Pseudomonas aeruginosa* and treatment consists of antibiotics combined with surgical drainage of the ear. We present the case of a diabetic patient with recurrent perichondritis of the pinna treated with hyperbaric oxygen therapy, with successful healing. Hyperbaric oxygen therapy has proved beneficial as adjunctive therapy of lesions in diabetic patients with foot ulcers, acting in the regeneration of intracellular free radicals and promotion of wound-healing factors. Thus, owing to its mechanisms of action, its effect on other injuries such as perichondritis in diabetic patients may be beneficial and lead to improvement.

Key words

Ear, infection, wound healing, hyperbaric oxygen therapy, case reports

Introduction

Auricular perichondritis is a disease of the pinna that may cause severe deformity.[1] The commonest predisposing factors for this condition are trauma, burns, insect bites, post-operative infections of the skin, bruising or seromas.[1] Diabetes mellitus contributes to the worsening of symptoms.[1,2] The clinical picture of auricular perichondritis includes pain, swelling, deformity and purulent discharge, with *Pseudomonas aeruginosa* as the most common aetiologic agent. Treatment usually consists of incision and drainage and local and systemic antibiotic therapy. In some cases, auricular debridement is also necessary. Other bacteria commonly causing the disease include *Staphylococcus aureus*, *Escherichia coli* and *Proteus sp*.[1,3]

We report the case of a patient with Type 2 diabetes mellitus and recurrent auricular perichondritis treated successfully with hyperbaric oxygen therapy (HBOT) and antibiotics.

Case report

A 55-year-old male presented with signs of perichondritis of the left ear with spontaneous drainage of secretions in the region of the anterior helix. He denied any history of trauma, fever, dizziness, tinnitus, hearing loss, or other co-morbidities. Otoscopy showed intact tympanic membranes bilaterally, without abnormalities.

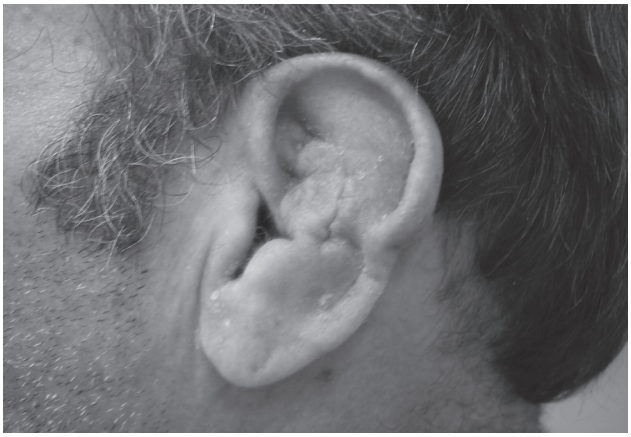
Ambulatory drainage with placement of a Penrose drain was performed and he remained hospitalized for 10 days for treatment with intravenous oxacillin (1000 mg three times a day) and ciprofloxacin (400 mg twice a day), and daily pressure dressing changes. On admission, his fasting glucose was 18.1 mmol L^{-1} (reference value 2.2–5.5 mmol L^{-1}), and he was commenced on subcutaneous soluble insulin according to pre-prandial blood glucose levels and a low-glycaemic diet. He was discharged in good condition on oral ciprofloxacin (500 mg daily), pressure dressings and insulin (NPH plus regular insulin before breakfast, regular insulin before lunch and dinner, and NPH insulin at bedtime)

Forty days after discharge he represented with intense swelling of the left pinna, with areas of fluctuation and signs of inflammation. The abscess was drained and he was commenced on IV ciprofloxacin (400 mg 8 hourly) and hydrocortisone (100 mg 6 hrly). Microbiology was positive for *E. coli* ESBL+ (Extended-spectrum beta-lactamase), and a search for fungi and acid-fast bacilli was negative. On the fourth day of antibiotic therapy, the culture results indicated a need to change to imipenem IV (500 mg 6 hrly). After 15 days of antibiotic treatment the patient was discharged without oedema or erythema in the pinna, and no auricular discharge.

One week after hospital discharge, the patient relapsed again and imipenem (500 mg, 6 hrly) and daily compressive dressings were restarted. CT scan of the neck revealed perichondritis of the left external ear, a left intra-parotid lymph node and enlarged lymph nodes in the cervical chains bilaterally.

Again, the patient improved and he was discharged on the eighth day. Hyperglycaemia was well controlled. However, this improvement was not sustained and a month later the use of HBOT was considered. He underwent 10 daily, 2 h HBOT at a pressure of 304 kPa, which resulted in complete remission of erythema and oedema of the ear, without purulent discharge (Figure 1). At a three-month follow up, otoscopy was normal and he was asymptomatic.

Figure 1
Left ear of patient after 10 HBOT at 304 kPa (with permission)



Concomitantly, the hyperglycaemia was investigated and, on fundoscopy, severe diabetic retinopathy and neovascular glaucoma were confirmed and glibenclamide 5 mg daily was commenced.

Discussion

Because of their vulnerability, any lesions of the pinna, if handled improperly or ignored, can lead to evident deformity. Treatments that have been proposed include: repeated incision and drainage with local instillation of antibiotics; excision of affected cartilage, anterior overlying skin and perichondrium; through-drainage tube method, and ultraviolet rays.[1–4]

HBOT may be beneficial as a complementary therapy for soft-tissue injuries in patients with diabetes, compromised skin flaps, osteoradionecrosis, soft tissue necrosis and gangrene.[5] With particular relevance to this case report, HBOT has proved beneficial in healing problem wounds in patients with diabetes mellitus.[6,7] The main mechanisms of action of HBOT are based on the regeneration of intracellular free radicals of oxygen and nitrogen.[8] Breathing oxygen at greater than 101.3 kPa increases production of reactive oxygen species (ROS). This is critically important as it is the molecular basis for a number of therapeutic mechanisms. In association with reactive nitrogen species (RNS), they serve as signalling molecules in transduction cascades, or pathways, for a variety of growth factors, cytokines and hormones. RNS include nitric oxide and agents generated by reactions between nitric oxide or its oxidation products, and ROS.[8] ROS are generated intracellularly as part of normal metabolism, acting in conjunction with several redox systems, and play central roles in coordinating cell signalling and also anti-oxidant, protective pathways. HBOT has been shown to improve diabetic wound healing by increasing circulating stem cells.[9]

HBOT is a relatively safe procedure. The risk of central nervous system oxygen toxicity is approximately 1 to 4 in 10,000 patient treatments, depending on both the pressure and duration of exposure.[8] The difficulties of treatment by HBOT include the cost and availability of hyperbaric facilities. Furthermore, patients require multiple sessions so it is demanding on their time. However, the number of HBOT chambers has increased, allowing greater access to this intervention.

This case of auricular perichondritis in a diabetic patient with multiple relapses which resolved with HBOT suggests this is a useful treatment option in this condition.

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Reprinted from other sources

Guidelines for rescue of an unresponsive diver from depth

Simon J Mitchell
on behalf of the Undersea and Hyperbaric Medical Society Diving Committee

Introduction

The management of drowning (and other emergencies) has been the subject of periodic evidence-based review by panels of experts who publish consensus guidelines.[1] In response to questions from the American Academy of Underwater Sciences and the Professional Association of Diving Instructors, the Diving Committee of the Undersea and Hyperbaric Medical Society conducted a similar review of methods for rescue of an unresponsive diver from depth.[2] The committee recommended actions on finding an unresponsive diver underwater in circumstances either where the disabling event is witnessed or where the period of unresponsiveness is uncertain and resuscitation must, therefore, be considered possible. The scope of diving to which the recommendations apply was limited to compressed gas bounce dives (dives in which the duration from leaving to returning to surface is of the order of minutes or hours) using a half-face mask, separate mouthpiece, and either open-circuit scuba equipment or a rebreather.

The rescue process was subdivided into three phases: preparation for ascent; retrieval to the surface; and procedure at the surface. A series of questions pertinent to each phase was researched, debated and answered. A decision tree for rescue of an unresponsive diver was constructed (Figure 1).[2] The process and logic underpinning the recommendations are fully described in the original publication, but several of the committee's important decisions on controversial matters are discussed briefly here.[2]

Underwater seizure

Where unresponsiveness is the result of an ongoing seizure, the recommended action depends on whether the mouthpiece remains in the mouth or not (Figure 1). If it is retained, then attempts should be made to hold it in the mouth and the rescuer should wait for the clonic phase of the seizure to subside before initiating ascent. If it is out of the mouth (anecdotally the most common scenario), the committee recommends bringing the diver straight to the surface even if the seizure continues. This contradicts a long standing belief that a seizing diver must be held at depth until the seizure has terminated to avoid pulmonary barotrauma precipitated by closure of the glottis during the ictal period. The committee reviewed the experimental evidence which suggests that the glottis does not completely close during the clonic phase of a seizure, and drew on its members' experience of being able to manually ventilate patients during a seizure.[3] On this basis it was judged that if the airway is completely unprotected the risk of pulmonary barotrauma during ascent while seizing is probably outweighed by the risk of drowning if ascent is delayed until the seizure finishes. In contrast, the small measure of airway protection afforded by a retained mouthpiece partly mitigates this concern and made the committee reluctant to abandon the 'traditional' advice under these circumstances. This dichotomy is reflected in the decision-making process depicted in Figure 1.

In-water rescue breathing

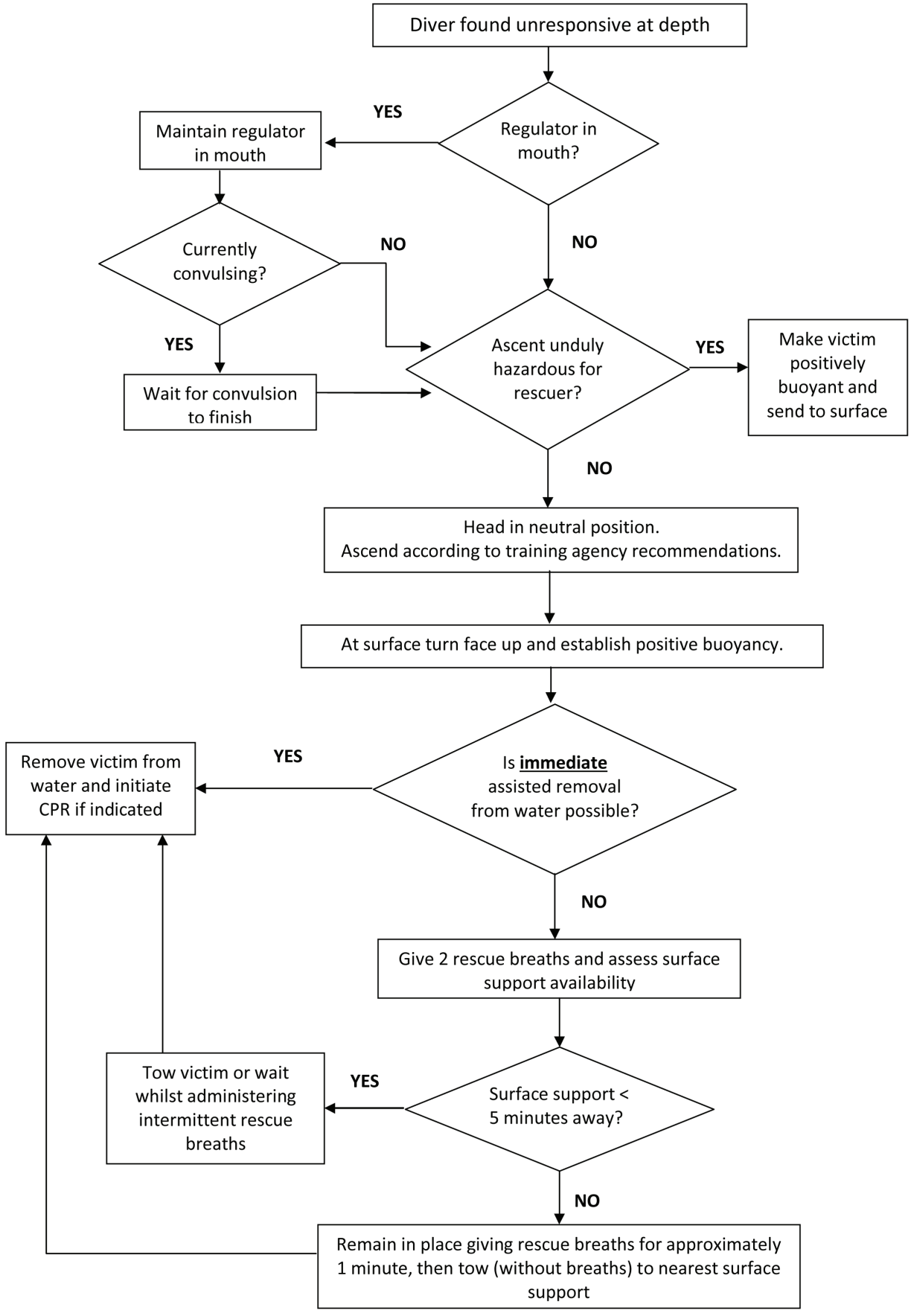
The committee considered the potential value of in-water rescue breaths administered to a non-breathing victim after surfacing. We found compelling evidence that there is frequently an interlude between respiratory and cardiac arrest in drowning, and that restoration of oxygenation during this period may prevent progression to full cardiac arrest.[4] Survival in the field after cardiac arrest is extremely unlikely and the committee, therefore, recognized the lifesaving potential of early lung ventilation. Although delivery of in-water rescue breaths might delay removal of the victim onto surface support for definitive cardiopulmonary resuscitation, the committee endorsed their use unless the rescuer is incapable of administering them, or removal from the water can be achieved virtually instantaneously.

Cardiopulmonary resuscitation

The committee reviewed the recent advocacy for 'compression only' CPR which has caused confusion for diver training organizations in relation to what method should be taught. This issue has also been confronted by experts whose primary interest is resuscitation in drowning scenarios where respiratory arrest is (as in diving) most likely to be due to asphyxia, and where there may be a significant interval between respiratory and cardiac arrest as discussed above.[1] Not surprisingly, commentators representing expert groups have argued that in drowning victims the correction of hypoxia is the first priority, and failure to provide ventilation to the victim may jeopardize outcome.[1,5] On this basis, the committee determined that the current advocacy for compression-only resuscitation in community cardiac arrest may not be relevant to diver rescue situations, and that initial in-water rescue breathing and subsequent pulmonary ventilation in conjunction with chest compressions during CPR are appropriate.

The committee was careful to contextualize their prescription of an approach to diver rescue with the observation that there is a high probability of a poor outcome when a diver becomes

Figure 1
Decision tree for the retrieval and resuscitation of an unconcious diver at depth (from reference 2)



unconscious underwater, even when the rescue is executed in a 'textbook' fashion. The recommendations are a living document and will be periodically updated.

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The above is a summary of the published recommendations of the Undersea and Hyperbaric Medical Society Diving Committee. Figure 1 is reprinted with kind permission from: Mitchell SJ, Bennett MH, Bird N, Doolette DJ, Hobbs GW, Kay E, et al. Recommendations for rescue of a submerged unresponsive compressed gas diver. *Undersea Hyperb Med*. 2012;39:1099-108.

Key words
Scuba diving, accidents, unconscious, rescue, resuscitation, flow chart, medical society, reprinted from

An analysis of the causes of compressed-gas diving fatalities in Australia from 1972-2005

Lippmann J, Baddeley A, Vann R, WalkerD

Abstract

In order to investigate causative factors, root cause analysis (RCA) was applied to 351 Australian compressed-gas diving fatalities from 1972–2005. Each case was described by four sequential events (trigger, disabling agent, disabling injury, cause of death) that were assessed for frequency, trends, and dive and diver characteristics. The average age increased by 16 years, with women three years younger than men annually. For the entire 34-year period, the principal disabling injuries were asphyxia (49%), cerebral arterial gas embolism (CAGE; 25%), and cardiac (19%). There was evidence of a long-term decline in the rate of asphyxia and a long-term increase in CAGE and cardiac disabling injuries. Asphyxia was associated with rough water, buoyancy trouble, equipment trouble, and gas supply trouble. CAGE was associated with gas supply trouble and ascent trouble, while cardiac cases were associated with exertion, cardiovascular disease, and greater age. Exertion was more common in younger cardiac deaths than in older deaths. Asphyxia became less common with increasing age. Equipment-related problems were most common during the late 1980s and less so in 2005. Buoyancy-related deaths usually involved loss of buoyancy on the surface but decreased when buoyancy control devices were used. Countermeasures to reduce fatalities based on these observations will require validation by active surveillance.

Reprinted with kind permission from: Lippmann J, Baddeley A, Vann R, Walker D. An analysis of the causes of compressed-gas diving fatalities in Australia from 1972–2005. *Undersea Hyperb Med*. 2013;40:49-61.

Key words
Diving deaths, scuba diving, cerebral arterial gas embolism (CAGE), cardiovascular, epidemiology, reprinted from

Editor's commentary:

Since the inception of Project Stickybeak in the early 1970s, this journal has published every annual report of Australian diving-related fatalities; the latest reported being those from 2008. Most of this time-consuming work was carried out by Dr Douglas Walker, but in recent years it has been assumed by a team coordinated by the Divers Alert Network–Asia Pacific in Melbourne. For a decade, I have been advocating for these records to be the subject of formal epidemiological analysis and this has finally occurred and is presented in this important paper on (mainly) recreational diving fatalities.

My only regret is that the authors, after the many years of support given to this work by this journal, did not publish this data analysis in an Australian journal such as *Diving and Hyperbaric Medicine*. From my perspective, the problem is that most members of the South Pacific Underwater Medicine Society are not members of the Undersea and Hyperbaric Medical Society (USA) and, therefore, will have difficulty in accessing this paper. If readers wish to discuss the report with the authors, they should contact John Lippmann at: <johnl@danasiapacific.org>.

Decompression illness: Clinical aspects of 5,278 consecutive cases treated in a single hyperbaric unit

Xu W, Liu W, Huang G, Zou Z, Cai Z, Xu W

Abstract

Background: Decompression illness (DCI) is a major concern in pressure-related activities. Due to its specific prerequisite conditions, DCI is rare in comparison with other illnesses and most physicians are inexperienced in treatment. In a fishery area in northern China, during the past decade, tens of thousands of divers engaged in seafood harvesting and thousands suffered from DCI. We established a hyperbaric facility there and treated the majority of the cases.
Methods and results: A total of 5,278 DCI cases were admitted in our facility from February 2000 through December 2010 and treated using our recompression schedules. Cutaneous abnormalities, joint and muscular pain and neurological manifestations were three most common symptoms. The initial symptom occurred within 6 h after surfacing in 98.9% of cases, with an overall median latency of 62 min. The shorter the latent time, the more serious the symptoms would be ($P < 0.0001$). Nine cases died before recompression and 5,269 were treated using four recompression schedules, with an overall effectiveness rate of 99.3%. The full recovery rate decreased with the increase of the delay from the onset of symptoms to the treatment ($P < 0.0001$).
Conclusions: DCI presents specific occurrence rules. Recompression should be administered as soon as possible and should never be abandoned irrespective of the delay. The recompression schedules used were effective and flexible for variety conditions of DCI.

***Plos One* is an international, peer-reviewed, open-access, online publication. We thank the publishers for permission to reproduce this abstract from: Xu W, Liu W, Huang G, Zou Z, Cai Z, XuW. Decompression illness: Clinical aspects of 5278 consecutive cases treated in a single hyperbaric unit. PLoS One. 2012;7(11): e50079. Published online 2012 November 21. doi: 10.1371/journal.pone.0050079**

Key words
Decompression sickness, decompression illness, arterial gas embolism, hyperbaric oxygen therapy, treatment sequelae, clinical audit, reprinted from

Editor's commentary:

This abstract does not give the whole story, and it is worth reviewing some of the data in this, probably the largest, published series of decompression illness (DCI) cases from a single hyperbaric facility.

The four treatment schedules used are somewhat different to those used widely in the Western World. Each involves air breathing with an initial 'pull' to 21 metres' sea water (msw) equivalent on air for mild DCI, and to 54 msw for the more severe cases. This is followed by staged decompression and a switch to oxygen at 18 msw. Once on oxygen, decompression continues in 2 msw stops to the surface, and the total treatment times range from approximately 4 h to 31 h.

Of the 5,278 cases reported, 3,831 (72.6%) were categorised as 'mild', 1,124 (21.3%) as 'moderate' and 323 (6.1%) as 'severe'. Thirty-three severe cases died (10.2%; overall mortality for all treated cases 0.6%), 30 of these being from the 85 cases diagnosed as arterial gas embolism (with or without evidence of pulmonary barotrauma). In the severe category, 185 of 314 (59%) reported outcomes were scored as complete recovery, but only nine cases showed no response to hyperbaric treatment (N.B. These figures do not tally with the mortality figure in the previous sentence).

Given the limited staffing (mostly only two doctors) and medical infrastructure reported in the paper, these are impressive results. However, most divers (they were all commercial fisheries divers) received only one hyperbaric treatment and follow up after discharge is lacking. Also, the number of divers presenting more than once is not reported. Nevertheless, the take-home message – treat as soon as possible, irrespective of the delay in presentation – is important.

Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning

Hampson NB, Piantadosi CA, Thom SR and Weaver LK

Abstract
Carbon monoxide (CO) poisoning is common in modern society, resulting in significant morbidity and mortality in the United States annually. Over the past two decades, sufficient information has been published about carbon monoxide poisoning in the medical literature to draw firm conclusions about many aspects of the pathophysiology, diagnosis, and clinical management of the syndrome, along with evidence-based recommendations for optimal clinical practice. This article provides clinical practice guidance to the pulmonary and critical care community regarding the diagnosis, management, and prevention of acute CO poisoning. The article represents the consensus opinion of four recognized content experts in the field. Supporting data were drawn from the published, peer-reviewed literature on CO poisoning, placing emphasis on selecting studies that most closely mirror clinical practice.

Reprinted with kind permission of the American Thoracic Society. Copyright © 2013 American Thoracic Society from: Hampson NB, Piantadosi CA, Thom SR, Weaver LK. Practice recommendations in the diagnosis, management, and prevention of carbon monoxide poisoning. *Am J Resp Crit Care Med*. 2012;186:1095-101. doi: 10.1164/rccm.201207-1284CI. Official journal of the American Thoracic Society.

Key words
Carbon monoxide, clinical toxicology, toxicity, treatment, hyperbaric oxygen therapy, reprinted from

Editor's commentary:

This is an excellent, useful review of an area of acute medical care that has been the subject of vigorous debate for at least four decades. The authors have been centrally involved in the study of CO poisoning for many years. This has proved a difficult poisoning to study clinically, histologically and biochemically, resulting in many widely differing opinions, often based on emotive and personal bias more than scientific evidence. For example, in Australia, one major hyperbaric centre will not treat CO poisoning at all, whereas in most others a wide range of clinical criteria are used on which to base clinical management decisions. The clinical management recommendations presented in this review are sensible. Readers who might wish to discuss this paper with the authors may contact Dr Hampson at: <Neil.Hampson@vmmc.org>.

Hyperbaric oxygen therapy improves peripheral insulin sensitivity in humans

Wilkinson D, Chapman IM and Heilbronn LK

Abstract
Aim: Hyperbaric oxygen therapy is known to reduce fasting blood glucose in individuals with Type 2 diabetes. However, the mechanisms of this effect are not clear. The aim of this study was to determine whether peripheral insulin sensitivity by hyperinsulinaemic euglycaemic clamp is increased in patients presenting for hyperbaric oxygen therapy.
Methods: Participants were non-obese individuals without Type 2 diabetes (n = 5) or obese patients with Type 2 diabetes (n = 5). Patients were given 100% oxygen at 2.0 absolute atmospheres for 2 h, six sessions per week for 5 weeks.
Results: Peripheral insulin sensitivity was increased in the whole cohort (P = 0.04). Subsequent analysis revealed that this was significant at both treatment 3 (+37.3 ± 12.7%, P = 0.02) and treatment 30 (+40.6 ± 12.6%, P = 0.009). HbA(1c) was significantly reduced in subjects without diabetes only ($P < 0.05$).
Conclusion: Insulin sensitivity increased within 3 days of hyperbaric oxygen treatment and this was maintained for 30 sessions. This increase in insulin sensitivity is equivalent to that observed following moderate weight loss. The mechanisms underlying the insulin-sensitizing effect of hyperbaric oxygen require further elucidation.

Reprinted with kind permission from: Wilkinson D, Chapman IM, Heilbronn LK. Hyperbaric oxygen therapy improves peripheral insulin sensitivity in humans. Diabet Med. 2012 Aug;29(8):986-9. doi: 10.1111/j.1464-5491.2012.03587.x.

Key words
Diabetes, hyperbaric oxygen, blood sugar level, endocrinology, hyperbaric research, reprinted from

Critical appraisals

Hyperbaric oxygen therapy no better than sham in improving post-concussion symptoms following mild traumatic brain injury

Bottom line:
No clear advantage of HBOT over the sham group; Improved symptoms over the treatment course in both sham and HBOT groups.

Citations:
1. Wolf G, Cifu D, Baugh L, Carne W, Profenna L. The effect of hyperbaric oxygen on symptoms after mild traumatic brain injury. *J Neurotrauma*. 2012;29:2606-12.
2. Weaver LK, Cifu D, Hart B, Wolf G, Miller S. Hyperbaric oxygen for post-concussion syndrome: design of Department of Defense clinical trials. *Undersea Hyperb Med*. 2012;39(4):807-14.

Lead author's name and e-mail:
David Cifu <dcifu@mcvh-vcu.edu>

Three-part clinical question:
For patients with post-concussion symptoms after mild traumatic brain injury does a course of hyperbaric oxygen therapy, combined with standard treatment approaches, result in any improvement in symptoms?

Search terms:
Mild traumatic brain injury, post-concussion syndrome

The study:
Double-blinded, concealed, randomised controlled trial with intention-to-treat.

The study patients:
Serving US military personnel with stable chronic post-concussion symptoms (2–71months) following one or more episodes of mild traumatic brain injury suffered during their service.

Control group:
(*n* = 25; 24 analysed)
Usual ongoing care plus a sham exposure breathing air at 131 kPa drifting down to 121 kPa for 90 minutes daily five days per week to a total of 30 sessions over eight weeks.

HBOT group:
(*n* = 25; 24 analysed)
Usual ongoing care plus hyperbaric sessions at 243 kPa, breathing 100% oxygen, on the same schedule as controls.

The evidence:
See Table 1.

Comments:
- Well-conducted trial; blinding tested in participants and was successful.
- Participants had experienced between 1 and 50 concussive episodes.
- Both groups showed improvement over the course of the study in both outcomes. This seems most likely owing to a participation effect.
- The ImPACT total scores at final follow up were better in the control group. Authors report significant downward trend in scores over the trial in both groups, but no significant differences between groups when testing against each other over all time periods. Our result (Table 1) only takes into account a single time point (6 weeks follow-up).
- Actual statistical method is not reported.

Appraised by: *Mike Bennett*
Prince of Wales Hospital, Sydney, July 2013
E-mail: *<m.bennett@unsw.edu.au>*

Key words
Brain injury, hyperbaric oxygen therapy, outcome, hyperbaric research, critical appraisal

Table 1
Outcome at 6 weeks after final treatment session.[1] Immediate Post-concussion Assessment and Cognitive Testing (scale unknown, lower score better);[2] Post-Traumatic Disorder Check List – Military Version (17 fields with 1 to 5 Likert scale each (range 17–85), lower score better (mean and SEM have been read off the figures in the paper, then SEM converted to SD)

Outcome at 6 weeks	Sham group Mean (SD)	HBOT group Mean (SD)	Difference	95% confidence interval
ImPACT[1]	26.0 (9.8)	32.8 (0.51)	-6.8	-10.8 to -2.8
PCL-M[2]	40.6 (4.9)	41.6 (4.9)	-1.0	-3.8 to 1.8

Weak evidence that HBOT is of benefit in the treatment of patients with *Herpes zoster*

Bottom line:
HBO may speed resolution of pain and rash from *Herpes zoster*.

Citation:
Peng Z, Wang S, Huang X, Xiao P. Effect of hyperbaric oxygen therapy on patients with herpes zoster. *Undersea Hyperb Med.* 2012;39:1083-7.

Lead author's name and e-mail:
Peng Z <pengzr138@yahoo.com.cn>

Three-part question:
For patients with *Herpes zoster*, does the addition of hyperbaric oxygen to standard care reduce the symptoms of the infection?

Search terms:
Herpes zoster, post-herpetic neuralgia

The study:
Non-blinded, randomized, controlled trial with intention-to-treat.

The study patients:
Patients diagnosed with *Herpes zoster* for less than 2 weeks.
Control group: (*n* = 32; 32 analysed)
Pharmacological treatment for *Herpes zoster* (acyclovir, mecobalamin, tramadol hydrochloride, nortriptyline); no sham treatment.
Hyperbaric group: (*n* = 36; 36 analysed)
As above, but patients were given 30 sessions of HBOT, twice daily at 223 kPa, breathing 100% oxygen for 80 minutes.

The evidence:
See Tables 1 and 2.

Comments:
- Study outcomes are measured when by natural history one would expect the infection to have resolved (5 weeks).
- The non-blinded study design without sham may have contributed to patient and investigator bias and a placebo effect.
- Study methodology was poorly reported.
- No significant difference in the number of patients who healed in each group.
- Probably no clinical significance in small difference in the numeric pain rating scale (NPRS).
- Hamilton depression rating scale (HAMD) may not be an appropriate tool. The authors' conclusion that HBOT is beneficial to depression is not reflected in the results.
- Results seem analyzed with intention-to-treat, despite the authors claiming that subject drop-outs were excluded from analysis.

Appraised by: *Alan Bourke and Bryan Hui, Prince of Wales Hospital, Sydney, June 2013*
E-mail: *<alan.bourke@sesiahs.health.nsw.gov.au>*

Key words
Infectious diseases, hyperbaric oxygen therapy, outcome, hyperbaric research, critical appraisal

Table 1
Proportion of patients who showed response to treatment at 3 weeks post-recruitment; NNT – Numbers needed to treat

Outcome at 3 weeks	Control group *n* = 32 (%)	HBOT group *n* = 36 (%)	Absolute risk reduction (%)	NNT
Complete healing (no pain or rash)	17	22	8	13
95% CI			-20 to 30	3 to INF NNH 6 to INF
Therapeutic efficacy (improved)	26	35	16	6
95% CI			1 to 31	3 to 70

Table 2
Changes in mean outcomes in clinical indicators for pain and depression; NPRS score ranges from 0 (no pain) to 10 (worst pain); HAMD score ranges from 1 (< 7 is normal) to 24 (> 20 indicates severe depression)

Outcome after 30 HBOT sessions	Control group Mean (sd)	HBOT group Mean (sd)	95% CI
Numeric Pain Rating Scale (NPRS)	3.53 (4.1)	1.8 (2.7)	-0.04 to -3.4
Hamilton Depression Rating Scale (HAMD)	10.9 (9.7)	16.5 (12.0)	0.3 to 10.9

Diving and Hyperbaric Medicine citation news

Diving and Hyperbaric Medicine has been indexed on MedLine since the March 2011 issue, and article abstracts are now accessible on PubMed going back to March 2008. This covers the entire period since SPUMS and EUBS became joint publishers of DHM. This was a very important milestone for the publishing longevity of this Journal. We submit from DHM original articles, reviews, technical reports, short communications, case reports, work in progress, some 'World as it is' and 'Opinion' articles and some 'Letters to the Editor'. We do not make the final decisions as to what is actually indexed from the Journal, this is made by the National Library of Medicine, based on our submission and their review of the entire contents of each issue.

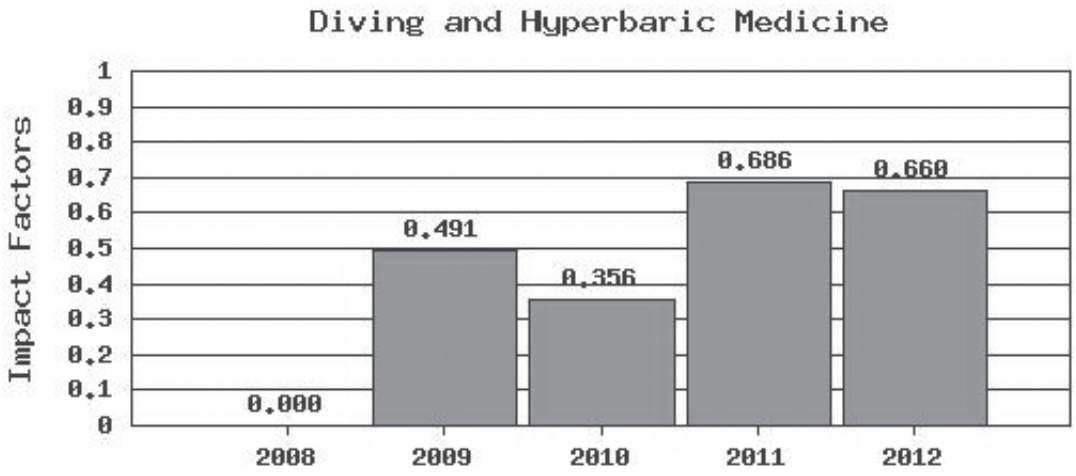
In addition, DHM was accepted in 2008 for indexing in the Thomson Reuters database Sci Search® – The Web of Science. Thomson Reuters publishes annual *Journal Citation Reports*®:
"*The JCR provides quantitative tools for ranking, evaluating, categorizing, and comparing journals. The impact factor is one of these; it is a measure of the frequency with which the "average article" in a journal has been cited in a particular year or period. The annual JCR impact factor is a ratio between citations and recent citable items published. Thus, the impact factor of a journal is calculated by dividing the number of current year citations to the source items published in that journal during the previous two years items published.*"
<ISI/Thomson Reuters _ The Thomson Reuters Impact Factor _ Science.htm>, Accessed: 19 July 2013.

Below are the latest data on the 5-year Impact Factor for DHM, taken from the DHM address:
<http://admin-apps.isiknowledge.com/JCR/JCR?RQ=RECORD&journal=DIVING+HYPERB+MED&rank=1#journal_title>.

This is a satisfactory outcome given the relatively recent addition of the Journal to Medline and Sci Search®, and is comparable to the 2012 IF for the UHMS journal, *Undersea and Hyperbaric Medicine*.

In addition, DHM (and before its name change, the *SPUMS Journal*) has been indexed on Elsevier's Excerpta Medica database EmBase/Scopus® since 2001. Almost all of the *SPUMS Journal* is in the Rubicon Foundation database:
< http://dspace.rubicon-foundation.org/xmlui/>

The Editorial Board believes that all this should encourage society members to contribute with some confidence to their own journal – at present only a minority of articles from Europe and the Asia-Pacific regions in the fields of diving and hyperbaric medicine is published in DHM. The Board and I are committed to delivering a diverse, interesting and up-to-date journal, but this can only be achieved with the enthusiastic support of EUBS and SPUMS members.



Journal Title	ISSN	Total Cites	Impact Factor	5-Year Impact Factor	Immediacy Index	Citable Items	Cited Half-life	Citing Half-life
DIVING HYPERB MED	1833-3516	93	0.660	0.541	0.448	29		9.4

Continuing professional development

Physiological effects of oxygen, breath-holding and immersion

CME activity 2013/3

Christian Fabricius

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 participant's 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
MOPS (maintenance of professional standards), blood pressure, cerebral blood flow, diving reflex, hyperbaric oxygen, immersion, physiology

Recommended background reading

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Some items may be difficult for SPUMS members to access. If so, please contact Dr Fabricius (see below).

How to answer the questions

Please answer all responses (A to E) as True or False.
Answers should be posted by e-mail to the nominated CPD co-ordinator. For EUBS members for this CPD issue this will be Christian Fabricius, ***E-mail:*** <christian.fabricius@gtclifescience.se>
For ANZCA DHM SIG and other SPUMS members, this will be David Smart, ***E-mail:*** <David.Smart@dhhs.tas.gov.au>

On submission of your answers, you will receive a brief model answer for each question. 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.

Questions

1. Breathing 100% oxygen at 101.3 kPa (1.0 ATA) causes:

A. Bradycardia, decreased cardiac output and peripheral vasoconstriction;

B. Tachycardia, increased cardiac output and cerebral blood flow;

C. Bradycardia with a normal cardiac output because of the increased intra-thoracic blood volume;

D. An age-dependent decrease in cerebral blood flow, more pronounced in younger persons, and a small decrease in end-tidal CO_2;

E. An age-dependent decrease in cerebral blood flow, more pronounced in older persons, and a small decrease in end-tidal CO_2.

2. Increasing the pressure > 101.3 kPa while breathing 100% oxygen causes:

A. Linear pressure-dependent peripheral regional vasoconstriction, hypertension and decreased cerebral blood flow;

B. Increased peripheral regional vasoconstriction, hypertension and decreased cerebral blood flow only as a sign of oxygen toxicity;

C. No further peripheral regional vasoconstriction at pressures > 203 kPa and an increase in cerebral blood flow;

D. No further peripheral regional vasoconstriction at pressures > 203 kPa and a decrease in cerebral blood flow;

E. An increase in peripheral regional vasoconstriction but no change in blood pressure.

3. The haemodynamic effect of immersion seems to be different for breath-holders compared with non-breath holders. How?

A. Breath-holding decreases the intra-thoracic blood volume;

B. Breath-holding impairs left ventricular function;

C. Decreased cardiac output for breath-holders and for non-breath-holders;

D. No effect on cardiac output for breath-holders and an up to 30% increase for non-breath-holders;

E. A lower peripheral vascular resistance for breath-holders.

4. During immersion, the 'dive reflex' and the 'cold shock response' affect our ability to dive. Face-only immersion in cold water and sudden whole-body immersion in cold water can cause a 'cold shock response'. What is it?

A. A reduction in the apneic time;

B. Respiratory gasps, tachycardia and decreased cerebral blood flow;

C. Tachycardia, hyperventilation, increased cardiac output and increased cerebral blood flow;

D. Systemic and pulmonary hypertension combined with an unchanged heart rate and stroke volume;

E. Hyperventilation, bradycardia, vasoconstriction and hypertension.

5. Dehydration is common in diving because of the effects of immersion, even if the divers eat and drink before and in between the dives. After a dive to 32 metres' depth with a total immersion time of 45 minutes, what proportion of body mass would be lost?

A. No effect after just one dive;

B. < 0.5% of body mass;

C. 0.5–1.5% of body mass;

D. 1.5–2.5% of body mass;

E. 2.5–3.5% of body mass.

6. The effect of hyperbaric oxygen (HBO) on the microcirculation:

A. Vasoconstriction is dependent on the oxygen partial pressure;

B. Vasoconstriction is dependent on the blood flow and local (regional) metabolism;

C. A 25% reduction of heart rate and an increase in pulmonary oxygen uptake;

D. Effect on the microcirculation is reversible and reverts to normal 20 seconds after HBO;

E. Effect on the microcirculation is reversible and reverts to normal 20 hours after HBO.

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>

EUBS notices and news

Phypode Fellows update

Peter Buzzacott

With our joint annual scientific meeting on the horizon it appears timely to provide an update on the Physiopathology of Decompression (Phypode) International Training Network, a European project funded by the FP-7 People Marie Curie Actions – Research Fellowship Programme. Fourteen research fellows are now engaged in dovetailing projects associated with 13 facilities in eight countries. Almost all are attending La Réunion in September.

At the University of Western Brittany in Brest, France, Aleksandra Mazur, "Ola" from Poland, is using isolated blood vessels in a rat model to investigate endothelium and vascular smooth muscle injury in decompression sickness (DCS). Now at the end of her second year of PhD candidature, Ola is submitting results for publication. Kate Lambrechts, from Belgium, has been measuring micro-vessel endothelial function both in a rat DCS model and in human divers. Kate is similarly some way into PhD candidature and has had her second paper accepted. Qiong Wang, from China, has designed a hyperbaric chamber of 1 cc volume, for the *ex vivo* study of cellular responses to decompression, using bovine cells. Qiong's PhD research forms the foundation for a new physiological model of decompression. Peter Buzzacott, from Australia, is on a two-year postdoctoral fellowship, dividing his time between supporting the development of research skills and conducting research primarily aimed at identifying biomarkers for DCS susceptibility.

At the Haute Ecole Henri Spaak in Brussels, Belgium, Frauke "Fry" Tillmans is investigating the effect of normobaric oxygen on response mechanisms in human blood cell cultures, healthy and cancerous. Immune response, mitochondrial capacities, nitrosative and oxidative stress are the targets of her research, which is collaborative with university clinics in Brussels and Ulm, Germany. Walter Hemelryck, from Belgium, is sharing his time between the military hospital in Brussels and the laboratory at the Haute Ecole Paul Henri Spaak. His research relates to narcosis, cold, neurology, exercise and ergonomics.

Also in Brussels, Virginie Papadopoulou is enrolled in a PhD from the Bioengineering Department of Imperial College London, UK, on mathematical modelling of bubble formation and decompression. Virginie has developed in MatLab™ a diving simulation platform for diving profiles, automated VGE counting for post-dive echocardiography and an experimental set-up for bubble growth observation. Miroslav Rozloznik, originally Slovakian, is researching exceptional exposure dives and the effects of preconditioning upon venous gas embolism. With a PhD in sports physiology, "Miro" supports the development of the postgraduate students in Brussels.

Alternating between Sharm El Sheikh, Egypt and DAN Europe at Roseto, Italy, Egyptian Amir Gerges is researching deep stops for a Masters in underwater medicine through the University of Stellenbosch, South Africa, and analysing the DAN Europe database to identify risk factors for diving injuries. In Gdansk, Poland, Ukrainian anesthesiologist and intensive care doctor Yurii Tkachenko is measuring oxidative stress during hyperoxia using exhaled breath condensate.

At Acreo in Austria, German Andreas Shuster is developing novel instrumentation to measure physiological parameters (ECG, blood pressure, breathing parameters) on divers underwater. The goal is to measure those parameters simultaneously and store them with dive profile data for later assessment. Andreas is enrolled in a PhD programme at the local medical university.

In Split, Croatia, American Dennis Madden is studying the relationship between exercise, diving and arterialization, also for a PhD. His research examines intense exercise before diving, microparticle production and how these factors impact gas emboli and decompression stress.

At the Mares facility in Rapallo, Italy, Bulgarian Georgi Popov is adapting decompression schedules to the physiology of divers in real time, accounting for hydration, fatigue and a variety of other conditions that affect how the human body responds to dive stress. Also in Italy, at Trieste, Mirac Memisoglu is developing devices allowing composition control of breathing mixtures during dives. Originally from Turkey and specialising in information technology, Mirac is investigating sensor technology and field measurements in special environments.

Collectively this group is engaged in research from the cellular level, through animal models to individual human divers and, ultimately, to large diving population datasets. There is much yet remaining to discover about decompression sickness; controversies to address, hypotheses to test, improvements to be made. So, while the recreational diving population generally appears to be aging it may be comforting to some that a new generation of decompression researchers are steadily making progress and forming international collaborations along the way.

In addition to developing scientific research skills, nearly all of us are learning at least one new language. While there are fourteen languages within the group, fortnightly Skype conferences are made in English. For more information about the team and the Phypode Project, including a list of publications, please visit: < http://www.phypode.org/>.

Peter Buzzacott, PhD, is a Marie Curie Experienced Research Fellow, Optimisation des Régulations Physiologiques (ORPhy) Laboratoire Université de Bretagne Occidentale, Brest, France, and Adjunct Lecturer, School of Sports Science, Exercise and Health, The University of Western Australia, Perth, Australia.

Key words
Research, diving research, hyperbaric research, decompression sickness, general interest

Figure 1
Left-to-right: Qiong Wang, Walter Hemelryck, Amir Gerges, Yurii Tkachenko, Georgi Popov, Frauke Tillmans, Dennis Madden, Aleksandra Mazur, Kate Lambrechts, Virginie Papadopoulou, Miroslav Rozloznik, Peter Buzzacott
(missing are Andreas Schuster and Mirac Memisoglu; photo taken in Rapallo, Italy, June 2013)

REUNION 2013
TRICONTINENTAL SCIENTIFIC MEETING ON DIVING AND HYPERBARIC MEDICINE
22–29 September

Preparations are now complete for the Tricontinental on Réunion Island.

Programme:

22 September	ECHM Workshop: *Diagnosis and treatment of mild decompression sickness in remote diving destinations*
23–28 September	Tricontinental Scientific Meeting on Diving and Hyperbaric Medicine
28 September	SPUMS, EUBS, SAUHMA General Assemblies
29 September	International DAN Diver's Day

The full programme is available on the conference website:
<www.reunion2013.org>
All enquiries: <info@reunion2013.org>

EUBS SPUMS Scott Haldane Diving Medicine

40th Wiesbaden, September 24th-27th
EUBS 2014
40th Annual Scientific Meeting of the European Underwater & Baromedical Society (EUBS)

Preliminary Announcement

Dates: 24–27 September 2014
Venue: Wiesbaden, Germany

The 40th EUBS Annual Scientific Meeting will be held in conjunction with the 2014 congress of the German Society for Diving and Hyperbaric Medicine (GTUeM) in Wiesbaden/Germany (near Frankfurt/Main). Dr Peter Müller has been appointed by both societies to serve as the Secretary General for the meetings.

Organising Committee
Dr Peter Müller (Secretary General), Dr Peter Germonpré (EUBS), Dr Karin Hasmiller (EUBS, GTUeM), Michael Kemmerer (EUBS, VDD, Wiesbaden), Dr Dirk Michaelis (EUBS, GTUeM, Wiesbaden)

Main topics

- Diving medicine – physiology, decompression theory and treatment
- HBO medicine – physiology and treatment
- Technical aspects, safety
- Pro/Con debate on DFS
- Guideline: Treatment of diving accidents

1st Announcement and Call for Abstracts will be in the December 2013 issue of DHM.
The Congress Secretariat will also then become available by telephone to assist you.

For further information and hotel bookings at this early stage see: <www.eubs.2014.org>
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EUBS European Underwater and Baromedical Society

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SPUMS Notices and news

SPUMS Diploma in Diving and Hyperbaric Medicine (updated March 2013)

Requirements for candidates

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

- The candidate must be medically qualified, and be a current financial member of the Society.
- The candidate 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 approved facilities providing two-week courses may be found on the SPUMS website.
- The candidate 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.
- The candidate 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.
- The candidate 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 written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

In the absence of documentation otherwise, it will be assumed that the paper is submitted for publication in *Diving and Hyperbaric Medicine*. As such, the structure of the paper needs to broadly comply with the 'Instructions to Authors' – full version, July 2011, 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 (e-mail is acceptable) to advise of their intended candidacy, and to discuss the proposed subject matter of their research. A written research proposal must be submitted before commencing 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 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 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 documented evidence of approval by an appropriate research ethics committee. 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.

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.

The Academic Board reserves the right to modify any of these requirements from time to time. As of October 2012, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer;
Associate Professor Simon Mitchell;
Associate Professor (retired) Mike Davis.

All enquiries and applications should be sent to:
Associate Professor David Smart
GPO Box 463, Hobart, Tasmania 7001
E-mail: *<david.smart@dhhs.tas.gov.au>*

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

ANZHMG Chairman's report

Medicare funding for hyperbaric oxygen therapy

This will be my last report as chairman of the ANZHMG, a position I have held now for a decade. The decade has been quite a battle, occupying many countless hours of time for Mike Bennett and myself, to try to preserve Medicare funding for patients being treated with hyperbaric oxygen (HBO). In that battle, we have had some success, preserving some funding for our patients, and a significant loss. In November last year, the Australian Federal Government removed funding for HBO treatment of non-diabetic problem wounds. The decision demonstrated very serious flaws in process when evidence-based medicine is misused to control costs and to review existing funded services.

These flaws were publicly aired at a Senate Standing Committee on Finance and Public Administration References Committee Inquiry into Medicare funding for Hyperbaric Oxygen Treatment held in November 2012. Following the hearing a detailed report was released,[1] in which the committee made several recommendations that vindicated the concerns of the ANZHMG:

Recommendation 1

2.82 The committee recommends that the Government continue Medicare Benefits Schedule interim funding for Hyperbaric Oxygen Treatment for non-diabetic wounds until the current randomised control trial is completed and assessed by the Medical Service Advisory Committee.

2.83 The committee also considers that there is a need for an independent review process to be established for MSAC decisions, using a similar approach to the independent review of the PBAC.

Recommendation 2

2.84 The committee recommends that an independent review process be established for decisions by the Medical Service Advisory Committee using a similar approach to the independent review of the Pharmaceutical Benefits Advisory Committee.

The Federal Labor party immediately issued a dissenting report citing the same rhetoric that had been promulgated by MSAC over the preceding five years. They ignored the Senate report findings and despite significant adverse findings regarding MSAC process, Medicare funding was not reinstated. The Minister has subsequently claimed in answer to questions on notice, that the clinical experts (including Smart and Bennett) unanimously supported the MSAC decision. Nothing could be further from the truth.

Finally, the roots of the Consumer Health Forum of Australia (CHFA) submissions were exposed in the report. The CHFA had not consulted a single consumer prior to making their adverse submission. In addition, it was revealed that they receive the majority of their funding from DOHA.

Associate Professors Smart and Bennett have no doubt that hyperbaric medicine was used as a test case for the Government to iron out the 'chinks in the armour' so they can take on the medical profession and 'disinvest' in the Medicare Schedule, citing 'evidence' as the guiding principle. Our view has been vindicated with the recent announcement that the next speciality for review will be Ear Nose and Throat services. We wish our ENT colleagues the best of luck as they are railroaded into the flawed MSAC process.

The final danger is that the findings of health technology reviews are shared around the world and erroneously given high status in the world of evidence. There is no doubt that the MSAC 1054.1 report will be quoted by bureaucrats in other countries. Unfortunately the dissenting report by Bennett and Smart was not attached to 1054.1, and is only on the MSAC website: <http://www.msac.gov.au/internet/msac/publishing.nsf/Content/1054.1>.

Reference

1 Finance and Public Administration References Committee. Medicare funding for Hyperbaric Oxygen Treatment. November 2012. Commonwealth of Australia. ISBN 978-1-74229-726-2.

Accreditation of hyperbaric facilities in Australia and New Zealand

This has not progressed any further, while the profession is waiting for AS 4774.2 to be revised (see below).

Australian Standards Report – Occupational Diving

There has been a huge amount of activity in relation to standards this year, with all standards that relate to diving and hyperbaric medicine being revised en bloc by Standards Australia. For those of us on the committees, it has resulted in Standards overload!

AS/NZS 2299 SERIES – OCCUPATIONAL DIVING OPERATIONS. STANDARD OPERATIONAL PRACTICE

After the change of Standards Australia to SAI Global, the 2299 series of standards requires sponsorship prior to further update. The 2299.1 standard is now referenced in the Federal Government Model Work Health and Safety Regulations as the appropriate standard for construction diving but there remains a serious disconnect between the work practices covered by AS/NZS 2299.1 and the legislation. The standard has been modernised to reflect changes in Australian Work Health and Safety Legislation and other standards such as 2815.5 and 2815.6. The Standard will be released for public comment soon.

The scope of AS/NZS 2299.1 has been widened to include nitrox diving. Guidance on delay to altitude exposure has been moved to an informative appendix rather than being

seen as a rigid set of rules to be followed. The appendix would be used when a specific protocol has not been produced for a given set of diving conditions. The values for travel to altitude provided in this Standard are generally consistent with other published guidelines.

The medical appendix has been updated, with minimal change to the medical documents. It was recognized that these documents serve as an entry point for discussion and collection of medical information. It was also recognized that there are two separate systems in operation in Australia and New Zealand. New Zealand has a centralized registry, whereas Australia operates a decentralized system, and defers to its recent WHS legislation.

The hygiene section has been updated, and there is a new section covering gauge calibration. Requirements for on-site chamber support for diving have been altered to reflect risk in certain types of work or factors that significantly increase the risk of arterial gas embolism or high gas load/ rapid progression decompression illness.

AS/NZS 2815.2 TRAINING & CERTIFICATION OF OCCUPATIONAL DIVERS, PART 2: AIR DIVING TO 30 M
AS/NZS 2815.5 TRAINING AND CERTIFICATION OF OCCUPATIONAL DIVERS, PART 5: DIVE SUPERVISOR

These standards have now been updated and have received public comment.

AS/NZS 2815.6 RESTRICTED OCCUPATIONAL SCUBA DIVER

This is a new standard covering occupational diving in low risk settings. The Standard specifies the training activities and competencies required for entry-level training and certification of divers to work safely and competently at a limited range of underwater tasks as members of an occupational diving team using scuba. The final draft of this standard has been prepared and will be released for public comment in August. This standard is not intended to train divers for surface-supply diving, construction diving or diving in hazardous diving conditions. Hazardous diving conditions were defined as the following:

- risk of entanglement – diving in and around nets and cages, multiple ropes and lines, tree branches, man-made underwater structures such as shipwrecks, sunken vehicles, or other sunken material;
- diving in an overhead environment, without vertical access to the surface;
- highly limited or zero visibility;
- work near outflow or inflow to pipes;
- diving in currents or fast flowing creeks, rivers and drains;
- diving associated with setting of weights or moorings or use of lifting devices for anything other than fish or shellfish;
- work in high boat-traffic areas such as navigation channels, entries to marinas, operational ports;
- use of plant powered from the surface;
- decompression diving;
- diving deeper than 30 metres.

AS 4005.1 TRAINING AND CERTIFICATION OF RECREATIONAL DIVERS PART 1: MINIMUM ENTRY-LEVEL SCUBA DIVING

This recreational diving training standard traditionally had the recreational diving medical forms attached to it. Recreational training agencies did not support the medical section of the Standard and they have moved to adopt international standards which appeared to be of a lower quality. As such, the Standard 4005.1 appears to be in 'no man's land'. The medical section has now been replaced by the updated SPUMS diving medical (2010). In the last 12 months, lobbying by training agencies was successful in removing compulsory diving medicals for recreational divers in Queensland. This has been met with jubilation by some: "*SSI prides itself on being leaders in the recreational dive industry. We have and continue to fight hard for increased diver safety but at the same time reduce the unnecessary barriers to entry. We have had a long held view that in line with RSTC guidelines a dive medical should only be required if indicated by a medical pre assessment. We strongly believe that the cost, time and inconvenience of a dive medical for the majority of people is a significant and unnecessary barrier.*" Time will tell if the unnecessary barrier was unnecessary.

AS 4774.2 WORK IN COMPRESSED AIR AND HYPERBARIC FACILITIES - HYPERBARIC OXYGEN FACILITIES

The HTNA has sponsored this standard that covers the safe operation of hyperbaric facilities. The ANZHMG is working to assist the review, which commenced this year. A draft for public comment is close to completion and should be released some time after a meeting to be held in August. Australian Federal Government Work Health and Safety Regulations make no reference to either AS 4774.1 or 4774.2, and hence workers in compressed air or hyperbaric facilities are not classified as divers.

Following completion of the current series of standards (end of 2013), I will be stepping down from the position of SPUMS representative, Occupational Diving Standards, which I have occupied for over a decade. It has been an interesting ride. I thank everyone for the opportunity to represent SPUMS in this important area.

David Smart, Hon. Chairperson, ANZHMG
E-mail: *David.Smart@dhhs.tas.gov.au*

Key words
Hyperbaric oxygen therapy, occupational diving, standards, meetungs, medical society

The SPUMS Annual General Meeting 2013, Notice of Meeting

The AGM for SPUMS 2013 will be held at La Tamarun Convention Centre, La Saline Les Bains, Réunion Island at 0900 h on Saturday 28 September 2013.

Agenda

1. Apologies
2. Minutes of the previous meeting:
   Minutes of the previous meeting will be posted on the notice board at La Tamarun and were published in *Diving and Hyperbaric Medicine*. (Minutes of the Annual General Meeting of SPUMS held at Madang Resort, Papua New Guinea, at 1500 h, Thursday 24 May 2012. *Diving Hyperb Med*. 2012;42:187-8. Officers' reports are on the SPUMS website <www.spums.org.au>).
3. Matters arising from the minutes:
4. Annual reports:
   President's report
   Secretary's report
   Educations Officer's report
   Treasurer's report and annual financial statement
   Journal Editor's report
5. Subscription fees for 2014:
   Treasurer
6. Election of office bearers:
   The date for nominations will have closed by the time this issue is posted out.
7. Appointment of the Auditor 2014:
   Treasurer
8. Business of which notice has been given:
   Motion to change paragraphs 39, 40 and 41 (Publications and publicity) of the Purposes & Rules of the Association
   Proposed: M Davis, seconded: M Bennett.

Karen Richardson, Secretary

Key words
Medical society, meetings

The

SPUMS

website is at
<www.spums.org.au>

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

SPUMS Annual Scientific Meeting 2014

Venue: Alila Manggis Resort, Bali
Dates: 18–25 May 2014

Themes: Patent foramen ovale (PFO); immersion pulmonary oedema; the older diver

Keynote Speaker: Dr Peter Wilmshurst, Cardiologist, UK

Dive sites: Tulamben (*SS Liberty* wreck); Nusa Penida/ Lembongen (Crystal Bay; Manta Point); Padang Bai

Lots of activities for non-divers (day spa, cooking school, shopping trips, volcano tours, Ubud, snorkelling and more). Alila Resort <www.alilahotels.com/manggis> has only 55 seaside rooms surrounding a fabulous pool. Pre- and post-conference options are extensive!

Further details including how to book and a Call for Abstracts will be on the SPUMS website soon.

Dr Neil Banham, Convenor

SPUMS and Facebook

I have recently been appointed the administrator of the SPUMS Facebook (FB) site. SPUMS has had a corporate page on the site since June 2011 and it can be viewed at: http://www.facebook.com/pages/SPUMS-South-Pacific-Underwater-Medicine-Society/221855494509119

FB is an excellent tool to disseminate information, via social media, to a mass audience. Current 'followers' of our FB page include medical/nursing personnel, as well as members of the general public. I am keen to increase the amount of information on the SPUMS FB page, and need the help of SPUMS members to do so.

I recently posted an ABC News video covering some research into nitrox diving being performed at the Townsville chamber. Within 24 hours, it had reached over 600 people, and been 're-posted' by a number of other sites, including Rubicon Foundation. This, I believe, is fairly impressive for a society such as SPUMS, and can only improve.

I would appreciate any news, articles, videos or photos from your units about diving and hyperbaric medicine being posted on the SPUMS FB page (remembering it can be seen by everyone). For those of you who have a FB account, please feel free to post yourself. Alternatively, they can be e-mailed to me at <clinton_gibbs@live.com>. If you have any questions/concerns, please do not hesitate to contact me. I look forward to hearing from you.

Clinton Gibbs
Townsville Hospital Hyperbaric Unit

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.

Eligibility criteria are:

1. Fellowship of a Specialist College in Australia or New Zealand. This includes all specialties, and the Royal Australian College of General Practitioners.
2. Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least four weeks' total duration. For example, one of:
   a ANZHMG course at Prince of Wales Hospital Sydney, **and** Royal Adelaide Hospital or HMAS Penguin diving medical officers course **OR**
   b Auckland University Postgraduate Diploma in Medical Science: Diving and Hyperbaric Medicine.
3. **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including six months' fulltime equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS **AND**
   b Completion of a further 6 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.

   OR:
   c Completion of 12 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA **AND**
   d Completion of a formal project in accordance with ANZCA Professional Document TE11 "*Formal Project Guidelines*". The formal project must be constructed around a topic which is relevant to the practice of diving and hyperbaric medicine, and must be approved by the ANZCA Assessor prior to commencement.
4. Completion of a workbook documenting the details of clinical exposure attained during the training period.
5. Candidates who do not hold an Australian or New Zealand specialist qualification in Anaesthesia, Intensive Care or Emergency Medicine are required to demonstrate airway skills competency as specified by ANZCA in the document "*Airway skills requirement for training in Diving and Hyperbaric Medicine*".

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

Dr Suzy Szekely, FANZCA
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine, Australia
E-mail: *<Suzy.Szekely@health.sa.gov.au>*

The ADAS Derek Craig Award (ADAS Safety Award)

The ADAS Derek Craig Award is awarded annually to recognise those who have improved occupational diver safety. It is a perpetual award overseen by the Australian Diver Accreditation Scheme (ADAS) and it recognises innovation or exceptional effort to advance the safety of the working diver.

The 2013 award has been presented to Associate Professor David Smart (Figure 1), not only in recognition of his research and medical contribution to the diving industry, but also for his practical work with aquaculture divers and the seafood industries in the development of safer work practices. This award also recognises Dr Smart's ongoing support and involvement in diver and diver medic training and the review and development of diving standards.

Figure 1
Associate Professor David Smart at the door of the Hobart Hospital recompression chamber, Hobart, Tasmania

The Poetry Doctor

Losing virginity

He was just a lad, young and gung ho,
Who was captivated by the stories of a Captain Jacques Cousteau.
Which fired his imagination and sparked his kindling ego,
Flaming him to action – a-diving he would go.

Finding a local dive club, too poor to travel away,
He started his new adventure diving "*Pembrokeshire, UK*".
Alas, it was a wintery March, the cold a bitter pill
But was reassured the club's drysuit would alleviate the chill.

At the Dale Fort Field Centre, he sank his very first dive
By joining a queue for the drysuit ... (and he was number five).
He watched each novice diver endure a squirming ordeal,
Fitting the club's one drysuit, squeezing through the neck seal.

At last it was his turn to perform this 'donning show'
Its innards awash with sea water, sweat and (I don't want to know!).
But once it was fully fitted and the seal tight to his neck
He totally forgot his discomfort and was straight to his buddy check.

All kitted and fitted and ready to dive he walked into the water
Still pure, innocent and naïve, the proverbial lamb to the slaughter.
A virgin diver entering his first underwater dip,
Each moment a frame captured in a nostalgic cartoon strip.

His memories are still vivid, "*10 minutes at 15 feet*",
"*Ear pain on descent*" and "*nothing of note"* to meet
For the "*vis was only 2 foot*", there was "*nothing of interest to see*"
Distracted by the effort of resisting the impulse to p..

It was all over so quickly, premature exhilaration at heart
For he was so thrilled and ecstatic and this was only the start.
He has avidly dived ever since, his prowess as ever nifty,
This year is his diving anniversary, a golden, number fifty!

He is still a promiscuous diver; wherever, whenever he can
And plans to keep on diving. It's part of his retirement plan.
I'm sure you have guessed the identity of this aquatic man, myth-like
It's none other than our amazing, prodigious editor Mike.

John Parker
E-mail: *<drjohnparker@hotmail.com>*

Editors note: I am somewhat embarassed by this. When I asked for a poem for this issue, I foolishly mentioned to John that I was celebrating 50 years' scuba this year and quoted from my old BSAC logbook my comments for my first dive on a cold March day in south-west Wales in 1963. Little did I realise he would turn this into verse!

Wikipedia

EUBS is mentioned in an article in the "Did you know" section of the English Wikipedia front page.
It is on the left-hand side below the featured article:
http://en.wikipedia.org/wiki/Main_Page.
Go check it out!

SPUMS has a Wikipedia entry too.
Go to: http:// http://en.wikipedia.org/wiki/South_Pacific_Underwater_Medicine_Society

18th International Congress on Hyperbaric Medicine

03–06 December 2014
Buenos Aires, Argentina

Dear Friends and Colleagues,
On behalf of the Organizing Committee of the **18th International Congress of Hyperbaric Medicine**, we would like to extend a warm invitation to attend the scientific sessions in Buenos Aires, Argentina to be held at the Catholic University of Argentina School of Medicine.

The congress programme will be built on a structured series of invited lectures and will be open to independent contributions in the traditional oral presentation and abstract formats. Main topics will include:

- Basic research relevant to mechanisms of hyperbaric oxygen therapy
- Current concepts and future directions of hyperbaric oxygen therapy
- Current concepts and future directions in diving research and operations

In addition to the regular programme, several pre-congress courses are at the planning stage.

The ICHM is a world-wide organization for physicians and scientists interested in all aspects of diving and hyperbaric medicine. The organization has minimal formal structure and is entirely dedicated to hosting an international scientific congress every three years with the purpose of improving understanding among the international hyperbaric community.

ICHM Committee (2011–2014):
President: Prof Dr Jorge B Pisarello (Argentina)
Executive Director: Dr Alessandro Marroni (Italy)
Secretary: Assoc Prof Michael Bennett (Australia)

Congress website: <http://ichm.drupalgardens.com/content/what-ichm-0>
University website: <www.uca.edu.ar>

Registration:
Please use on-line registration on our website, beginning 30 November 2013

Registration type	Until 30 April 2014	After 30 April 2014
Members of UHMS, EUBS, SPUMS	U$D 550	U$D 650
Non-Members	U$D 600	U$D 750
Nurse, Technician, Student	U$D 250	U$D 300
Accompanying person	U$D 200	U$D 250

Buenos Aires is a friendly and charming city with a rich history and architecture. The city is within easy reach of unique locations, such as the Iguazu Falls, Perito Moreno Glacier, the vineyards of Mendoza as well as diving sites with sea lions in the South of Argentina. During your stay you will surely be able to enjoy Argentinian cuisine and share our passion for tango, local folklore and other expressions of Argentine culture.

The ANZ Hyperbaric Medicine Group Introductory Course in Diving and Hyperbaric Medicine 2014

Dates: 24 February–07 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 as a CPD Learning Project by ANZCA: Cat 2, Level 2 – 2 credits per hr (approval no. 1191).

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>

Royal Adelaide Hospital Hyperbaric Medicine Unit Courses 2013

Medical Officers' Course
Week 1: 02–06 December
Week 2: 09–13 December

All enquiries to:
Lorna Mirabelli
Senior Administrative Asst/Course Administrator
Hyperbaric Medicine Unit
Level 2, Theatre Block
Royal Adelaide Hospital
North Terrace, Adelaide, SA 5000
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lorna.Mirabelli@health.sa.gov.au>

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

P O Box 347, Dingley Village
Victoria, 3172, Australia
E-mail:
<deswill@dingley.net>
Website:
<www.classicdiver.org>

Royal Australian Navy Medical Officers' Underwater Medicine Course 2013

Dates: 11–22 November 2013
Venue: HMAS PENGUIN, Sydney

The MOUM course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses. The course includes scenario-based simulation focusing on management of diving emergencies and workshops covering the key components of the diving medical.

Cost: AUD705 without accommodation
(AUD1,600 with accommodation at HMAS Penguin)

For information and application forms contact:
Rajeev Karekar, for Officer in Charge,
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Rd, Mosman
NSW 2088, Australia
Phone: +61-(0)2-9647 5572
Fax: +61-(0)2-9960 4435
E-mail: <Rajeev.Karekar@defence.gov.au>

ICASM 2013
The 61st International Congress of Aviation and Space Medicine

Dates: 06–10 October 2013
Venue: Inbal Hotel, Jerusalem, Israel

For further information: <www.icasm2013.org>

Canadian Chapter of the Undersea and Hyperbaric Medical Society Annual Meeting

Dates: 01–03 November 2013
Venue: Lord Nelson Hotel, Halifax, Nova Scotia

Topics include:
Dysbaric osteonecrosis
Fitness to dive
Dive computers
Medications and diving update
Stop time distribution in air decompression

For further information:
<http://www.cc-uhms.ca/wp/contact-us/>

Scott Haldane Foundation

The Scott Haldane Foundation is dedicated to education in diving medicine, and has organized more than 150 courses over the past 15 years, both in the Netherlands and abroad. Below is a list of remaining courses for 2013.

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

Remaining courses for 2013

09–16 November: Medical Examiner of Divers Part I. Dauin, Philippines
16–23 November: Fitness to dive in normal and extreme conditions.* Dauin, Philippines
23–30 November: Fitness to dive in normal and extreme conditions.* Malapascua, Philippines

Topics of the two November in-depth courses are: *Diving and diabetes, diving and epilepsy, diving under extreme conditions*, presented by Michael Bennett and Michael Lang.

For further information: <www.scotthaldane.org>

German Society for Diving and Hyperbaric Medicine (GTUeM)

An overview of basic and refresher courses in diving and hyperbaric medicine, accredited by the German Society for Diving and Hyperbaric Medicine (GTUeM) according to EDTC/ECHM curricula, can be found on the website:
<http://www.gtuem.org/212/Kurse_/_Termine/Kurse.html>

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 or computer for later viewing.

We offer video lectures from:
- The 5th Karolinska PG course in clinical hyperbaric oxygen therapy, 07 May 2009
- The European Committee for Hyperbaric Medicine "*Oxygen and infection"* Conference, 08–09 May 2009
- The 17th International Congress on Hyperbaric Medicine, Cape Town, 17–18 March 2011

Also available is the 2011 Stockholm County Council report: *Treatment with hyperbaric oxygen (HBO) at the Karolinska University Hospital*

For further information contact:
Folke Lind, MD PhD
E-mail: <folke.lind@karolinska.se>
Website: <www.hyperbaricoxygen.se>

DAN Europe

DAN Europe has a fresh, multilingual selection of recent news, articles and events featuring DAN and its staff.
It can be accessed at:
<http://www.daneurope.org/web/guest/>

Keeping the whole DAN Europe family updated with what is going on...enjoy!

Instructions to authors

(Short version, updated June 2013)

Diving and Hyperbaric Medicine (*DHM*) welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *DHM*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts will be subject to peer review. Accepted contributions will also be subject to editing. An accompanying letter signed by all authors should be sent.

Contributions should be sent to:

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

Individual correspondence should be addressed to:

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

Requirements for manuscripts

Documents should be submitted electronically. The preferred format is Microsoft® Office Word or rich text format (RTF). Paper submissions will not be accepted. All articles should include a title page, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address. The text should generally be subdivided into the following sections: a structured Abstract of no more than 250 words, Introduction, Methods, Results, Discussion, Conclusion(s), Acknowledgements and References. Acknowledgements should be brief. Legends for tables and figures should appear at the end of the text file after the references. Conflicts of interest and funding sources should be identified.

The text should be 1.5 lines spaced, using both upper and lower case. Headings should conform to the current format in *DHM*. All pages should be numbered. Underlining should not be used. SI units are to be used (mmHg is acceptable for blood pressure measurements; bar for cylinder pressures); normal ranges should be shown. Abbreviations may be used after being shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

Preferred length for **Original Articles** is up to 3,000 words. Inclusion of more than five authors requires justification, as does that of more than 30 references. **Review Articles** are welcomed. **Case Reports** should not generally exceed 1,500 words, and a maximum of 15 references. Abstracts are required for all articles. **Letters to the Editor** should not exceed 500 words and a maximum of five references. Legends for figures and tables should generally be shorter than 40 words in length.

Illustrations, figures and tables must NOT be embedded in the word processor document, only their position in the text indicated, and each should be submitted as a separate file.

Tables should be presented either with tab-separated columns (preferred) or in table format. No gridlines, borders or shading are to be used.

Illustrations and figures should be submitted in greyscale TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file. Give careful thought to fonts being of an adequate size for legibility.

Printed **photographs** should be glossy black-and-white. Colour is available only if essential and will be at the authors' expense. Indicate magnification for photomicrographs.

References

The *DHM* Journal reference style is based closely on the *International Committee of Medical Journal Editors (ICMJE) Uniform Requirements for Manuscripts*. To ensure the correct formatting of all types of citation go to: <http://www.nlm.nih.gov/bsd/uniform_requirements.html> (last updated July 2011). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] Number them in order of quoting (including in tables). Use Index Medicus abbreviations for journal names: <http://www.nlm.nih.gov/tsd/serials/lji.html>

Examples of the exact format for a standard paper and a book are given below:

1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol*. 1972;95:556-63.

2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

Accuracy of references is the responsibility of the authors.

Manuscripts not complying with the above requirements will be returned to the author(s) before consideration.

Consent and ethics

Studies on human subjects must state that they comply with the Declaration of Helsinki (1964, revised 2008) and those using animals must comply with health and medical research council guidelines or their national equivalent. A statement affirming ethics committee (institutional review board) approval should be included in the text. A copy of that approval (and consent forms) should be provided.

Copyright

Authors must agree to accept the standard conditions of publication. These grant *Diving and Hyperbaric Medicine* a non-exclusive licence to publish the article in printed form in *Diving and Hyperbaric Medicine* and in other media, including electronic form; also granting the right to sub-licence third parties to exercise all or any of these rights. *Diving and Hyperbaric Medicine* agrees that in publishing the article(s) and exercising this non-exclusive publishing sub-licence, the author(s) will always be acknowledged as the copyright owner(s) of the article.

Full instructions to authors (revised July 2011) may be found on the DHM Journal, EUBS and SPUMS websites and must be consulted in preparing a submission.

DIVER EMERGENCY SERVICES PHONE NUMBERS

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

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

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

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

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

UNITED KINGDOM
+44-7740-251-635

USA
+1-919-684-9111

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

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>

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>

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