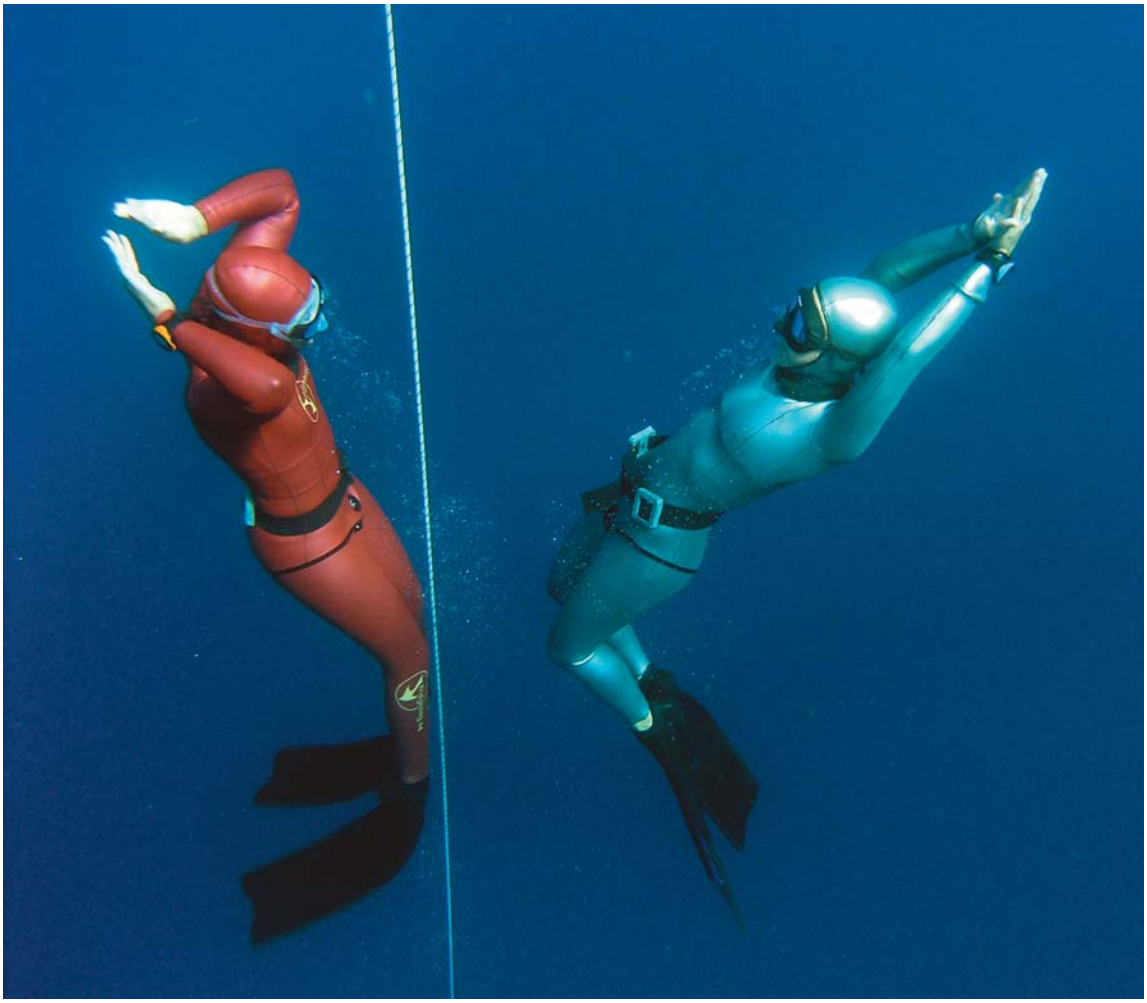
Diving and Hyperbaric Medicine

The Journal of the South Pacific Underwater Medicine Society (Incorporated in Victoria) A0020660B and the European Underwater and Baromedical Society

SPUMS

Volume 39 No. 2 June 2009

EUBS



Extreme breath-hold diving

Decompression stress in recompression chamber attendants

Health surveillance of recompression chamber attendants

Exhaled nitric oxide after hyperbaric oxygen treatment

Adjunctive therapy for decompression illness

ISSN 1833 - 3516
ABN 29 299 823 713

Print Post Approved
PP 331758/0015

PURPOSES OF THE SOCIETIES

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

SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

OFFICE HOLDERS

President
Mike Bennett <M.Bennett@unsw.edu.au>
Past-President
Chris Acott <cacott@optusnet.com.au>
Secretary
Sarah Lockley <spumssecretary@gmail.com>
Treasurer
Jan Lehm <LehmJ@SESAHS.NSW.GOV.AU>
Education Officer
David Smart <david.smart@dhhs.tas.gov.au>
Public Officer
Vanessa Haller <vanessa.haller@cdmc.com.au>
Chairman ANZHMG
David Smart <david.smart@dhhs.tas.gov.au>
Committee Members
Glen Hawkins <hawkeye@swiftdsl.com.au>
Scott Squires <scottandsandsquires@bigpond.com>
Guy Williams <spums@fastmail.net>

ADMINISTRATION

Membership
Steve Goble <spumsadm@bigpond.net.au>

MEMBERSHIP

For details of the different types of SPUMS membership and further information on the Society, or to complete a membership application, go to the Society's **website:** <www.SPUMS.org.au>
or **E-mail:** <spumsadm@bigpond.net.au>
The official address for SPUMS is:
c/o Australian and New Zealand College of Anaesthetists,
630 St Kilda Road, Melbourne,
Victoria 3004, Australia

EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY

OFFICE HOLDERS

President
Alf Brubakk <alf.o.brubakk@ntnu.no>
Vice President
Peter Germonpré <peter.germonpre@mil.be>
Immediate Past President
Noemi Bitterman <noemib@tx.technion.ac.il>
Past President
Ramiro Cali-Corleo <irocali@daneurope.org>
Honorary Secretary
Joerg Schmutz <joerg.schmutz@hin.ch>
Member at Large 2008
Dr Peter Knessl <pknessl@bluewin.ch>
Member at Large 2007
Phil Bryson <phil.bryson@ddrc.org>
Member at Large 2006
Maide Cimsit <mcimsit@istanbul.edu.tr>

ADMINISTRATION

Honorary Treasurer & Membership Secretary
Patricia Wooding
16 Burselm Avenue, Hainault, Ilford
Essex, IG6 3EH, United Kingdom
Phone & Fax: +44-20-85001778
E-mail: <patriciawooding@btinternet.com>

For further information on EUBS and to complete a membership application go to the Society's **website**: <www.eubs.org>
or **E-mail:** <patriciawooding@btinternet.com>

DIVING and HYPERBARIC MEDICINE
The Journal of the South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society

Invited editorial

The further development of medical support for professional diving

David Elliott

SPUMS was in the forefront of those who established the need for a regional registry of doctors who were competent to decide on an individual's fitness to dive. It was agreed that this would include the names of only the medical practitioners who had attended an approved course in diving medicine. In contrast, the fitness of sport divers in much of the world was determined by each recreational training agency. For divers wishing to work in the North Sea, the medical examination needed to be conducted by a doctor approved by the government of that national sector. There was much reciprocation between the European countries involved in the North Sea for the approval of suitable doctors to follow the various national standards and, because offshore divers tend to follow contracts and the seasons around the world, this recognition was also extended to some doctors outside Europe. All went well until one erroneous overseas decision demonstrated to the national lawyers that their government had no powers of medical audit or investigation beyond their own borders. As a result, in 2001, the Health and Safety Executive in the United Kingdom removed doctors outside of the UK from the designated list.

From this arose the need for international recognition of fitness standards for working divers by the European Diving Technology Committee (EDTC) and, jointly with the European Committee of Hyperbaric Medicine (ECHM), of the training required by examining doctors (at Level I) with content for working divers audited by DMAC. Although the style and frequency, and even some details about content, of such medical examinations may vary internationally, the principles are effective and so perhaps now it is only the need for periodical refresher training for 'approved' doctors that needs wider implementation.

The training of doctors to manage diving accidents and illnesses is still evolving but common to both recreational and working divers is the treatment of those admitted for recompression by a land-based chamber (e.g., EDTC-ECHM: Level II basic). After this beginning, the further training separates into those with responsibilities for working divers (Occupational, Level IIa) and those in clinical HBO therapy (Hyperbaric Medicine, Level IIb). Many doctors become dually qualified. The application of occupational medicine to all working divers plus the use of different equipment and procedures from those used in recreational and technical diving, have led to continued developments in training for doctors who need to be 'on call' for diving companies and employed divers. In many countries, there are no Level IIa courses, possibly because the naval and academic course providers who cover the needs of military, recreational and some occupational diving have neither the hours nor the budget to expand them. All this lies behind parts of the 'Opinion' section in this issue.

The DMAC 'top-up' course in Malaysia last December was run with the support of the Asian Hyperbaric and Diving Medical Association for the benefit of international doctors well experienced in the management of recreational diving accidents. For those readers who are not familiar with the broad challenges of working dives, the following are some of the topics covered by Level IIa courses:

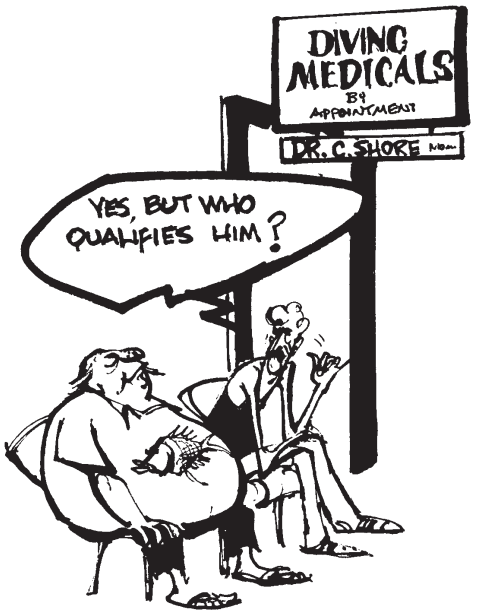
- The multiplicity of different underwater tasks
- The need
  - to complete each specific task effectively
  - to follow agreed diving procedures
  - to dive how and when required to do so
- In-water control of the diver by a surface supervisor
- Line-management's responsibilities for the diver's health and safety
- pO_2 limits in nitrox diving; on-line monitoring; management strategies for seizures
- Surface decompression, its advantages and safety constraints (e.g., hot-water suits)
- DCS is rare in commercial diving and PB/AGE almost unknown. Nevertheless a requirement is for chamber availability without delay (often on site)
- Fitness to return to all diving and associated duties after illness, surgery or injury
- Workplace assessments:
  - risk elimination, avoidance and control
  - acceptance of residual risk
  - maintenance of exposure and diving records
  - individual health surveillance after exposures to specific hazards
- Long-term health effects (neurological, pulmonary, NIHL, bone, etc)
- Assessment of exposure to hazards at depth:
  - *Physical:* noise, radiation, cold, differential pressures, tools, electric fields
  - *Biological:* from leptospirosis to hippopotami
  - *Chemical:*
    - equipment, off-gassing, etc (e.g., caprolactam, epichlorhydrin)
    - chamber atmosphere (solvents, ultrafine welding particles, contaminant gases)
    - environmental sources (petrochemicals, degraded muds, lead, H_2S)
- Saturation compression rates and control of HPNS
- Atmosphere monitoring and control
- pO_2 limits for each phase of saturation and excursions
- Maximum duration of saturation dives, bell-runs and in-water excursions
- Weight loss during saturation dives
- Saturation chamber hygiene (particularly *Pseudomonas,* HIV)
- Physiological assessment of any new procedures and of breathing equipment

- In-water diver monitoring and need for continuous communication
- Hot-water suits, control of thermal balance in deep dives
- Monitoring and control of hyperbaric contaminants in a welding habitat at depth
- Special management requirements for accidents and illness in saturation
- Medical advice, to be given on-site or remotely, on the management of complex diving or major emergencies whether occurring on the surface, in-water and/or in saturation (e.g., a lost bell on the seabed, hyperbaric evacuation by HRV, fire in control room)
- Recovery of an unconscious diver into the bell, resuscitation while upright
- In-chamber medical intervention (e.g., traumatic amputation, crushed chest management)
- Maintaining the skills of trained diver-medics

A more complete syllabus of training objectives, including revision of Level I and Level II-basic, can be completed by distance learning (e.g., at the University of Stellenbosch) plus less than a week of simulated cases and appropriate in-water training on location. The International Marine Contractors Association (IMCA) recognises the relevance of such training for doctors retained by its world-wide member companies. Not many Level IIa doctors may be needed in a region but the working divers and their employers within it will depend on their competence.

David Elliott, Past President, UMS; Past President, EUBS; Past Chairman, DMAC; Past Chairman, EDTC

Key words
Occupational diving, occupational health, health surveillance, training, editorials



Peter Harrington, first published in *SPUMS Journal*. 1985;15(1).

The Editor's offering

Little has been published on the health and safety of chamber attendants in the therapeutic hyperbaric field compared with that of caisson workers and commercial divers. Exposure pressures tend to be low (less than 304 kPa) and exposure times short (less than two hours) in the vast majority of 'dives', although this is not true for treatments dealing with severe decompression illness. Perhaps also, there is little commercial imperative, and nurses come cheaply in the salary pecking ranks.

Therefore, the two papers by Cooper and colleagues from Hobart, Tasmania, are important contributions to our occupational health knowledge in these hyperbaric workers. In the first, decompression stress is meticulously assessed using Doppler in 163 exposures to what is probably the single most commonly used therapeutic regimen (with minor variations, as they point out) internationally – the 243 kPa 90-120min table. They report that 90% of these pressurizations were associated with low bubble counts – a surrogate for a low level of risk for decompression sickness. This, and a zero rate of decompression sickness in attendants in more than 6,000 pressurisations over a 14-year period, are very reassuring for the therapeutic hyperbaric community. In the second, epidemiological report, documenting the medical records and medical problems of 155 personnel over the 14 years, only 0.41% of exposures were associated with pressure-related medical problems. Almost all of these were trivial in nature, and largely avoidable with an even greater focus on safety than already employed in this safety-conscious hyperbaric unit.

Extreme freediving is one of those sports about which one shakes one's head and wonders why people are so crazy. Nevertheless, the rapid developments and increasing popularity of competitive freediving challenge us to understand better the pathophysiology of extreme breath-hold. There are several laboratories currently focusing interest on this area of environmental physiology, of which Professor Schagatay's unit in Sweden is one. Here, in the first of two reviews, she discusses the physiology of extreme-duration static apnoea and courageously predicts the future. In a forthcoming issue, she will discuss 'dynamic' apnoea.

Michael Davis

The front-page photo was taken by Annelie Pompe, one of the deepest diving females in the world, whilst freediving. A freediver is followed to the surface by a safety diver at the Blue Hole, Dahab, Egypt.

Original articles

Hyperbaric chamber attendant safety I: Doppler analysis of decompression stress in multiplace chamber attendants

P David Cooper, Corry Van den Broek, David R Smart, Ron Y Nishi and David Eastman

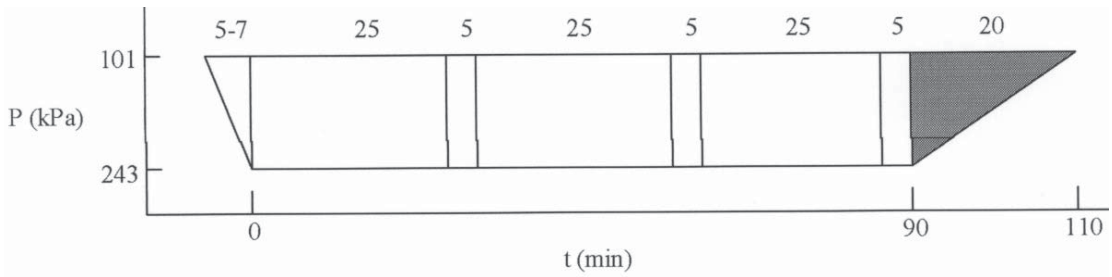
Key words
Decompression, decompression sickness, bubbles, Doppler, nursing, hyperbaric facilities, occupational health

Abstract
(Cooper PD, Van den Broek C, Smart DR, Nishi RY, Eastman D. Hyperbaric chamber attendant safety I: Doppler analysis of decompression stress in multiplace chamber attendants. *Diving and Hyperbaric Medicine.* 2009;39(2):63-70.)
Introduction: Incidences of decompression sickness of 0.76% have been reported in hyperbaric attendants exposed to routine 243 kPa treatment tables. Occupational health risks of this magnitude are not acceptable in routine clinical practice. Significant variations in procedures are therefore found between institutions in an attempt to enhance staff safety. In extreme cases, the use of multiplace chambers has been abandoned. Doppler ultrasound provides an objective tool to assess the sub-clinical decompression stress associated with any particular exposure.
Aims: To assess the decompression stress imposed upon staff exposed to our routine 243 kPa table and to elucidate demographic details within the attendant population that impact upon that stress.
Methods: Design: prospective observational cohort study. Profile: 243 kPa for 90 min with a 20 min decompression on oxygen. Subjects: 28 nursing and medical personnel routinely undertaking patient care under hyperbaric conditions. Procedure: Doppler assessment at 20 min intervals for up to 120 min post-exposure. Scoring: aural grading of intravascular bubbles using the Kisman-Masurel (K-M) scoring system; 163 exposures were scrutinized in this manner.
Results: 68% of exposures resulted in 'low' (K-M Grades 0-I), 22% in 'intermediate' (Grade II) and 10% in 'high' sub-clinical decompression stress (Grades III-IV). Female gender and increasing age, weight and exposure frequency showed trends towards higher bubble grades. There were no cases of clinical decompression sickness.
Conclusions: Our standard 243 kPa table conforms to DCIEM definitions of 'acceptable' decompression stress (Grade II or fewer bubbles in ≥50% of the subjects). Significant inter- and intra-individual variability was evident even within this one, tightly controlled dive profile.

Introduction

Hyperbaric oxygen therapy, when provided in a multiplace chamber, involves the exposure of at least one attendant, and a variable number of patients, to increased atmospheric pressure. During treatment the multiplace chamber is pressurized with air and patients breathe 100% oxygen through tightly fitting oronasal 'aviator-style' masks or free-flow oxygen hoods. At the Royal Hobart Hospital (RHH), attendants breathe air throughout the time at pressure then, from the start of decompression, breathe 100% oxygen. The 243 kPa (14 metres' sea water depth, 2.4 ATA) treatment table currently in use was first implemented in January 1997 at RHH and accounts for the majority of attendant exposures to pressure at our facility (Figure 1). It provides strict control of depth and bottom time, with decompression time based on triple the DCIEM air-diving schedule requirement, to minimise the risk of clinical decompression sickness (DCS) in our attendants.[1] Rates of DCS up to 0.76% have been reported in hyperbaric attendants exposed to routine 243 kPa treatment tables.[2,3] In today's occupational health and safety climate it is not acceptable to expose staff members, potentially thousands of times in a career, to health risks of this magnitude.

Figure 1. Royal Hobart Hospital 243 kPa treatment table (RHH 14:90:20)
White = attendant on air. Grey = attendant on oxygen. Patient air-breaks marked to indicate periods of increased attendant activity in-chamber



Important demographic differences may exist between the hyperbaric attendant population (predominantly female, 30–50 years of age, hospital-based nurses, variable fitness, seated for much of the treatment) and the population against whom the safety of commonly used decompression tables is typically validated: predominantly male, 20–40 years of age, fitness-conscious military or emergency services personnel, either exercising during 'wet-chamber' dives or recumbent during 'dry-chamber' dives, and all self-selected volunteers.[4] Accurate assessment of the occupational health risk posed to in-chamber attendants working at hospital-based hyperbaric facilities is, therefore, difficult.

Any exposure to compressed air carries with it the risk of tissue and intra-vascular nitrogen bubble formation on decompression. Theoretically, when these bubbles exceed certain thresholds (e.g., number or bubble radius) the probability of clinical DCS rises sharply.[5–8] Doppler ultrasound is a technique that has been extensively used and refined by organizations such as Defence R&D Canada – Toronto (DRDC Toronto, formerly the Defence and Civil Institute of Environmental Medicine, DCIEM) and Duke University, USA, to assess the decompression stress of new dive profiles and validate the safety of existing empirical or theoretically-derived decompression tables.[5–13] Gas bubbles in the circulatory system occur more frequently than does symptomatic DCS, can be detected even in known 'safe' dive profiles and are able to be graded by readily available Doppler technology.[5–8] The detection of bubbles in this way therefore provides more detailed information about the decompression stress associated with a given dive profile than does the binary outcome of 'DCS versus no DCS'.[8,14]

Various publications on DCS-related staff health risks have kept the issue of "How safe is 'safe'?" topical in the minds of personnel and institutions alike.[15–17] In January 2001 it was therefore decided to attempt to quantify the decompression-related risks associated with the RHH standard 243 kPa table; despite 1,570 attendant exposures up to that time without a case of clinical DCS.

Aims

Primary endpoint: To assess, using Doppler ultrasound, the extent and significance of decompression stress experienced by attendants routinely exposed to the standard RHH 243 kPa treatment table; and compare these results with established DCIEM tolerances for decompression stress.
Secondary endpoint: To evaluate the demographic variation within the hyperbaric attendant population and its relationship to decompression stress.

Methods

STUDY DESIGN

A prospective, observational, cohort study was conducted using Doppler ultrasound to assess sub-clinical decompression stress over 44 months (April 2001 to November 2004). This project was approved by the Research Ethics Committee of the RHH Research Foundation and the Human Research Ethics Committee at DRDC Toronto, Canada.

SUBJECTS

The Royal Hobart Hospital is the major university teaching hospital and tertiary referral centre for the State of Tasmania, Australia. The RHH hyperbaric unit accepts approximately 120 new referrals and performs some 2,000 patient treatments annually. All RHH nursing and medical personnel working in the hyperbaric environment during the study period were invited to participate. Personnel undergoing hyperbaric exposure were all medically certified fit-to-dive in accordance with the appropriate Australian Standard in force at the time. Personnel under the age of 40 years are re-certified biennially, and those 40 years and older annually.

There were no exclusion criteria as this was an observational study undertaken on personnel in the performance of their normal duties. Participation was voluntary and informed consent was gained from all participants. Baseline demographic data were collected for all eligible personnel, irrespective of whether they participated in the Doppler study. These data included age, sex, height, weight and calculated body mass index (BMI), and the frequency with which they underwent hyperbaric exposure.

HYPERBARIC PROCEDURES

All exposures took place in the RHH multiplace chamber (Hydro Electric Commission, Hobart, Tasmania, 1993). The established RHH 243 kPa table was adhered to throughout the study. The attendant was active around the chamber at the beginning of the isobaric phase and for three five-minute periods during the course of the dive whilst the patients received their air-breaks (assisting the patients donning and removing their face masks or oxygen hoods). For the remainder of the time the attendant was seated and relatively inactive unless a problem arose with a patient. The compression phase was generally 5–7 minutes, but could extend to a maximum of 12 minutes if a patient encountered difficulties (usually with middle-ear equalization). In the unlikely event of bottom time exceeding 110 minutes – but being less than 120 minutes – a five-minute decompression stop was mandated at 9 metres, otherwise a linear decompression over 20 minutes was performed.

Personnel were routinely restricted to a maximum of four hyperbaric exposures per week, with no more than three days of consecutive pressure exposure. Additionally, because of Hobart's mountainous terrain (highest habitation 550 m, routine attendant travel to >600 m, sealed roads to 1,250 m), attendants living ≥300 metres above sea level were required to remain at sea level for at least four hours before travelling home. In practice, these personnel were rostered for the morning treatment, allowing off-gassing in the afternoon. A

minimum 18-hour break was required between hyperbaric exposures to ensure attendants had returned to DCIEM repetitive factor 1.0 (i.e., no residual nitrogen load) prior to their next dive.[1]

DATA COLLECTION

Attendant Doppler sampling was undertaken according to the techniques described by Eatock and Nishi.[18] One Australian author (CVdB) travelled to Canada prior to the study to receive training in Doppler monitoring at DCIEM. All measurements were performed by this individual, or under his direct supervision. Recordings were undertaken using a 2.5MHz continuous-wave Doppler ultrasound device (TSI DBM 9008, Techno Scientific Inc., Ontario, Canada) with a precordial Doppler array probe (TSI-DPA7). Doppler recordings were taken over the precordium and each subclavian vein at 20-minute intervals for up to two hours from the start of decompression (or until any bubbles detected had peaked and clearly started to decline) and recorded onto magnetic audio cassettes. The first recording was performed immediately after the attendant exited the chamber. Each 20-minute recording included the following:

- precordium, at rest – 60 seconds
- precordium, three squats – 30 sec after each
- subclavian veins, at rest – 30 sec
- subclavian veins, three hand clenches – 15 sec after each.

Subclavian measurements were performed bilaterally. A standard questionnaire was completed pre- and post-exposure on days of Doppler scanning. Personnel were also required to report any symptoms arising within 24 hours post-exposure.

DATA ANALYSIS

Doppler recordings were graded aurally using the methodology described by Kisman and Masurel (K-M code).[8,19] This consists of a three-part assessment that analyzes (i) frequency, (ii) either percentage [at rest] or duration [following movement] and (iii) amplitude of detected bubbles, to yield a single bubble grade (0-IV). K-M Grades 0-I may be considered to indicate 'low', Grade II 'intermediate' and Grades III-IV 'high' sub-clinical decompression stress. It was decided in advance that our 243 kPa table would be deemed 'safe' if it complied with DCIEM-defined limits of acceptability (Grade II or fewer bubbles in 50% or more of the subjects), or in need of revision if it fell outside these limits.

Aural scoring is known to be observer-dependent; therefore all Doppler recordings were graded by the single author who had undergone DCIEM training. A random sample of 10% of recordings was scored independently at DCIEM and the results compared. No grading discrepancies between observers occurred in this sample.

Bubble grades were treated as categorical data for statistical analysis. The highest K-M bubble grade following each hyperbaric exposure was tabulated for statistical comparison. Analysis was completed using GraphPad Prism® version 4.03 for Windows (GraphPad Software, San Diego, California, USA, 2005). Given the relatively small numbers in this study, bubble grades were dichotomized into 'acceptable' (Grades 0-II) versus 'unacceptable' (Grades III-IV) and demographic variables similarly dichotomized to facilitate subsequent statistical analysis. The thresholds for division of each demographic variable were as follows: Age $<$ or $\geq$ 40 years (age when institutional policy mandates change from biennial to annual medical examination), BMI $\leq$ or $>$ 25.5 (underweight/normal versus overweight/obese), and sex (male versus female). The resulting 2 x 2 contingency tables were subjected to Fisher's exact test. All tests were two-tailed and $P < 0.05$ was considered statistically significant.

Results

Fifty personnel underwent 1,887 attendant exposures to our 14:90:20 profile between April 2001 and November 2004. Of these, 28 (56%) participated in the Doppler research. These 28 individuals contributed the vast majority of personnel exposures to pressure, performing 1,743 (92%) of the dives. Of these 1,743 exposures, 163 were subject to Doppler analysis (9.4%). Baseline demographic data revealed that the study participants and non-participants were comparable in all respects other than frequency of hyperbaric exposure (Table 1). The reasons for non-participation were invariably logistic (personnel with other commitments following completion of exposure).

Two sub-groups of participants were compared based upon work patterns: regular (multiple exposures per week) versus casual personnel (less than two exposures per week). No significant demographic differences were found between these groups, except for hyperbaric exposure frequency

Table 1
Demographic data (participants versus non-participants)

Variable	Participants (n = 28)	Non-Participants (n = 22)	*P*-value
Age (yrs)			
Mean (SD)	37.2 (7.7)	35.6 (5.3)	0.43
Sex; n (%)			
Male	9 (32)	7 (32)	1.00
Female	19 (68)	15 (68)	
BMI ($kg.m^{-2}$)			
Mean (SD)	25.0 (2.8)	24.3 (4.3)	0.51
No. dives in study period			
Range	1–416	1–22	
Mean (SD)	62 (103)	6 (5)	0.015
Total (%)	1743 (92.4)	144 (7.6)	

Table 2
Demographic data of participants (casual versus regular attendants)

Variable	Casual (n = 23)	Regular (n = 5)	*P*-value
Age (yrs)			
Mean (SD)	36.8 (7.2)	39.0 (7.7)	0.57
Sex; n (%)			
Male	8 (34.8)	1 (20.0)	1.00
Female	15 (65.2)	4 (80.0)	
BMI (kg.m^{-2})			
Mean (SD)	25.2 (1.5)	23.9 (2.2)	0.50
No. dives during study period			
Range	1–98	37–416	
Dopplered	*1–8*	*15–26*	
Mean (SD)	28 (27)	219 (172)	<0.0001
Dopplered	*2 (1)*	*21 (5)*	*<0.0001*
Total (%)	648 (37.2)	1095 (62.8)	
Dopplered; n (%)	*57 (35.0)*	*106 (65.0)*	

Table 3
Individual attendants' bubble grades; BMI - body mass index

	Age yrs	Sex	BMI kg.m^{-2}	Monitored **dives** (n)	Bubble range Median	Mode	Range
Regular attendants							
1	42–46	F	22.5	26	II	II	O–III
2	32–35	F	23.8	25	I	I	O–II
18	25–27	F	20.5	17	O	O	O–I
21	41	M	26.5	15	O	O	O–III
23	47–50	F	26.4	23	II	II	I–III
Casual attendants							
3	28–30	M	22.5	2	O/I	O/I	O–I
4	34–35	M	26.1	4	I	I	I–III
5	41–42	M	27.5	3	I	I	O–I
6	44	F	23.6	4	II	II	I–III
7	25	F	20.2	1	O	O	O
8	28	F	26.2	1	II	II	II
9	44–46	F	25.2	8	I	O	O–III
10	23	F	22.8	2	O	O	O
11	31	M	21.1	1	O	O	O
12	36	F	33.7	1	O	O	O
13	35	F	22.8	2	O	O	O
14	31–32	F	25.1	5	O	O	O
15	37–38	F	23.3	2	O/I	O/I	O–I
16	31	M	19.0	2	O	O	O
17	35	F	32.3	3	O	O	O–I
19	30–31	M	20.7	2	O	O	O
20	52–53	F	24.2	3	I	I	O–I
22	37	F	25.6	2	O	O	O
24	52	F	28.7	1	II	II	II
25	42	M	31.0	1	II	II	II
26	33	M	30.2	3	I	I	O-I
27	38–39	F	28.4	2	O/I	O/I	O–I
28	41	F	20.3	2	O/IV	O/IV	O–IV

Table 4
Relationship of bubble grade to demographic variables

Variable	Grade O-II	Grade III-IV	*P*-value
Age (yrs)			
20–39	76	1	0.0004
40–54	71	15	
Sex			
Male	31	2	0.53
Female	116	14	
BMI (kg.m^{-2})			
≤25.5	93	9	0.59
>25.5	54	7	
Exposure			
Casual	53	4	0.58
Regular	94	12	

Table 5
Times to onset and peak bubble grades

	Onset (precordial at rest)	**Onset** (all sites/states)	*P*-value
Number (%)	50 (31)	98 (60)	
Mean (SD)	41 (17)	29 (13)	<0.0001
Range (min)	18–80	18–95	
	Peak (precordial at rest)	**Peak** (all sites/states)	*P*-Value
Number (%)	50 (31)	98 (60)	
Mean (SD)	54 (17)	51 (18)	0.21
Range	18–74	20–95	

(Table 2). Individual attendants' bubble grades are presented in Table 3. The relationships between bubble grades and demographic variables are shown in Table 4. No cases of clinical DCS were identified following any of the 1,887 attendant exposures to this profile during the study period.

Bubbles were first detectable in the circulation an average of 29 minutes post-decompression and peak grades were achieved at around the 50-minute mark. There was a significant delay in onset time of detectable bubbles if only the precordial readings taken at rest were considered. The times to onset (non-zero) and peak bubble grades encountered in our cohort are shown in Table 5.

K-M bubble grades of II or less were encountered in 147 (90%) of the exposures studied when data from all sites/states (i.e., subclavian or precordial, at rest or following movement) were included, with 68% of exposures resulting in 'low', 22% in 'intermediate' and 10% in 'high' subclinical decompression stress (Figure 2). These figures changed to 94%, 5% and 1% respectively when only the precordial readings taken at rest were considered – with 161 dives (99%) now having a K-M bubble grade of II or less (Figure 3). These results were within the DCIEM-defined limits of acceptability.

Figure 2
Distribution of maximum bubble grades (all sites and states)

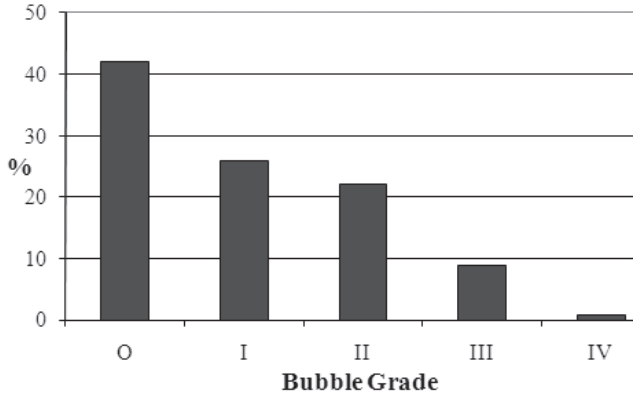
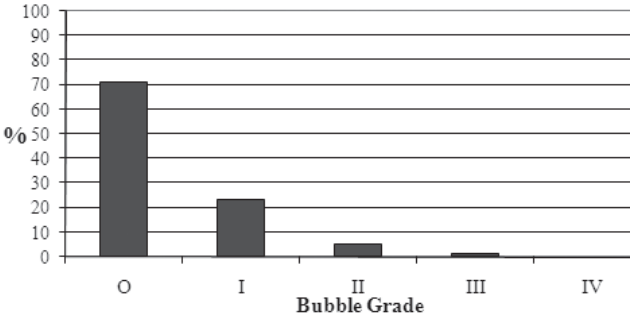


Figure 3
Distribution of bubble grades (precordial at rest)



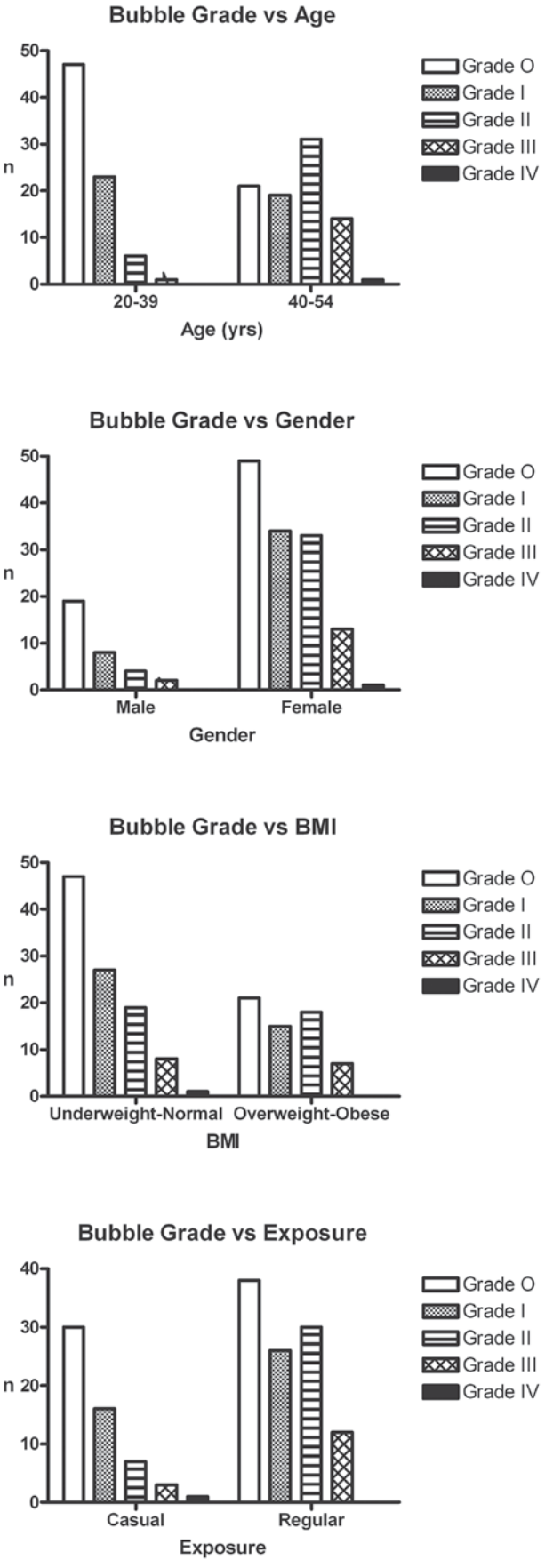
Increasing age, BMI, female gender and frequency of hyperbaric exposure were all associated with a trend towards higher bubble grades (Figure 4). However, only the relationship between bubble grade and age reached statistical significance in this cohort (Table 4).

Similarly, a considerable degree of intra-individual variability became evident as sample numbers on individual attendants increased. Higher bubble grades than usual for a given individual were encountered following injury, illness or exertion; unfortunately the small numbers involved precluded meaningful statistical analysis. Three individual cases may illustrate this point.

CASE A

Attendant 21 (41-year-old male – studied on 15 occasions) scored Grade 0 bubbles on the first 11 occasions. Following a gym-related groin strain he scored Grade II–III bubbles for more than three weeks post-event without further recognized injury (four further sets of Doppler recordings). His injury remained symptomatic throughout this time. This individual stopped working as an attendant because of this unusual and prolonged elevation in bubble grades and subsequently left the service. Follow-up data are not available.

Figure 4
Relationship of bubble grade to demographic variables



CASE B
Attendant 1 (female aged 42–46 years – studied on 26 occasions) sustained multiple musculoskeletal injuries playing netball during the course of the study – including a fractured finger, bilateral ankle sprains, hamstring injuries and numerous contusions. Elevated bubble grades were apparent when pre- and post-injury results were compared. These injuries occurred with such frequency that her true 'baseline' (totally uninjured) level of bubbling was difficult to establish. The majority of bubbles post-injury were detected coming from the affected limb (precordial for lower limb, precordial plus subclavian on affected side for upper limb injuries) with a smaller but more generalized elevation in bubble grades apparent in readings from uninjured limbs.

CASE C
The only Grade IV bubbles in this study occurred on the first occasion that Doppler was performed on Attendant 28 (41-year-old female – studied twice). Review of her pre-exposure questionnaire revealed chronic neck/back pain, a 'slight cold' (but able to equalize middle-ear pressures easily) and performance of 40 minutes of moderately strenuous gym exercise prior to the hyperbaric exposure. Follow-up Doppler 18 hours later gave Grade 0 bubbles. After her next hyperbaric exposure six days later, she had a Grade 0 bubble score. Within the next week she discovered that she was pregnant and ceased work at the chamber. The chronic neck/back ache and upper respiratory tract symptoms remained unchanged throughout.

Smaller fluctuations around an individual's mode were also seen, often not obviously associated with any specific identifiable event but appearing to reflect a 'normal' day-to-day variation.

Discussion

The maintenance of a safe working environment in hyperbaric medicine is of paramount importance to employers and employees worldwide. Most attention has been paid to the incidence of decompression sickness (DCS), and a ten-fold variation in incidence rates (0.076%–0.76%) has been reported from various hyperbaric units.[2,20] No episodes of clinical DCS occurred in over 4,000 exposures to our 243 kPa treatment table during the first fourteen years of chamber operations (January 1992 to December 2005), (95% CI 0.00, 0.09% incidence of DCS).[21] Nearly 10% of the hyperbaric exposures during the study period were subjected to Doppler analysis and demonstrated a bubble grade distribution within DCIEM 'safe' decompression recommendations (Grade II or fewer bubbles in 50% or more of the subjects).

Despite the increasing complexity of techniques used to model dive profiles, to date no theoretical model has been able to offer more than an approximation to the profound physiological subtleties encountered in real life. Hence, the need to develop experimental and investigational techniques to complement the modelling processes has long been recognized.[4,5,9,10] No 'gold standard' test is yet available, however, that can be said unequivocally to measure decompression stress throughout the body.

Of the techniques developed so far, Doppler detection of intra-vascular bubbles has arguably the greatest utility and most extensive evidence base.[4–9,22] The technology is relatively inexpensive, portable, robust and readily available and the skills necessary for standardized data acquisition are easily learned. Bubble detection provides significantly more information about the relative severity of a given exposure than does the simple incidence of clinical DCS. Despite these advantages, Doppler is not without its critics and certain limitations to the technique must be acknowledged.

- It is time consuming and labour intensive.
- It detects moving bubbles within the vascular tree only, which may not be representative of events in other tissues.
- Data analysis is dependent upon aural grading, requires more training to perform reproducibly than does simple data acquisition and is still potentially subject to inter-observer variability.
- The data collected are only semi-quantitative – with a non-linear correlation between grades assigned and bubble size or number.
- Bubble grades are ranked (non-parametric) data and the intervals between the ranks cannot be assumed to be uniform.
- The relationship between bubble grade and risk of DCS is non-linear and dependent on, amongst other things, gas mix breathed (e.g., Heliox versus air).[6]
- Intermittent data acquisition raises the possibility of missing the highest bubble grade.

The issues of where and when to obtain Doppler data post-exposure also remain open to debate. Some authorities contend that, since the final common pathway for venous bubbles is the right heart, precordial readings alone should be adequate. However, the difference noted between our all-sites readings and precordial readings alone of 60% versus 47% suggest a considerable reduction in sensitivity if this approach is adopted, possibly because of the increased complexity of identifying and classifying bubbles in the high-noise environment of the precordium.[5,7,8]

A further potential confounding variable also exists. The administration of oxygen during decompression may, by preferentially enhancing denitrogenation of the fast tissues, introduce a lead-time bias into the evolution of maximum bubble grades.[23] This delay in onset and time to peak may cause Doppler sampling to be ceased prematurely and with a false sense of security. Given the relatively short time to onset of Doppler-detectable bubbles encountered in this group (Table 5), and our policy of ensuring that sampling was continued for two hours or until any bubbles detected had peaked and clearly started to decline, we believe this risk to be minimized.

If these limitations are understood and accepted, then the Doppler detection of intra-vascular bubbles remains a useful tool in the assessment of sub-clinical degrees of decompression stress. To date, no other technology has demonstrated superiority over Doppler in the evaluation of decompression stress.

This was a single-centre study designed primarily to assess the safety of one, highly conservative, hyperbaric exposure profile. This end was achieved and a number of demographic variables were identified as predisposing attendants to increased sub-clinical decompression stress. Of these variables, in this series, older age appears to be the most important criterion to differentiate between individuals' decompression risks. Within a given attendant, injury, illness and peri-exposure exertion also appear to increase decompression stress. A generalized increase in bubble grade (i.e., not just arising from the affected limb) supports the presence of both systemic and local effects in the increased predisposition to bubble formation seen post-injury. The difference between Grade IV and Grade 0 bubbles in Attendant 28 appears due to her vigorous pre-dive physical work-out – although hormonal changes over this six-day period of very early pregnancy (i.e., surrounding blastocyst implantation) may have contributed.

The main limitation of this study was the percentage (56%) of eligible attendants studied. Despite the fact that these participants performed 92% of the dives on this table during the study period, a larger cohort undergoing Doppler monitoring would have enhanced the strength of the study. No attendant actually declined to participate in the study, but other duties frequently prevented casual personnel from remaining in the unit for the requisite two hours post-exposure. Likewise, other demands on technical personnel prevented more exposures being captured with Doppler.

Conclusions

This is the largest Doppler series of a single hyperbaric profile yet published and, we believe, demonstrates that maintenance of a safe workplace for in-chamber attendants does not pose an insurmountable problem. Our institutional policies and procedures appear to provide an acceptably safe working environment and will therefore remain unchanged. Differences between decompression strategies are likely to be the reason for our improved outcomes when compared with previously published series. More research and larger numbers will be needed to resolve issues such as optimal retirement age from in-chamber duties, appropriate stand-down times following injury and restrictions on pre- and post-dive exercise.

Acknowledgements

The authors thank the Tasmanian Salmonid Association and the Defence Reserves' Support Council for financial support to send Mr Van den Broek to DCIEM, Canada for training in vascular Doppler techniques. The participation of the personnel who were our subjects is greatly appreciated.

References

1 *DCIEM Diving Manual. Part 1: Air decompression procedures and tables.* DCIEM No. 86-R-35. Toronto, ON: Defence and Civil Institute of Environmental Medicine; 1992.

2 Brattebø G, Aanderud L, Risberg J, Thorsen E, Forland M. Incidence of decompression illness among HBO nurses. J Slovenian Med Assoc. *23rd Annual Scientific Meeting of the European Underwater and Baromedical Society*, Programme and Abstracts. Bled: Slovenia; 1997.

3 Risberg J, Englund M, Aanderud L, Eftedal O, Flook V, et al. Venous gas embolism in chamber attendants after hyperbaric exposure. *Undersea Hyperb Med*. 2004;31(4):417-29.

4 Nishi RY, Kisman KE, Eatock BC, Buckingham IP, Masurel G. Assessment of decompression profiles and divers by Doppler ultrasonic monitoring. In: *Proceedings of the 7th Symposium on Underwater Physiology*. Bethesda, MD: Undersea Medical Society; 1981. p. 717-27. (Also: DCIEM Report No. 80-P-18; 1980).

5 Eatock BC, Nishi RY. Analysis of Doppler ultrasonic data for the evaluation of dive profiles. In: *Proceedings of the 9th International Symposium on Underwater and Hyperbaric Physiology*. Bethesda, MD: Undersea and Hyperbaric Medical Society; 1987. p.183-94.

6 Sawatzky KD. *The relationship between intravascular Doppler-detected gas bubbles and decompression sickness after bounce diving in humans*. MSc Thesis. Toronto: York University, Canada; 1991.

7 Nishi RY. Doppler evaluation of decompression tables. In: Lin YC, Shida KK, editors. *Man in the Sea. Volume 1*. San Pedro, CA: Best Publishing; 1990. p. 297-316.

8 Nishi RY, Brubakk AO, Eftedal OS. Bubble detection. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th ed. London: Saunders; 2003. p. 501-29.

9 Nishi RY. Design of decompression trials - DCIEM experience. In: Lang MA, Vann RD, editors. *Proceedings of the Repetitive Diving Workshop*, AAUSDSP-RDW-02-92. Costa Mesa, CA: American Academy of Underwater Sciences; 1992. p. 311-20.

10 Nishi RY. The DCIEM decompression tables and procedures for air diving. In: Nashimoto I, Lanphier EH, editors. *Proceedings of the 36th Undersea and Hyperbaric Medical Society Workshop, Decompression in Surface-Based Diving*. UHMS Publication No. 73. Bethesda, MD: Undersea Medical Society; 1987. p. 80-3.

11 Pollock NW. Use of ultrasound in decompression research. *Diving and Hyperbaric Medicine*. 2007;37(3):68-72.

12 Vann RD, Pollock NW, Freiberger JJ, Natoli MJ, Denoble PJ, et al. Influence of bottom time on preflight surface intervals before flying after diving. *Undersea Hyperb Med*. 2007;34(2):211-20.

13 Eatock BC. Correspondence between intravascular bubbles and symptoms of decompression sickness. *Undersea Biomed Res*. 1984;11:326-9.

14 Walker MB. Doppler bubble detection after hyperbaric exposure. *SPUMS Journal*. 1996;26(3):146-54.

15 Doolette DJ, Goble S, Pirone C. Health outcome of hyperbaric chamber inside attendants following compressed air exposure and oxygen decompression. *SPUMS Journal*. 2004;34(2):63-7.

16 Walker MB, Capps R, Pirone C, Ramsay R. Doppler detection of circulating bubbles in attendants, decompressed on oxygen, following routine hyperbaric treatments. *SPUMS Journal*. 1995;25(2):62-4.
17 Klossner J, Niinikoski J, Kaunisto M, Virolainen R. Risk of decompression sickness (DCS) among attending nursing personnel during HBO therapy in multiplace chamber: what measures should be taken to optimise the safety of personnel? In: *Proceedings of the 12th International Congress on Hyperbaric Medicine*. Milan: Italy; September 4-8, 1996. p. 335-8.
18 Eatock BC, Nishi RY. *Procedures for Doppler ultrasonic monitoring of divers for intravascular bubbles*. DCIEM No. 86-C-25. Toronto, ON: Defence and Civil Institute of Environmental Medicine; 1986.
19 Kisman KE, Masurel G, Guillerm R. Bubble evaluation code for Doppler ultrasonic decompression data. *Undersea Biomed Res*. 1978;5(Suppl):28.
20 Dietz SK, Myers RAM. Decompression illness in HBO inside tenders: a review of 23 years of exposures. *Undersea Hyperb Med*. 1995;22(Suppl):57.
21 Cooper PD, Van den Broek C, Smart DR. Hyperbaric chamber attendant safety II: 14-year health review of multiplace chamber attendants. *Diving and Hyperbaric Medicine*. 2009; 39(2):71-6.
22 Eftedal OS, Lydersen S, Brubakk AO. The relationship between venous gas bubbles and adverse effects of decompression after air dives. *Undersea Hyperb Med*. 2007;34(2):99-105.
23 Pollock NW, Natoli MJ, Gerth WA, Thalmann ED, Vann RD. Risk of decompression sickness during exposure to high cabin altitude after diving. *Aviat Space Environ Med*. 2003;74:1163-8.

Received: 03 December 2008
Accepted: 31 March 2009

Paul David Cooper, MB, ChB, FJFICM, FANZCA, DipDHM, was initially Senior Registrar in Hyperbaric & Diving Medicine, then subsequently Deputy Director of Anaesthesia and Staff Specialist in Intensive Care and Diving & Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Australia during the study period.
Corry Van den Broek, DipAppSc, CPO-CD (RANR), is the Facility Manager and Senior Hyperbaric Technical Officer, Department of Diving & Hyperbaric Medicine, Royal Hobart Hospital, Hobart.
David Smart, MBBS (Hons), MD, FACEM, FIFEM, FACTM, FAICD, DipDHM, is Medical Co-Director, Department of Diving & Hyperbaric Medicine, Royal Hobart Hospital, and Director of Emergency Medicine, Calvary Health Care, Hobart.
Ron Nishi, MASc, PEng, (retd), was Senior Scientist, Experimental Diving Unit, Defence R&D Canada – Toronto.
David Eastman, BSc, is a Diving Research Technician, Experimental Diving Unit, Defence R&D Canada – Toronto, Ontario, Canada.

Address for correspondence:
Dr David Cooper,
Department of Diving & Hyperbaric Medicine,
Royal Hobart Hospital,
GPO Box 1061L, Hobart,
Tasmania 7001, Australia
Phone: *+61-(0)3-6222-8193*
Fax: *+61-(0)3-6222-7268*
E-mail: *<david.cooper@dhhs.tas.gov.au>*

This paper is based on part of the thesis submitted by Dr Cooper for the SPUMS Diploma of Diving and Hyperbaric Medicine.

News item

Explosion in Florida monoplace chamber

On Friday 1 May 2009, at the Ocean Hyperbaric Neurologic Center in Lauderdale-by-the-Sea, Florida, a free-standing facility established by the late Dr Richard Neubauer, an explosion and fire occurred. Initial reports indicate a failure of one of the Vickers clam-shell monoplace chambers, leading to a flash fire. A 61-year-old woman and her four-year-old grandson in the chamber at the time were both critically injured including severe burns, from which the grandmother has subsequently died. No other injuries occurred from the blast, and there is no apparent damage to the exterior of the building.

The facility operated both Sechrist front-loading and Vickers clam-shell monoplace chambers. The cause of the explosion and origin of the ensuing fire are currently unknown, and it is too early to report any further details. This is the first fire or explosion-related injury reported anywhere in the United States in either a monoplace or multiplace therapeutic facility during several decades of hyperbaric use.

Hyperbaric chamber attendant safety II: 14-year health review of multiplace chamber attendants

P David Cooper, Corry Van den Broek, David R Smart

Key words
Decompression sickness, barotrauma, ear barotrauma, hyperbaric facilities, nursing, occupational health

Abstract
(Cooper PD, Van den Broek C, Smart DR. Hyperbaric chamber attendant safety II: 14-year staff health review of multiplace chamber attendants. *Diving and Hyperbaric Medicine*. 2009;39(2):71-6.)
Introduction: The multiplace hyperbaric chamber provides a unique working environment for health-care personnel. The major foci of concern regarding staff health under these conditions have tended to be decompression sickness (DCS) and barotrauma. Incidences of DCS as high as 1.3% have been reported in hyperbaric attendants exposed to routine treatment tables. Occupational health risks of this magnitude are not acceptable in routine clinical practice. Significant variations in procedures exist between institutions in an attempt to enhance staff safety. In extreme cases the use of multiplace chambers has been abandoned.
Aim: To determine the actual incidence of work-related health issues amongst attendants at a full-time clinical hyperbaric unit.
Methods: Design: retrospective staff health survey. Facility: university teaching hospital. Subjects: 155 medical, nursing and technical staff routinely exposed to hyperbaric conditions.
Results: There were no cases of DCS encountered in 6,062 attendant exposures, across all hyperbaric profiles, during this 14-year period (95% CI 0, 0.06%). Twenty-eight work-related injuries occurred during this time (0.46%), of which 25 (0.41%) were hyperbaric-specific.
Conclusion: A multiplace hyperbaric chamber can be viewed as a relatively safe working environment.

Introduction

The multiplace hyperbaric chamber provides a unique working environment for health-care personnel. The exposure of attendant staff to environmental conditions of increased ambient pressure and various inhaled gas mixtures during the performance of their duties is unmatched elsewhere in the health-care industry. Traditionally the most prominent focus of concern regarding staff health under these conditions has been decompression sickness (DCS), with barotrauma coming a distant second. Rates of DCS up to 0.76% have been reported in hyperbaric attendants exposed to routine 243 kPa (14 metres' of sea water (msw), 2.4 ATA) treatment tables, with higher rates reported for deeper tables.[1–3] This level of injury is not acceptable in today's workplace and significant variation in procedures, therefore, may be found between institutions in an attempt to enhance staff safety. As an example, within Australian and New Zealand hyperbaric units, bottom times for 243 kPa hyperbaric treatment tables, vary from 90 to 105 minutes and decompression times from 10 to 30 minutes; with both linear and staged decompression profiles being used (Figure 1).

The 243 kPa treatment table currently in use at the Royal Hobart Hospital (RHH) was first implemented in January 1997. Conservative decompression procedures were empirically chosen to ensure more than triple the recommended DCIEM Air Diving Tables' decompression time.[4] Oxygen breathing by the attendants was added for the duration of the decompression phase as an additional aid to nitrogen off-gassing. Doppler ultrasound evaluation of attendants exposed to this institution-specific table has demonstrated low levels of decompression stress.[5] With a zero incidence of clinical DCS and low levels of sub-clinical decompression stress evident from that study, we decided to review other health issues potentially attributable to working in the hyperbaric environment.

Aim

To determine the incidence and severity of work-related health issues amongst personnel exposed to increased pressure at a full-time clinical hyperbaric unit.

Methods

STUDY DESIGN

A comprehensive review of the on-site medical records of all hyperbaric attendants to have worked in this facility since the year prior to its commissioning (together, where appropriate, with individual interviews and cross-referencing against other sources of medical information) was performed. These records covered a 14-year period from January 1992 to December 2005. The chamber dive-log and computer database (in which the hyperbaric technical officers independently record any variances to standard pathways) were also searched for evidence of incidents that might not have resulted in an entry in the medical records. The project was approved by the relevant institutional ethics committee.

SUBJECTS

The records of all nursing, technical and medical staff working in the hyperbaric environment at RHH during the study period were reviewed. There were no exclusion criteria. Staff members undergoing hyperbaric exposure were all medically certified fit-to-dive in accordance with the appropriate Australian Standard in force at the time. Staff under the age of 40 years are re-certified biennially and those 40 years and over are re-certified annually. These obligatory medical examinations are performed in-house by hyperbaric physicians and the records retained on-site indefinitely. It is departmental policy for all general health-related issues to be reported and included in this record, together with any symptoms arising within 24 hours of hyperbaric exposure.

Baseline demographic data were collected for all eligible staff. These data included age, sex, height, weight and calculated body mass index (BMI), and the frequency with which they underwent hyperbaric exposure (Table 1).

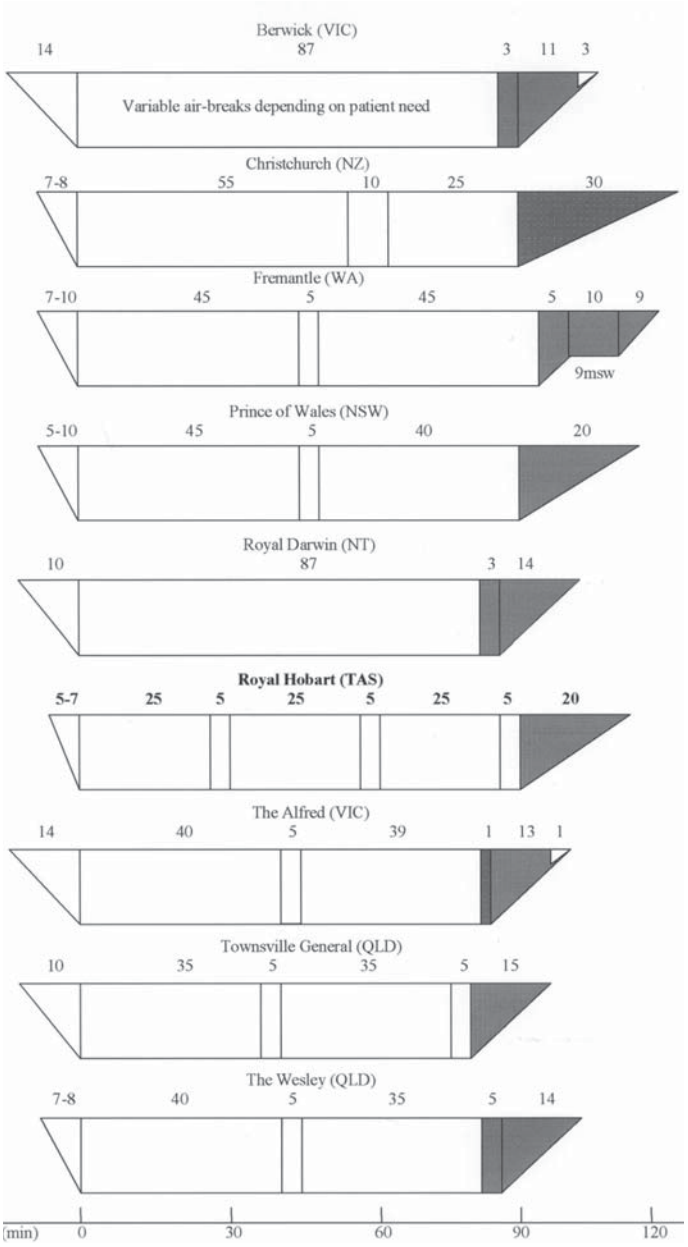
HYPERBARIC PROCEDURES

All exposures took place in the RHH multiplace chamber (Hydro Electric Commission, Hobart, Tasmania, 1993) – a 28-cubic metre, double-lock, cylindrical facility with a maximum operating pressure of 608 kPa. Established treatment tables (e.g., the 'RHH 14:90:20', Royal Navy treatment table 62 (RN 62) / US Navy treatment table 6 (USN 6), 'Comex 30', etc) accounted for the majority of hyperbaric exposures. Any non-standard exposure profiles (e.g., for training purposes) were conducted in accordance with DCIEM Air Diving Tables.[4] Oxygen (O_2) was used by attendants for the duration of all decompressions. If a patient required extensions on RN 62/USN 6, the attendant's O_2 breathing was extended to include the duration of the patient's final O_2 period at 182 kPa (i.e., the attendant received 90 minutes on O_2).

Personnel were routinely restricted to a maximum of four hyperbaric exposures per week, with no more than three consecutive days of pressure exposure. For most treatment tables a minimum 18-hour break was required between hyperbaric exposures to ensure attendants had returned to DCIEM repetitive factor 1.0 (i.e., no residual nitrogen load) prior to their next dive.[4] Following longer or deeper tables (e.g., RN 62/USN 6, Comex 30) this was extended to 48 hours.

Flying was forbidden for 24 hours after the attendant's last hyperbaric exposure. Additionally because of Hobart's mountainous terrain (highest habitation 550 m, routine attendant travel to >600 m, sealed roads to 1,250 m), attendants living >300 metres above sea level were required to remain at sea level for a minimum of four hours before travelling home. In practice, these staff were rostered for the morning treatment, allowing off-gassing in the afternoon.

Figure 1
Australasian 243 kPa (14 msw) chamber attendant tables – valid at November 2008
White = attendant on air. Grey = attendant on oxygen; patient air-breaks marked to indicate periods of increased chamber attendant activity in-chamber



Results

A total of 155 nursing, medical and technical staff underwent 6,062 hyperbaric exposures during 5,821 chamber pressurizations between January 1992 and December 2005. Medical records were available for 142 (92%) of these individuals. The chamber was under pressure for 10,895 hours during this time. Hyperbaric exposure profiles are summarized in Table 2.

There were no recorded cases of DCS amongst the chamber attendants over this 14-year period (95% CI 0.00, 0.06% incidence of DCS).

Review of the medical and technical records during this period revealed 28 potentially work-related health incidents (0.46%), of which 25 (0.41%) were hyperbaric-specific (Table 3). The most common complaints were of a minor ENT nature, with fifteen (1 in 400 compressions) minor to moderate middle-ear barotrauma episodes involving eighteen ears (nine Teed-Edmonds Grade O, five Grade I and four Grade II), one external ear barotrauma related to a plug of cerumen and three episodes of sinus squeeze on descent. One episode of sinus squeeze and eleven of middle-ear barotrauma were associated with recent upper respiratory tract infections (URTI). Five of these episodes of middle-ear barotrauma resulted in the attendant aborting the dive during pressurization (at 110–200 kPa).

Three episodes of odontalgia and one of obvious dental barotrauma (all associated with old dental work) occurred. Restorative dental work was paid for by the hyperbaric unit in the case of obvious dental barotrauma, with the attendant self-funding extensive simultaneous dental work on neighbouring carious teeth. One individual with recurrent gastrointestinal bloating following routine hyperbaric exposures (sometimes associated with vomiting or explosive diarrhoea after exiting the chamber) retired from in-chamber service when it became apparent that her tendency to air-swallow could not be overcome.

One attendant failed to divulge in her pre-employment medical examination that she had previously been diagnosed with 'benign fasciculation syndrome' after seeking investigation for multiple sclerosis from a neurologist. She reported symptom exacerbation (fatigue and increased fasciculations in her back and arms) lasting up to 24 hours post-dive; symptoms becoming continuous if she dived twice or more per week. A clear temporal relationship was established between symptom deterioration and hyperbaric exposure and, when her background medical condition was clarified, she was stood down from hyperbaric work and declared unfit for recreational diving.

Several workplace medical problems unrelated to hyperbaric exposure occurred.

- A trainee nurse attendant who put her foot through an open access panel in the floor during mopping-up operations following an in-chamber fire drill sustained a soft-tissue neck injury and a spiral fracture of the right fibula.
- A technician whose hand slipped whilst working on a valve/pipeline stabbed himself in the left first web-space with a screwdriver (no neurovascular damage).
- A nurse developed right neck/trapezius pain and tenderness after attending to a patient's dressing in an awkward position (outside chamber, no dives for >48 hours prior to injury).

Table 1
Demographic data for 155 chamber attendants exposed to pressure January 1992 to December 2005

Variable	Female	Male	Combined
Number (%)	108 (70)	47 (30)	155 (100)
Age, yrs Mean (SD)	36.0 (8.5)	35.8 (7.1)	35.9 (8.1)
BMI, $kg.m^{-2}$ Mean (SD)	23.5 (3.1)	26.0 (3.5)	24.4 (3.3)
Exposures during study period			
Range	1–601	1–107	1–601
Mean (SD)	64 (117)	19 (38)	47 (95)
Total	5,153	909	6,062
%	85	15	100

Table 2
Hyperbaric chamber runs 1992 to 2005
msw – metres' sea water depth
† pressure (msw):time at pressure (min): decompression time (min)
‡ binomial 95% confidence intervals for actual zero incidence of DCS

Treatment tables	Number	DCS 95% CI‡
Comex 30	1	
30 msw (misc.)	4	
RN 62 / USN 6	169	0.00, 2.16
RN 61 / USN 5	8	0.00, 36.94
'18:60:30'†	561	0.00, 0.66
18 msw (misc.)	39	
>14<18 msw (misc.)	1	
'14:90:20'†	4,079	0.00, 0.09
'14:60:15'† (obsolete)	815	0.00, 0.45
>10<14 msw (misc.)	81	
10 msw (misc.)	45	
<10 msw (misc.)	18	
Total	5,821	0.00, 0.06

One individual passed his initial pre-employment dive medical but encountered difficulties with recurrent sinus pain during training, with three training dives being aborted. No symptoms or signs other than pain (fully reversed on return to surface) were encountered. He discontinued training as a hyperbaric attendant.

Three instances of pre-existing acute illness (not work-related) were recorded as impacting on chamber operations. One attendant reported for work following a febrile illness the previous night (not disclosed prior to pressurization) and had to be replaced during a patient treatment when she spiked a fever at depth. Another attendant was replaced during a treatment when discomfort from an ocular foreign body acquired on the way to work became intolerable. One episode of non-hyperbaric-related middle-ear barotrauma

Table 3
Work-related hyperbaric personnel injuries from January 1992 to December 2005
URTI – upper respiratory tract infection

Condition	Number	Associated with URTI
Decompression sickness	0	
Gas toxicity incidents	0	
Barotrauma		
Inner ear	0	
Middle ear (15 episodes, 3 bilateral)		
Grade O	9	3
Grade I	5	5
Grade II	4	3
Grade III	0	
Grade IV/V	0	
External ear	1	
Sinus	3	1
Dental/Odontalgia	4	
Gastrointestinal	1	
Worsening of pre-existing neurological condition	1	
Unrelated to hyperbaric exposure	3	
Total	**31 (28 episodes)**	

was noted in a nurse who reported for work with a URTI and remained outside the chamber but was subsequently unable to return home to >500 metres above sea level (hospital at sea level) at the end of the day. Review following her attempt to get home demonstrated bilateral Grade III middle-ear barotrauma. She was obliged to stay with friends at sea level for several days before being physically able to return to her own home.

Four individuals failed their pre-employment dive medicals; one for severe hypertension, two for profound unilateral sensorineural deafness and one for mild bilateral sensorineural deafness combined with difficulty equalizing middle-ear pressures during the attendant training course.

In summary, during a 14-year period, 18,124 patient treatments were performed, requiring 6,062 attendant exposures in a multiplace chamber. No cases of DCS occurred (95% CI 0.00, 0.06%). The incidence of work-related staff health problems was 28 per 6,062 exposures (0.46%), with 25 per 6,062 (0.41%) being pressure-related, the vast majority of which were of a minor or trivial nature. No hyperbaric-specific injuries resulted in formal injury compensation claims, though restorative dental work was paid for in one case of obvious dental barotrauma.

Discussion

Workplace injuries pose a major concern to the healthcare industry. Registered nurses in Australia are reported to have an incidence rate of compensated injuries of 14.34 per million hours worked.[6] Given that it is widely acknowledged that over half of the injuries or illnesses sustained by this group at work are not reported, and only a small proportion of those reported result in compensation, the true incidence of workplace injury is likely to be considerably higher.[6–8] It is against this backdrop that the risks of employment as a multiplace hyperbaric chamber attendant must be measured.

Registered nurses constitute the majority of in-chamber attendant exposures to hyperbaric conditions (98.4% in this series). In its general (i.e., non-hyperbaric) duties this professional group is second only to truck drivers and manual labourers in its incidence of musculoskeletal injuries resulting in lost work days.[9] These injuries primarily involve the back and are generally related to the manual handling of patients. Low back problems are reported to have a point prevalence of 17%, an annual incidence of 40–50% and a lifetime incidence of 35–80% amongst nurses.[10] In our study, this type of injury was not encountered as a result of work within the hyperbaric environment. The traditional concern regarding DCS risk within the hyperbaric community means that personnel maintain a high index of suspicion for musculoskeletal symptoms and are obliged to report any such symptoms arising during their employment with us.

DCS, although potentially serious if it occurs, is an injury the incidence of which can be reduced to acceptable (near-zero) levels by the adoption of suitably conservative decompression strategies and the addition of O_2 breathing. Published data for USN 6 quotes a 6.2% probability of DCS in attendants, rising to 11.1% if the table is extended at 284 kPa, if the attendant breathes air throughout.[11] These rates are halved if the attendant breathes oxygen for the 30-minute decompression to the surface, and diminished to nearly zero if the attendant's O_2-breathing time is extended to coincide with the patient's final 60-minute oxygen period at 182 kPa.

A ten-fold variation in the incidence of DCS in chamber attendants has been reported to date. Dunford reported a 0.31% incidence in 8,424 hyperbaric exposures over 14 years and Dietz a 0.076% incidence in 25,164 exposures over 23 years.[12,13] Both of these authors report a correlation between increasing pressure and DCS incidence. Klossner described a 1.3% DCS incidence over 232 exposures on a 284 kPa table derived from Finnish amateur diving tables.[2] The risk of DCS was reduced to 0.14% over the next 713 exposures by a combination of reducing the treatment pressure to 253 kPa, extending decompression times and adding oxygen breathing by the attendant both during decompression and for 10 minutes at the start of the table.

The highest incidence of DCS reported on a 240 kPa table (100-minute isobaric phase, seven-minute decompression) is 0.76%. Those authors attempted to reduce this incidence

by modification of their attendants' breathing gases.[1,3] Unfortunately they were unable to establish a procedure that met institutional acceptance criteria.[3] Using DCIEM tables, their profile would have required a five-minute decompression stop at 3 msw gauge pressure and their routine seven-minute linear decompression from 240 kPa may not have been adequate to compensate for this. The University Hospital to which they were affiliated subsequently discontinued elective hyperbaric treatments in the multiplace chamber to eliminate the inherent risk to their staff. This contrasts strongly with the zero incidence of clinical DCS in our study. Had these reported DCS incidence rates held true for our table, 46–60 cases of clinical DCS would have occurred during our first 14 years of operation and the three most prolific regular attendants (with between 369 and 601 dives each) could have expected to be 'bent' three to six times each. This was fortunately not the case.

With no DCS amongst our chamber attendants, our focus turned to other staff health concerns. Minor ENT barotrauma was the most common hyperbaric-specific injury evident in this study. If injuries unrelated to pressurization (e.g., incurred during patient dressings, equipment maintenance and cleaning) are discounted, what proportion of the remaining hyperbaric-specific injuries was potentially preventable? Grade 0 middle-ear barotrauma (symptoms but no signs) may be legitimately thought of as a warning of impending harm rather than an injury per se. If Grade 0 middle-ear barotrauma, together with the situation where an individual deliberately withheld pertinent medical information at the time of employment (a pre-existing neurological condition), were excluded, we were left with 18 barotraumatic injuries (1 per 337 exposures) – half of which occurred in conjunction with a recent URTI. Existing unit guidelines require attendants not to dive following URTI until free movement of their tympanic membranes is verified by the duty doctor. More rigorous enforcement of these guidelines may therefore reduce this problem. Likewise the incidence of odontalgia or dental barotrauma may be amenable to modification by insisting on regular dental review for all personnel.

This leaves a 0.08% incidence (5 per 6,062) of potentially non-preventable hyperbaric-specific events: one episode of unilateral Grade II middle-ear barotrauma (in an experienced attendant); two episodes of sinus pain, not associated with obvious recent URTI; one episode of external ear barotrauma associated with unsuspected cerumen plugging; and one idiosyncratic case of gastrointestinal bloating due to air swallowing, not amenable to remediation. A multiplace hyperbaric chamber can, therefore, be viewed as a potentially safe working environment, especially when compared to the known incidence of back injury associated with general ward nursing duties.

Although the respective merits and disadvantages of multiplace versus monoplace facilities have caused considerable debate – with strong proponents for both views – it is our opinion that there will always be some patients for whom the immediate, hands-on attendance of a trained nurse (+/- a physician) will be mandatory. If these patients are not to be disadvantaged by either being denied access to HBOT or subject to it under conditions which separate them from immediate direct contact with the staff caring for them, then multiplace chambers (and the associated exposure of attendants to pressure) will remain a necessary part of hyperbaric medicine. Likewise, if multiplace chambers remain in service at any level, it would seem obligatory to ensure that personnel exposed to this environment are sufficiently comfortable and proficient at functioning in-chamber that there is no reduction in the response time to potential crises. We believe that regular exposure to this environment during routine treatments plays an essential role in the maintenance of a safe workplace.

Conclusion

This 14-year review of multiplace hyperbaric attendant health demonstrates that maintenance of a safe workplace for in-chamber attendants does not pose a serious problem. Conservative decompression strategies on our most frequently used (institution-specific) 243 kPa table and routine use of oxygen are likely to account for our low DCS risk when compared with previously published series. Minor degrees of ENT barotrauma were the most commonly encountered hyperbaric-specific injury. Overall injury rates compare favourably with those encountered by the nursing profession in other areas of practice. Our institutional policies and procedures appear to provide an acceptably safe working environment and therefore, will remain unchanged.

Acknowledgements

The authors thank Mr Sean Rubidge, Senior Hyperbaric Technical Officer, for establishing the hyperbaric database in the first instance and, more recently, his assistance in data extraction, and all our chamber personnel for their cooperation and support.

References

1 Brattebø G, Aanderud L, Risberg J, Thorsen E, Forland M. Incidence of decompression illness among HBO nurses. J Slovenian Med Assoc. *23rd Annual Scientific Meeting of the European Underwater and Baromedical Society*, Programme and Abstracts. Bled, Slovenia; 1997.

2 Klossner J, Niinikoski J, Kaunisto M, Virolainen R. Risk of decompression sickness (DCS) among attending nursing personnel during HBO therapy in multiplace chamber: what measures should be taken to optimise the safety of personnel? In: *Proceedings of the 12th International Congress on Hyperbaric Medicine*. Milan, Italy; 1996. p. 335-8.

3 Risberg J, Englund M, Aanderud L, Eftedal O, Flook V, et al. Venous gas embolism in chamber attendants after hyperbaric exposure. *Undersea Hyperb Med*. 2004;31(4):417-29.

4 *DCIEM Diving Manual. Part 1: Air decompression procedures*

and tables. DCIEM No. 86-R-35. Toronto, ON: Defence and Civil Institute of Environmental Medicine; 1992.
5 Cooper PD, Van den Broek C, Smart DR, Nishi RY, Eastman D. Hyperbaric chamber attendant safety I: Doppler analysis of decompression stress in multiplace chamber attendants. *Diving and Hyperbaric Medicine.* 2009;39(2):63-70.
6 *Compendium of Workers' Compensation Statistics, Australia 1990-00.* Canberra, ACT: National Occupational Health and Safety Commission; 2002.
7 Brown JG, Trinkoff A, Rempher K, McPhaul K, Brady B, et al. Nurses' inclination to report work-related injuries. *AAOHN J*. 2005;53(5):213-7.
8 Langford E. *Buried but not dead, a survey of occupational illness and injury incurred by nurses in the Victorian health service industry*. Melbourne, VIC: Injured Nurses Support Group, Victorian Branch; Australian Nursing Federation; 1997. p. 7.
9 Wunderlich GS, Sloan FA, Davis CK, editors. *Nursing staff in hospitals and nursing homes – is it adequate?* Committee on the Adequacy of Nurse Staffing in Hospitals and Nursing Homes; Institute of Medicine; Washington DC: National Academy Press; 1996. p. 170-2.
10 Hignett S. Work-related back pain in nurses. *J Adv Nurs*. 2008;23(6):1238-46.
11 Thalmann ED. Principles of US Navy recompression treatments for decompression sickness. In: Moon RE, Sheffield PJ, editors. *Treatment of decompression illness*. 45th UHMS Workshop. Kensington, MD: Undersea and Hyperbaric Medical Society; 1996. p. 75-95.
12 Dunford RG, Hampson NB. Gender-related risk of decompression sickness in hyperbaric chamber attendants: a case control study. *Undersea Biomed Res*. 1992;19(Suppl.):37.
13 Dietz SK, Myers RAM. Decompression illness in HBO inside tenders: a review of 23 years of exposures. *Undersea Hyperb Med*. 1995;22(Suppl.):57.

Received: 03 December 2008
Accepted: 31 March 2009

Paul David Cooper, MB, ChB, FJFICM, FANZCA, DipDHM, was initially Senior Registrar in Hyperbaric & Diving Medicine, then subsequently Deputy Director of Anaesthesia and Staff Specialist in Intensive Care and Diving & Hyperbaric Medicine, Royal Hobart Hospital, Hobart, Australia during the study period.
Corry Van den Broek, DipAppSc, CPO-CD (RANR), is the Facility Manager and Senior Hyperbaric Technical Officer, Department of Diving & Hyperbaric Medicine, Royal Hobart Hospital, Hobart.
David Smart, MBBS (Hons), MD, FACEM, FIFEM, FACTM, FAICD, DipDHM, is Medical Co-Director, Department of Diving & Hyperbaric Medicine, Royal Hobart Hospital, and Director of Emergency Medicine, Calvary Health Care, Hobart.

Address for correspondence:
Dr David Cooper,
Department of Diving & Hyperbaric Medicine,
Royal Hobart Hospital,
GPO Box 1061L, Hobart,
Tasmania 7001, Australia
Phone: *+61-(0)3-6222-8193*
Fax: *+61-(0)3-6222-7268*
E-mail: *<david.cooper@dhhs.tas.gov.au>*

This paper is based on part of the thesis submitted by Dr Cooper for the SPUMS Diploma of Diving and Hyperbaric Medicine.

Time course of the reduction in nitric oxide concentration in exhaled gas after exposure to hyperbaric hyperoxia

Ida Kjelkenes and Einar Thorsen

Key words
Hyperbaric oxygen therapy, inflammation, lung function, oxygen toxicity

Abstract

(Kjelkenes I, Thorsen E. Time course of the reduction in nitric oxide concentration in exhaled gas after exposure to hyperbaric hyperoxia. *Diving and Hyperbaric Medicine*. 2009;39(2):77-80.)
Exposure to hyperoxia is associated with oxidative stress and is known to cause inflammation in the lung and the airways. Exhaled nitric oxide concentration (FE_{NO}) is a marker of some inflammatory processes in the lung and airways, and is reduced immediately after a single session of hyperbaric oxygen (HBO) exposure. The purpose of this study was to characterize the time course of this decrease in FE_{NO}. Ten patients who had HBO treatment were included. The daily HBO exposures were at a pressure of 240 kPa for 90 min. FE_{NO} was measured before a single HBO treatment session and immediately after and 30, 60, 120, and 240 minutes after. Thirteen healthy controls had FE_{NO} measured at the same time intervals as the patients without hyperoxic or hyperbaric exposure. FE_{NO} was sigificantly reduced by 30.0 (SD 22.3) % (P = 0.009) immediately after HBO treatment. It remained reduced by 27.3 (SD 19.6) % (P = 0.013) at 120 min, and had not recovered completely by 240 min. There were no changes in FE_{NO} in the control group. The results confirm the finding of a decrease in FE_{NO} immediately after exposure to hyperbaric hyperoxia. The reduction in FE_{NO} persists for up to 240 min.

Introduction

Exposure to hyperbaric oxygen (HBO) is associated with an increased production of reactive oxygen species (ROS) and may induce an acute toxic effect on the lung.[1] The ROS reacts non-specifically with DNA, fatty acids and proteins causing intracellular and membrane dysfunction. There is a dose dependent reduction in vital capacity when the oxygen pressure is over 50 kPa.[2] Forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), maximal expiratory flow rates, and transfer factor for carbon monoxide (Tl_{CO}) are all reduced, and the exposure to hyperoxia contributes to the acute effects of a saturation dive on lung function.[3] Reductions in maximal expiratory flow rates and Tl_{CO} have also been demonstrated in patients having HBO therapy.[4–6] These studies describe a variable reduction in lung function, but they all conclude that the effects on lung function of a standard HBO treatment series of 20 – 30 HBO exposures are of minor clinical importance. However, residual effects of repeated treatment series may accumulate to a long-term effect, and more sensitive indicators of oxygen toxicity than the traditional lung function variables may be needed to detect such effects.

Nitric oxide (NO) concentration in exhaled gas (FE_{NO}) is a biochemical marker of some airway inflammation.[7] It is consistently increased with the eosinophilic airway inflammation of asthma, and may be used to monitor disease severity and response to treatment.[8] With other lung disease, including chronic obstructive lung disease and exacerbations thereof which are associated with neutrophilic inflammatory responses, the FE_{NO} may be unchanged or increased.[9] FE_{NO} is consistently decreased in current smokers.[10] Because of the non-invasive technique and simple procedure, FE_{NO} measurements are ideal for monitoring inflammatory responses that are associated with changes in the NO production in the lungs and airways.

A reduction in FE_{NO} immediately after a single session of HBO exposure in patients having HBO therapy has recently been demonstrated in two studies.[11,12] In the study of Puthucheary et al, baseline FE_{NO} was larger than in the control group.[11] In the study of Taraldsøy et al, baseline FE_{NO} was not different from the control group, and it remained unchanged during the course of a four-week HBO treatment series (20 HBO exposures), with all FE_{NO} measurements taken before HBO treatment.[12] The decrease in FE_{NO} immediately after a single HBO treatment session was the same and approximately 30% on both the first and ninteenth day of treatment, indicating complete recovery within 24 hrs.

The aim of this study was to characterize the time course of the reduction in FE_{NO} after a single exposure to hyperbaric hyperoxia. The time course has to be known before further studies of the mechanisms of these changes are initiated, and the time course itself may give some indication of which mechanisms are operative.

Methods

SUBJECTS

Ten non-smoking patients (seven women), aged 43–73 years, with normal lung function and receiving HBO treatment for radiation-induced tissue injury were studied. Participants

Table 1
Subject demographics; FVC – forced vital capacity; FEV_1 – forced expiratory volume in 1 sec

	HBO exposed (n = 10)		**Controls** (n = 13)	
Age (yrs, range)	57	(43–73)	48	(24–62)
Height (m, range)	1.69	(1.55–1.79)	1.73	(1.60–1.86)
FVC (L) (SD)	3.83	(0.88)	4.71	(0.91)
FVC (% predicted) (SD)	100.3	(15.2)	106.0	(10.2)
FEV_1 (L) (SD)	2.82	(0.74)	3.67	(0.74)
FEV_1 (% predicted) (SD)	90.7	(12.9)	100.5	(12.3)

were selected from consecutive patients referred for HBO treatment. Exclusion criteria were respiratory symptoms, known heart or lung disease, diabetes mellitus and atopy. The lungs and trachea had not been included in the radiation field. Thirteen healthy subjects, all never smokers, and mainly recruited from the hospital staff served as controls. There were eight men and five women, aged 24–62 years. Subjects' characteristics, including lung function by spirometry, are given in Table 1. The study was approved by the Regional Ethics Review Committee and all subjects gave written informed consent.

PROTOCOL

The HBO exposure was at a pressure of 240 kPa for 90 min, five days a week and scheduled for 2–6 weeks. All HBO treatments were given in a monoplace hyperbaric chamber, and oxygen exposure was in three cycles of 30 minutes interrupted by five-minute breaks breathing air.

All measurements took place between 0900 h and 1600 h on one of the HBO treatment days during the second week. FE_{NO} was measured within 30 minutes before HBO treatment and was repeated as soon as practically possible after treatment, usually within 10 min. Thereafter measurements of FE_{NO} were taken 30, 60, 120 and 240 min after treatment. The baseline measurement was at least one hour after breakfast and drinking water only was allowed until the measurement at 60 min after treatment was accomplished. A meal was allowed between the measurements 60 and 120 min after HBO exposure. Measurements of FE_{NO} were not done before the start on the first day of HBO treatment. The control group followed the same time interval and food and drink restrictions as the patients.

SPIROMETRY

Forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) were measured using a wedge spirometer (Vitalograph Ltd., Buchingham, England). Spirometric testing was performed in the sitting position with a nose clip. The highest result of at least three satisfactory tests was recorded for each subject.

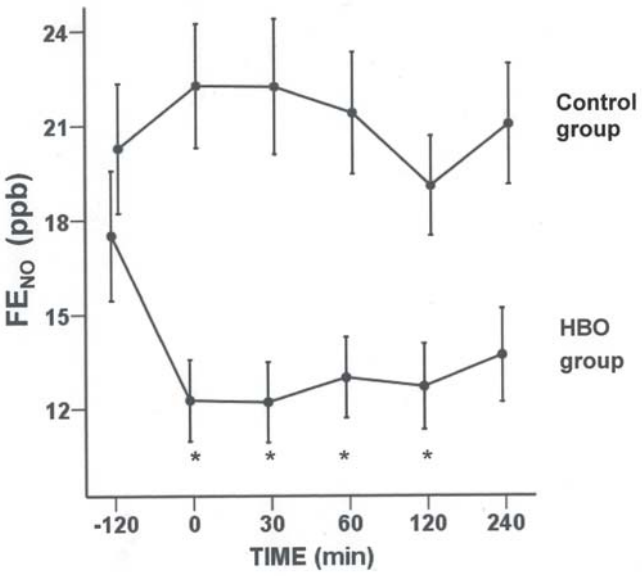
CONCENTRATION OF NITRIC OXIDE IN EXHALED GAS (FE_{NO})

Exhaled nitric oxide concentration (FE_{NO}) was measured by an online chemiluminiscence analyser (Niox, Aerocrine AB, Stockholm, Sweden). It was done with controlled expiration of 50 ml per second from total lung capacity after inhalation from functional residual capacity as recommended by the American Thoracic Society and European Respiratory Society.[13] The mean value of three tests not differing more than 10% or 2 ppb was used in the analyses.

STATISTICS

All data are given as mean (SD). Changes in FE_{NO} from baseline were tested by repeated measures ANOVA. A *P*-value < 0.05 was considered statistically significant.

Figure 1
Nitric oxide concentration (FE_{NO}), mean and SEM, in exhaled gas in the control group and HBO-exposed group; exposure took place between -120 and 0 min. * *P* < 0.05 compared with control group



Results

The results of the measurements of FE_{NO} are shown in Figure 1. FE_{NO} was significantly reduced by 30.0 (SD 22.3)% ($P = 0.009$) immediately after HBO treatment, and FE_{NO} was still significantly reduced 120 min after HBO treatment ($P = 0.013$). FE_{NO} was not statistically different from baseline after 240 min, the reduction being 21.8 (SD 15.0)% ($P = 0.083$). There were no changes in FE_{NO} in the control group.

Discussion

This study demonstrated that the reduction in FE_{NO} persists for up to four hours after exposure to hyperbaric hyperoxia. A free radical chain reaction involving NO and O_2 can propagate and scavenge NO in the gas phase of the bronchi and alveoli and within the tissue at the end of HBO therapy. In the gas phase, this process is likely to be reversed after complete washout of the gas in the lung, which takes less than ten minutes in subjects with normal lung function.[14] However, in the airway and alveolar walls more complex chemical reactions scavenging NO may still propagate.

Inducible nitric oxide synthase (iNOS) is the major airway epithelial isoform of the nitric oxide synthases.[15] iNOS activity is regulated by oxygen concentration in normobaric hyperoxic, normoxic and hypoxic conditions,[16] but there are no studies of iNOS regulation in hyperbaric hyperoxic conditions. iNOS is generally not expressed, but it is activated by inflammatory cytokines as for example peroxynitrite.[17,18] It could be expected that NO levels would rise after exposure to toxic HBO concentrations. However, the findings of this study and the study by Puthucheary et al showed the opposite. FE_{NO} was decreased after hyperbaric hyperoxia.[11] Inhibition of iNOS could be a mechanism of the reduction of FE_{NO} seen with HBO therapy.[19,20] The patients studied by Puthucheary et al had a higher baseline FE_{NO} compared with the controls.[11] This could support an upregulation of iNOS. Nevertheless, FE_{NO} was decreased after hyperoxic exposure. Taraldsøy et al did not demonstrate higher baseline levels of FE_{NO} in the HBO treated subjects.[12] Cucchiaro et al observed an upregulation of iNOS expression in rat lungs after exposure to 85% oxygen without increase in FE_{NO}.[20] This could be explained by a concomitant inhibition of iNOS activity, but biochemical mechanisms for this are not known. Another explanation could be that iNOS is induced, but the produced NO is not measurable in exhaled gas because it is scavenged in the interstitium.

Studies of rabbits have shown that NO detected in expired gas is derived primarily from the lung epithelial cells, and not by diffusion from the interstitium and blood circulation.[21] If so, it can be assumed that the reduction in FE_{NO} after HBO therapy is due to processes involving the production or scavenging of NO in the airway epithelium.

NO is involved in the regulation of vasomotor tone, and hyperoxia induces vasoconstriction. Studies of NO in coronary arteries in pigs provide evidence that hyperoxia reduces the basal release of NO leading to endothelium-dependent vasoconstriction.[22] It is not known how the bronchial circulation responds to changes in NO.

Patients having HBO treatment for some underlying disorder do not constitute an ideal group for studying these effects. The patients in Putucheary's and our studies could have ongoing inflammatory processes influencing the NO turnover, and patients having had radiation for head-and-neck cancers could have processes in the mouth with a bacterial flora influencing the FE_{NO}. Even though the patients as well as the controls had normal dynamic lung volumes, the phenomenon ought to be confirmed in studies of healthy subjects.

Conclusion

FE_{NO} is reduced for at least four hours after a single hyperbaric hyperoxic exposure to 240 kPa. The time course of the changes indicates more complex mechanisms than simple scavenging of NO by O_2 in alveolar and bronchial gas.

Acknowledgements

This study was supported by StatoilHydro and ExxonMobile Norway.

References

1 Freeman BA, Crapo JD. Hyperoxia increases oxygen radical production in rat lungs and lung mitochondria. *J Biol Chem*. 1981;256:10986-92.
2 Clark JM, Lambertsen CJ. Pulmonary oxygen toxicity: a review. *Pharmacol Rev*. 1971;23:37-133.
3 Thorsen E, Segadal K, Reed JW, Elliott C, Gulsvik A, Hjelle JO. Contribution of hyperoxia to reduced pulmonary function after deep saturation dives. *J Appl Physiol*. 1993;75:657-62.
4 Thorsen E, Aanderud L, Aasen TB. Effects of standard hyperbaric oxygen treatment protocol on pulmonary function. *Eur Respir J*. 1998;12:1442-5.
5 Mialon P, Barthlemy L, Michaud A, Lacour JM. Pulmonary function in men after repeated sessions of oxygen breathing at 0.25 Mpa for 90 Min. *Aviat Space Environ Med*. 2001;72:215-8.
6 Pott F, Westergaard P, Mortensen J, Jansen EC. Hyperbaric oxygen treatment and pulmonary function. *Undersea Hyperb Med*. 1999;26:225-8.
7 Kharitonov SA, Barnes PJ. Exhaled markers of pulmonary disease. *Am J Respir Crit Care Med*. 2001;163:1693-722.
8 Kharitonov SA, Barnes PJ. Clinical aspects of exhaled nitric oxide. *Eur Respir J*. 2000;16:781-92.
9 Maziak W, Loukides S, Culpitt S, Sullivan P, Kharitonov SA, Barnes PJ. Exhaled nitric oxide in chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 1998;157:998-1002.

10 Malinovschi A, Janson C, Holmkvist T, Norbäck D, Meriläinen P, Högman M. Effect of smoking on exhaled nitric oxide and flow-independent nitric oxide exchange parameters. *Eur Respir J*. 2006;28:339-45.
11 Puthucheary ZA, Liu J, Bennett M, Trytko B, Chow S, Thomas PS. Exhaled nitric oxide is decreased by exposure to hyperbaric oxygen therapy environment. *Mediators Inflamm*. 2006;doi:2006(5):72620.
12 Taraldsøy T, Bolann BJ, Thorsen E. Reduced nitric oxide concentration in exhaled gas after exposure to hyperbaric hyperoxia. *Undersea Hyperb Med*. 2007;34:321-7.
13 ATS/ERS. Recommendations for standardized procedures for the online and offline measurements of exhaled lower respiratory nitric oxide and nasal nitric oxide in adults and children. *Am J Respir Crit Care Med*. 2005;171:912-30.
14 Fowler WS, Cornish ER Jr, Kety SS. Lung function studies VIII. Analysis of alveolar ventilation by pulmonary N2 clearance curves. *J Clin Invest*. 1952;31:40-50.
15 Guo FH, De Raeve HR, Rice TW, Stuehr DJ, Thunnissen FB, Erzurum SC. Continous nitric oxide synthesis by inducible nitric oxide synthase in normal human airway epithelium in vivo. *Proc Natl Acad Sci USA*. 1995;92:7809-13.
16 Dweik RA, Laskowski D, Abu-Sound HM, Kaneko T, Hutte R, et al. Nitric oxide synthesis in the lung. *J Clin Invest*. 1998;101:660-6.
17 Hecker M, Cattaruzza M, Wagner AH. Regulation of inducible nitric oxide synthase expression in vascular smooth muscle cells. *Gen Pharmacol*. 1999;32:9-16.
18 Cooke CL, Davidge ST. Peroxynitrite increases iNOS through NF-kappaB and decreases prostacyclin synthase in endothelial cells. *Am J Physiol Cell Physiol*. 2002;282:C395-C402.
19 Pedoto A, Nandi J, Yang ZJ, Wang J, Bosco G, et al. Beneficial effect of hyperbaric oxygen pretreatment on lipopolysaccharide-induced shock in rats. *Clin Exp Pharmacol Physiol*. 2003;30:482-8.
20 Cucchiaro G, Tatum AH, Brown MC, Camporesi EM, Daucher JW, Hakim TS. Inducible nitric oxide synthase in the lung and exhaled nitric oxide after hyperoxia. *Am J Physiol*. 1999;277:L636-L644.
21 Gustafsson LE, Leone AM, Persson MG, Wiklund NP, Moncada S. Endogenous nitric oxide is present in the exhaled air of rabbits, guinea pigs and humans. *Biochem Biophys Res Commun*. 1991;181:852-7.
22 Pasgaard T, Stankevicius E, Jørgensen MM, Ostergaard L, Simonsen U, Frøbert O. Hyperoxia reduces basal release of nitric oxide and contracts porcine coronary arteries. *Acta Physiology (Oxf)*. 2007;191:285-96.

Received: 17 February 2009
Accepted: 18 May 2009

Ida Kjelkenes is a student at the Medical School, University of Bergen, holding a research grant at the Institute of Medicine, University of Bergen.
Einar Thorsen, MD, PhD, is Professor of Hyperbaric Medicine, Institute of Medicine, University of Bergen and Consultant in Hyperbaric Medicine at the Department of Hyperbaric Medicine, Haukeland University Hospital, Bergen, Norway.

Address for correspondence:
Einar Thorsen, Institute of Medicine, University of Bergen, Haukeland University Hospital, N-5021 Bergen, Norway
Phone: *+47-(0)55-973973*
Fax: *+47-(0)55-975137*
E-mail: *<einar.thorsen@helse-bergen.no>*

This paper is based on a presentation at the 2008 EUBS Meeting, Graz, Austria, and is a revised version of the manuscript appearing in the EUBS Proceeedings.

Review articles

Adjunctive therapy for decompression illness: a review and update

Richard E Moon

Key words
Decompression illness, decompression sickness, diving accidents, air embolism, first aid, treatment, review article

Abstract

(Moon RE. Adjunctive therapy for decompression illness: a review and update. *Diving and Hyperbaric Medicine*. 2009;39(2):81-7.)
Therapeutic interventions may augment the outcome or provide temporizing support pending recompression. Effective measures include first aid (surface) oxygen therapy, fluid resuscitation, non-steroidal anti-inflammatory drugs and avoidance of fever. Lignocaine may also be effective, particularly for cerebral arterial gas embolism (CAGE). For critically ill patients with CAGE, animal studies do not support the use of hyperventilation. There is strong experimental evidence in animals for the efficacy of intravenous perfluorocarbon. When lower limb paralysis occurs, low molecular weight heparin is recommended to reduce the risk of venous thromboembolism.

Introduction

Adjunctive therapies had their origins in the nineteenth century during the early days of compressed air work. Prior to the advent of therapeutic recompression therapy a variety of concoctions and techniques were empirically used. These included ergot, morphine, atropine, alcohol, ginger and phlebotomy. Dr Andrew Smith, physician for the Brooklyn Bridge construction wrote the following:

"*On my recommendation a cup of good coffee was served to each man immediately upon leaving the caisson. It appeared to relieve, in a measure, the nervous prostration which marked the return to the open air; and possibly, by the effect which coffee is known to have, it may have done something, also to check the tendency to too rapid tissue-change.*

In some instances I have obtained the very best results from hypodermic injections of atropine at the seat of the pain; but in other cases it failed to procure relief, and, upon the whole, I consider it inferior to morphine."

"Starting from the theory already given as to the mode in which the disease is produced (a theory which was constructed wholly upon the observations of others), I was led to the idea that benefit would be derived from the use of an agent that would induce contraction of the capillaries, and thus correct the want of tone which I consider to lie at the foundation of the difficulty. For this purpose I proposed the use of ergot before I had ever seen a case of the disease. I reasoned that it would be useful, first, by contracting the vessels of the brain and spinal cord, and relieving their congested state; and secondly, by restoring tone to the superficial vessels, and thus imparting vigor to the circulation.

An extended trial warrants me in saying that the results have justified the theory. In my hands, though not always successful, ergot has certainly been very useful in a considerable number of cases. I have seen very severe pain completely relieved within half an hour after the administration of a drachm of the fluid extract. In other instances, unsteadiness of the limbs, which seemed about to usher in paralysis, has yielded promptly to one or two doses."

"But perhaps the best evidence of its usefulness is to be found in the preference for it of the night-porter, who had charge of the hospital at night, and who was instructed in the use of the few medicines employed, and treated such cases as occurred among the men composing the night gangs. Having both morphine and ergot at hand, he gradually, and of his own accord, almost abandoned the former, declaring that the ergot was more prompt and certain in relieving the pains. This from an intelligent, unprejudiced, non-professional source, is strong testimony in favor of the efficacy of the drug."[1]

After recompression therapy became routine, adjunctive therapies fell out of favour until the latter part of the twentieth century, when it became apparent that decompression illness was often accompanied by severe dehydration and can be complicated by thromboembolism, which is sometimes fatal. Additionally, because civilian diving accidents often occur far from recompression chambers, there has been an impetus to develop therapies that can provide temporary support during transport. Currently available adjunctive therapies are shown in Table 1.

Table 1
Adjunctive agents for decompression illness

First-aid oxygen
Fluid resuscitation and management of plasma glucose
Antiplatelet agents and DVT prophylaxis
Corticosteroids
Lignocaine (lidocaine)
Body temperature management
Manipulation of $PaCO_2$
Perfluorocarbons

First-aid (surface) oxygen (O_2)

Paul Bert published his observations on experimental animals with decompression sickness in 1878.[2] By direct observation, he found that oxygen (O_2) administration to dogs with decompression sickness caused resolution of intravascular bubbles. The value of first-aid oxygen (F_AO_2) has been confirmed by clinical experience and a systematic review of 2,231 consecutive diving accident reports.[3] F_AO_2 was often associated with complete relief of symptoms prior to recompression, and reduced need for more than one recompression treatment (Figure 1). Receiving early F_AO_2 (within four hours of surfacing) was more efficacious.

Fluid resuscitation and management of plasma glucose

In 1871, Alphonse Jaminet, physician for the Mississippi Bridge project at St Louis, published his observation on urine specific gravity of men with and without decompression sickness.[4] Although he did not interpret the results in this context, retrospective analysis of his observations shows that urine specific gravity was significantly higher in the men with decompression sickness compared to those without (Figure 2). The higher specific gravity in symptomatic men is consistent with dehydration. Studies published in the middle of 20th century described haemoconcentration in cases of severe decompression sickness (Figure 3).[4,6] In 1964 Brunner published two cases of severe decompression sickness (DCS) after experimental dives with haemoconcentration. In those cases he used radioactive tagging to demonstrate that plasma volume was reduced in the face of normal packed erythrocyte volume and that the plasma deficit could be corrected with administration of plasma.[7]

A more recent paper has described the association of haemoconcentration with sequelae of decompression illness (DCI) (Figure 4).[8] Divers with neurological DCI with and without neurological sequelae one month after treatment and a series of control divers without symptoms were compared; the haematocrit was significantly higher in the divers with

Figure 1
First-aid oxygen and outcome in a series of recreational divers with DCI; * $P < 0.05$; ** $P < 0.001$

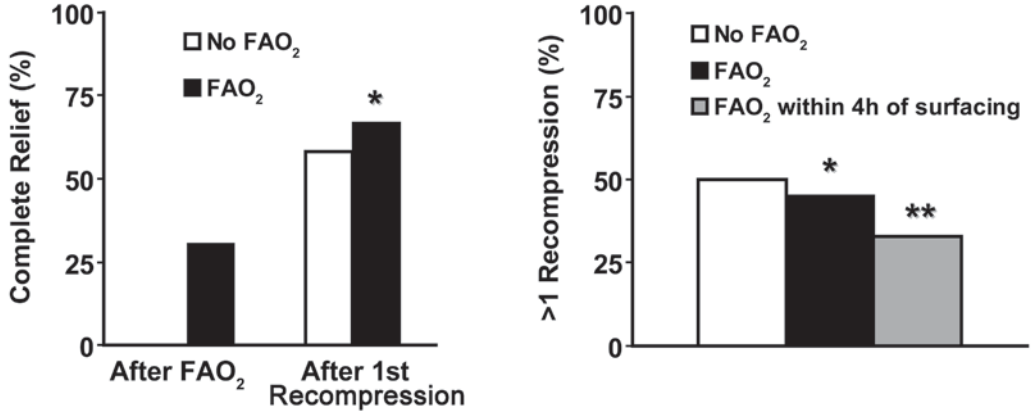


Figure 2
Urine specific gravity in compressed air workers with and without DCS (mean ± SD); * $P < 0.05$
Data plotted from observations by Jaminet[5]

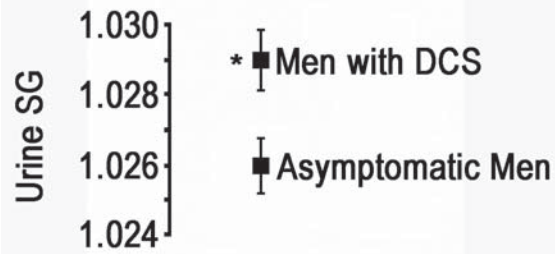


Figure 3
Plasma volume decrement in 7 cases of severe altitude-induced DCS (from Malette[6])

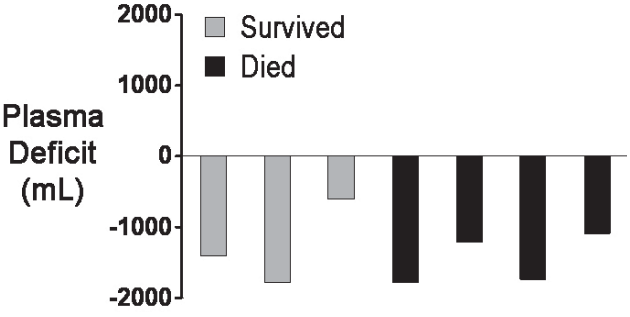


Figure 4
Haematocrit in divers with DCI; median, minimum and maximum haematocrit values are shown. Data plotted from Boussuges[8]

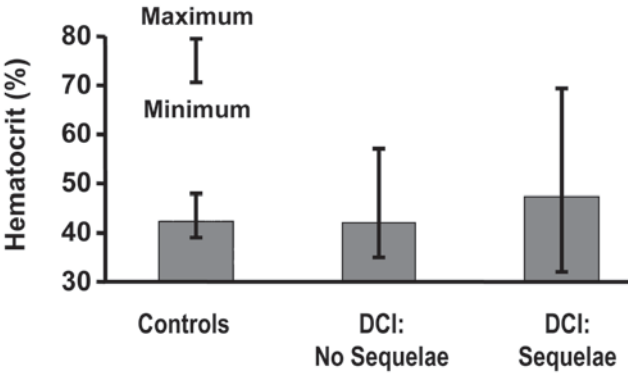
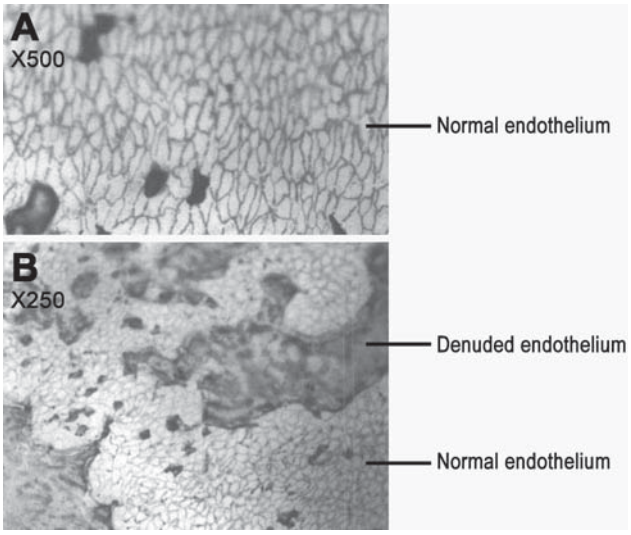


Figure 5
Light microscopy of endothelium from the pulmonary artery of a pig subjected to decompression-related venous gas embolism (VGE); A: normal endothelium; B: endothelium partially denuded after VGE; from Nossum[9], with permission



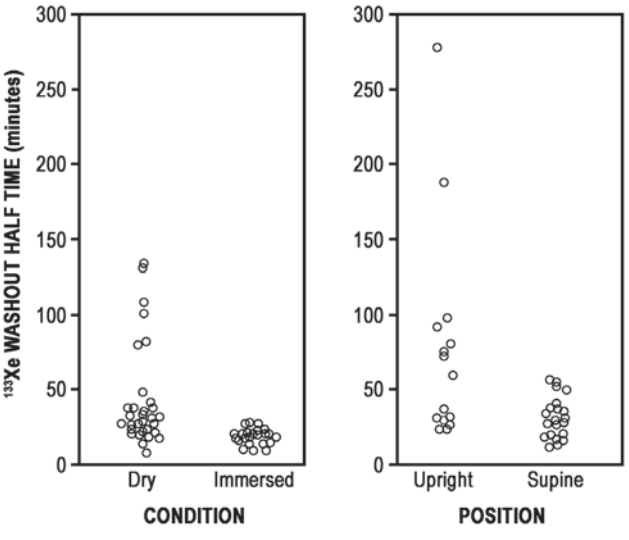
sequelae. The mechanism for haemoconcentration appears to be the result of bubble-induced endothelial damage (Figure 5) and extravasation of plasma into the interstitial space.

Evidence that fluid administration may be helpful in DCI is suggested by a study by Balldin and Lundgren in which inert gas washout (^{133}Xe) from the anterior tibial muscle is facilitated by immersion in water and the supine position (Figure 6).[10] Both head-out immersion and supine position were associated with faster Xe washout, consistent with the expected greater tissue perfusion. These data support the use of fluid resuscitation in patients with DCI in order to facilitate inert gas washout. Immersion causes redistribution of blood from venous capacitance vessels to the central circulation, equivalent to a blood or fluid transfusion, and hence increased tissue perfusion.

The accumulated experience over the last 130 years strongly supports the use of fluid resuscitation in decompression illness. Hypotonic fluids, such as dextrose-water or half normal saline, reduce plasma osmolality and can cause swelling of the central nervous system.[11] Moreover, elevation of plasma glucose can augment CNS injury.[12] Therefore, fluids that contain glucose should be avoided. Ideal fluids include isotonic fluids, either colloids or crystalloids. For severe cases large volumes are often required. In one patient with severe DCI treated at our facility, reduction in his haematocrit from 66% to 48% required 12 litres of crystalloid and 1 litre of starch solution. Aggressive fluid resuscitation is not recommended for patients with isolated cerebral arterial gas embolism (CAGE), either diving-related or iatrogenic, unless there is concomitant haemoconcentration.

If oral fluids are used for resuscitation, their ideal composition for rapid absorption and maintenance of electrolyte balance has been determined. The ideal oral fluid for resuscitation of a dehydrated patient should contain 30–60 mM.L^{-1} sodium, have an osmolality of around 240 mOsm.kg^{-1} and a glucose concentration of 70–150 mmol.L^{-1}.[13] The addition of glucose facilitates water absorption from the gut, and in non-diabetics causes no significant rise in plasma glucose. Some fluids (e.g., Gatorade® in the USA) are close to ideal but most commercially available fluids, such as juices or carbonated beverages, are far from ideal. However, in the absence of the ideal fluid, water or other beverages can be used. Protein-containing fluids such as milk are not recommended because they delay absorption of fluid from the gut. Beverages containing caffeine or alcohol are not recommended because of their tendency to promote diuresis.

Figure 6
^{133}Xe washout half times from the anterior tibial muscle in human volunteers. Graphs drawn from the data published by Balldin & Lundgren[10]



Anti-platelet agents and deep vein thrombosis (DVT) prophylaxis

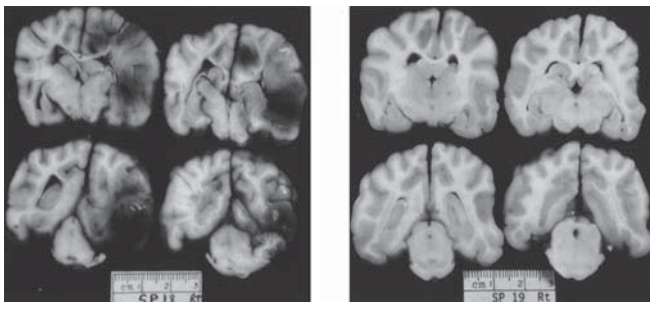
Evidence that bubbles in divers may activate platelets has suggested the use of aspirin and dextran to inhibit clot formation in symptomatic divers after decompression.[14–17] Retrospective anecdotal evidence supporting the use of aspirin, oxygen, corticosteroids and dextran was first published by Fructus.[18] To test this hypothesis, Mike Bennett and colleagues performed a randomized double blind controlled study of the non-steroidal anti-inflammatory drug (NSAID) tenoxicam (a non-selective cyclooxygenase inhibitor) versus placebo. The results of the trial indicated that although there was no long-term outcome difference in divers who received tenoxicam, the number of recompressions required to reach a therapeutic plateau was reduced by one (two treatments versus three treatments in the placebo group).[19]

Leg immobilization due to spinal cord DCS is likely to promote deep vein thrombosis and thromboembolism.[20] Therefore, while routine full anticoagulation is not indicated, prophylactic measures such as low molecular weight heparin are recommended when significant leg weakness is present.[21]

Corticosteroids

In addition to their anti-inflammatory effects and ability to reduce brain volume, corticosteroids may have antioxidant properties. However, several studies of corticosteroids in animal models of decompression illness have revealed no advantage in short term outcome.[22–25] Nevertheless, some human outcome studies have suggested that a loading dose of methylprednisolone 30 $mg.kg^{-1}$ with a 23-hour infusion at 5.4 $mg.kg^{-1}$ per hour has resulted in improved long-term outcome in humans with spinal cord trauma.[26] Unfortunately, the outcome scales used in this study and others performed by the same group are difficult to interpret, and the improvements in outcome may be marginal.[27] A more recent trial did not find a benefit for methylprednisolone in acute spinal cord injury but did report more infections when it was used.[28] The challenges of performing outcome studies in acute spinal cord injury have recently been discussed.[29] A review of the use of high-dose methylprednisolone in DCI has resulted in a recommendation that corticosteroids not be used in DCI.[21]

Figure 7
Brain slices from dogs in which injury was induced by inflation of an epidural balloon; left – brain of a normothermic dog; right – brain of a dog cooled to a core temperature of 31°C (see text for details); from Pomeranz, with permission[38]



Lignocaine (lidocaine)

Lignocaine (lidocaine) has been examined in various forms of DCS, particularly CAGE, in animals and found to have beneficial effects.[30,31] Case reports in which administration of lignocaine appeared to be beneficial for DCS and gas embolism and systematic reviews provide support for its administration to patients.[30–33] Randomized outcome studies in diving-related CAGE are virtually impossible to perform because of the rarity of the condition. However, open-heart surgery may be a reasonable surrogate, as arterial bubbles are commonly observed during these procedures. A randomized study of lignocaine in open-heart surgery was associated with a significant improvement in long-term outcome.[34] This has been followed by two other studies demonstrating improvement in outcome with lignocaine after cardiopulmonary bypass.[35,36] Lignocaine is a relatively benign drug when given intramuscularly to provide a short term effect or when administered via infusion when it can be monitored. It therefore remains an optional therapy that may provide some benefit, particularly in CAGE.

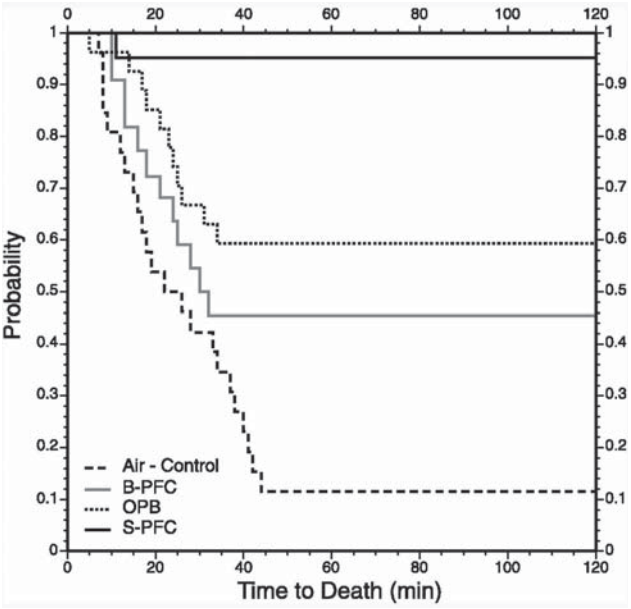
Body temperature management

Induced hypothermia has been observed to be beneficial in numerous animal models of brain injury.[37] Figure 7 shows brain slices from dogs in which injury was induced by inflation of an epidural balloon to maintain an intraventricular pressure of 62 mmHg for 90 minutes. On the left is the brain of a normothermic dog who developed brain death. On the right is the brain of a dog in which cooling was instituted 15 minutes after balloon inflation. A core temperature of 31°C was maintained for 5 hours, after which a core temperature of 35°C was maintained until 7–62 hours post insult; standard resuscitative maneouvres and anesthesia were maintained throughout.[38] Conversely hyperthermia is detrimental. Two hypothermia studies have demonstrated improved outcome after global ischaemia during cardiac arrest.[39,40] Although there is no evidence as yet that hypothermia is useful for other forms of brain injury,[41] there is evidence in humans that hyperthermia is detrimental.[42,43] Therefore, although hypothermia has not been embraced as a therapy for neurological DCS it is strongly recommended that fever in the setting of DCI be aggressively treated.[21]

Manipulation of arterial PCO_2

Hyperventilation and the resulting hypocapnia generally cause cerebral vasoconstriction and a reduction in intracranial pressure. CAGE can cause brain swelling and

Figure 8
Probability of death in pigs after decompression from 5 atmospheres in a dry hyperbaric chamber for 22 hours. Animals were randomized to one of four groups: air and saline (Air-Control); oxygen pre-breathe and saline (OPB); OPB with intravenous PFC given at depth (B-PFC); OPB with PFC given after surfacing (S-PFC).
Reproduced from Dainer[49], with permission



increased intracranial pressure,[44] thus, it has often been standard practice to induce hyperventilation following severe CAGE in an attempt to maintain cerebral perfusion and oxygenation. However, studies by Van Hulst have failed to show any evidence of improved cerebral perfusion pressure as a result of hyperventilation.[45] Moreover, brain glucose is lower and brain lactate is high in hyperventilated animals.[46] These studies support maintaining arterial PCO_2 within the normal range in patients with CAGE.

Perfluorocarbons

A new modality for treatment of DCS consists of perfluorocarbon (PFC) emulsion. These substances have a high solubility for both oxygen and inert gases. Administration of PFC emulsions in animal models of gas embolism or DCS have demonstrated improvement.[47–50] In particular, administration of PFC after decompression from air saturation in pigs has resulted in reduced DCS and mortality (Figure 8).[48,49] Animals were randomized to one of four groups: air and saline; O_2 pre-breathed (OPB) for 9 minutes before decompression at an inspired concentration >90% and saline; OPB with intravenous PFC (Oxygent, Alliance Pharmaceutical, San Diego, CA) 6 $mL.kg^{-1}$ given at depth; OPB with PFC given after surfacing. OPB combined with PFC administered after surfacing was associated with the greatest reduction in death rate due to DCI. While these substances are not yet commercially available and have never been tested in settings other than saturation-related DCS in animals, they hold great promise for use in acute DCS and CAGE. One caveat is that after administered PFC emulsions brain PO_2 may be increased, and hence CNS oxygen toxicity more likely during subsequent hyperbaric oxygen treatment. Human and animal studies are planned to address this issue.

Summary

In summary, administration of intravenous or oral fluid to maintain intravascular volume, blood pressure and urine output are recommended in addition to surface oxygen for all cases of decompression illness. NSAIDs, aspirin and lignocaine remain options. DVT prophylaxis in the form of low molecular weight heparin is recommended for patients in whom there is reduced mobility. $PaCO_2$ should be maintained within normal limits when treating critically ill patients with CAGE. Perfluorocarbons may be available within a year or two and may have a significant impact on outcome, particularly when recompression is not immediately available.

References

1 Smith AH. *The physiological, pathological and therapeutic effects of compressed air*. Detroit, MI: George S. Davis; 1886.
2 Bert P. *Barometric pressure (La pression barométrique)*. Bethesda, MD: Undersea Medical Society; 1978.
3 Longphre JM, Denoble PJ, Moon RE, Vann RD, Freiberger JJ. First aid normobaric oxygen for the treatment of recreational diving injuries. *Undersea Hyperb Med*. 2007;34:43-9.
4 Berry CA, King AH. Severe dysbarism in actual and simulated flight; a follow-up study of five cases. *US Armed Forces Med J*. 1959;10:1-15.
5 Jaminet A. *Physical effects of compressed air and of the causes of pathological symptoms produced on Man by increased atmospheric pressure employed for the sinking of piers in the construction of the Illinois and St. Louis bridge*. St. Louis, MO: Ennis; 1871.
6 Malette WG, Fitzgerald JB, Cockett AT. Dysbarism. A review of thirty-five cases with suggestion for therapy. *Aerospace Med*. 1962;33:1132-9.
7 Brunner F, Frick P, Bühlmann A. Post-decompression shock due to extravasation of plasma. *Lancet*. 1964;1:1071-3.
8 Boussuges A, Blanc P, Molenat F, Bergmann E, Sainty JM. Haemoconcentration in neurological decompression illness. *Int J Sports Med*. 1996;17:351-5.
9 Nossum V, Koteng S, Brubakk AO. Endothelial damage by bubbles in the pulmonary artery of the pig. *Undersea Hyperb Med*. 1999;26:1-8.
10 Balldin UI, Lundgren CEG, Lundvall J, Mellander S. Changes in the elimination of ^{133}Xe from the anterior tibial muscle in man induced by immersion in water and by shifts in body position. *Aerosp Med*. 1971;42:489-93.
11 Ayus JC, Varon J, Arieff AI. Hyponatremia, cerebral edema, and noncardiogenic pulmonary edema in marathon runners. *Ann Intern Med*. 2000;132:711-4.

12 Drummond JC, Moore SS. The influence of dextrose administration on neurologic outcome after temporary spinal cord ischemia in the rabbit. *Anesthesiology*. 1989;70:64-70.
13 Rao MC. Oral rehydration therapy: new explanations for an old remedy. *Annu Rev Physiol*. 2004;66:385-417.
14 Philp RB, Ackles KN, Inwood MJ, Livingstone SD, Achimastos A, et al. Changes in the hemostatic system and in blood and urine chemistry of human subjects following decompression from a hyperbaric environment. *Aerosp Med*. 1972;43:498-505.
15 Thorsen T, Klausen H, Lie RT, Holmsen H. Bubble-induced aggregation of platelets: effects of gas species, proteins, and decompression. *Undersea Hyperb Med*. 1993;20:101-19.
16 Baj Z, Olszanski R, Majewska E, Konarski M. The effect of air and nitrox diving on platelet activation tested by flow cytometry. *Aviat Space Environ Med*. 2000;71:925-8.
17 Bosco G, Yang ZJ, Savini F, Nubile G, Data PG, et al. Environmental stress on diving-induced platelet activation. *Undersea Hyperb Med*. 2001;28:207-11.
18 Fructus X. Treatment of serious decompression sickness. In: Davis JC, editor. *Treatment of Serious Decompression Illness and Arterial Gas Embolism*: Proceedings of the 20th Undersea Medical Society Workshop. Bethesda, MD: Undersea Medical Society; 1979. p. 37-43.
19 Bennett M, Mitchell S, Dominguez A. Adjunctive treatment of decompression illness with a non-steroidal anti-inflammatory drug (tenoxicam) reduces compression requirement. *Undersea Hyperb Med*. 2003;30:195-205.
20 Spadaro MV, Moon RE, Fracica PJ, Fawcett TA, Saltzman HA, et al. Life threatening pulmonary thromboembolism in neurological decompression illness. *Undersea Biomed Res*. 1992;19 (Suppl):41-2.
21 Undersea & Hyperbaric Medical Society. UHMS Guidelines for Adjunctive Therapy of DCI. In: Moon RE, editor. *Adjunctive therapy for decompression illness*. Kensington, MD: Undersea & Hyperbaric Medical Society; 2003. p. 184-9.
22 Francis TJR, Dutka AJ, Clark JB. An evaluation of dexamethasone in the treatment of acute experimental spinal decompression sickness. In: Bove AA, Bachrach AJ, Greenbaum LJ, Jr, editors. *Underwater and Hyperbaric Physiology IX* Proceedings of the Ninth International Symposium on Underwater and Hyperbaric Physiology. Bethesda, MD: Undersea and Hyperbaric Medical Society; 1987. p. 999-1013.
23 Francis TJR, Dutka AJ. Methylprednisolone in the treatment of acute spinal cord decompression sickness. *Undersea Biomed Res*. 1989;16:165-74.
24 Dutka AJ, Mink RB, Pearson RR, Hallenbeck JM. Effects of treatment with dexamethasone on recovery from experimental cerebral arterial gas embolism. *Undersea Biomed Res*. 1992;19:131-41.
25 Dromsky DM, Weathersby PK, Fahlman A. Prophylactic high dose methylprednisolone fails to treat severe decompression sickness in swine. *Aviat Space Environ Med*. 2003;74:21-8.
26 Bracken MB, Shepard MJ, Collins WF, Holford TR, Young W, et al. A randomized, controlled trial of methylprednisolone or naloxone in the treatment of acute spinal-cord injury. Results of the Second National Acute Spinal Cord Injury Study. *New Engl J Med*. 1990;322:1405-11.
27 Bracken MB, Shepard MJ, Holford TR, Leo-Summers L, Aldrich EF, et al. Administration of methylprednisolone for 24 or 48 hours or tirilazad mesylate for 48 hours in the treatment of acute spinal cord injury. Results of the Third National Acute Spinal Cord Injury Randomized Controlled Trial. National Acute Spinal Cord Injury Study. *JAMA*. 1997;277:1597-604.
28 Pointillart V, Petitjean ME, Wiart L, Vital JM, Lassie P, et al. Pharmacological therapy of spinal cord injury during the acute phase. *Spinal Cord*. 2000;38:71-6.
29 Tator CH. Review of treatment trials in human spinal cord injury: issues, difficulties, and recommendations. *Neurosurgery*. 2006;59:957-82; discussion 82-7.
30 Mitchell SJ, Benson M, Vadlamudi L, Miller P. Cerebral arterial gas embolism by helium: An unusual case successfully treated with hyperbaric oxygen and lidocaine. *Ann Emerg Med*. 2000;35:300-3.
31 Mitchell SJ. Lidocaine in the treatment of decompression illness: a review of the literature. *Undersea Hyperb Med*. 2001;28:165-74.
32 Cogar WB. Intravenous lidocaine as adjunctive therapy in the treatment of decompression illness. *Ann Emerg Med*. 1997;29:284-6.
33 Drewry A, Gorman DF. Lidocaine as an adjunct to hyperbaric therapy in decompression illness: a case report. *Undersea Biomed Res*. 1992;19:187-90.
34 Mitchell SJ, Pellett O, Gorman DF. Cerebral protection by lidocaine during cardiac operations. *Ann Thorac Surg*. 1999;67:1117-24.
35 Wang D, Wu X, Li J, Xiao F, Liu X, Meng M. The effect of lidocaine on early postoperative cognitive dysfunction after coronary artery bypass surgery. *Anesth Analg*. 2002;95:1134-41.
36 Mathew JP, Mackensen GB, Phillips-Bute B, Grocott HP, Glower DD, et al. Randomized, double-blinded, placebo controlled study of neuroprotection with lidocaine in cardiac surgery. *Stroke*. 2009;40:880-7.
37 Polderman KH. Induced hypothermia and fever control for prevention and treatment of neurological injuries. *Lancet*. 2008;371:1955-69.
38 Pomeranz S, Safar P, Radovsky A, Tisherman SA, Alexander H, Stezoski W. The effect of resuscitative moderate hypothermia following epidural brain compression on cerebral damage in a canine outcome model. *J Neurosurg*. 1993;79:241-51.
39 Bernard SA, Gray TW, Buist MD, Jones BM, Silvester W, et al. Treatment of comatose survivors of out-of-hospital cardiac arrest with induced hypothermia. *New Engl J Med*. 2002;346:557-63.
40 Safar PJ, Kochanek PM. Therapeutic hypothermia after cardiac arrest. *New Engl J Med*. 2002;346:612-3.
41 Saxena M, Andrews PJ, Cheng A. Modest cooling therapies (35 degrees C to 37.5 degrees C) for traumatic brain injury. *Cochrane Database Syst Rev*. 2008:CD006811.
42 Childs C. Human brain temperature: regulation, measurement and relationship with cerebral trauma: part 1. *Br J Neurosurg*. 2008;22:486-96.
43 Sacho RH, Childs C. The significance of altered temperature after traumatic brain injury: an analysis of investigations in experimental and human studies: part 2. *Br J Neurosurg*. 2008;22:497-507.
44 van Hulst RA, Lameris TW, Hasan D, Klein J, Lachmann B. Effects of cerebral air embolism on brain metabolism in pigs. *Acta Neurol Scand*. 2003;108:118-24.
45 van Hulst RA, Haitsma JJ, Lameris TW, Klein J, Lachmann B. Hyperventilation impairs brain function in acute cerebral air embolism in pigs. *Intensive Care Med*. 2004;30:944-50.
46 van Hulst RA, Lameris TW, Haitsma JJ, Klein J, Lachmann B. Brain glucose and lactate levels during ventilator-induced

hypo- and hypercapnia. *Clin Physiol Funct Imaging*. 2004;24:243-8.
47 Spiess BD, Braverman B, Woronowicz AW, Ivankovich AD. Protection from cerebral air emboli with perfluorocarbons in rabbits. *Stroke*. 1986;17:1146-9.
48 Dromsky DM, Spiess BD, Fahlman A. Treatment of decompression sickness in swine with intravenous perfluorocarbon emulsion. *Aviat Space Environ Med*. 2004;75:301-5.
49 Dainer H, Nelson J, Brass K, Montcalm-Smith E, Mahon R. Short oxygen prebreathing and intravenous perfluorocarbon emulsion reduces morbidity and mortality in a swine saturation model of decompression sickness. *J Appl Physiol*. 2007;102:1099-104.
50 Zhu J, Hullett JB, Somera L, Barbee RW, Ward KR, Berger BE, et al. Intravenous perfluorocarbon emulsion increases nitrogen washout after venous gas emboli in rabbits. *Undersea Hyperb Med*. 2007;34:7-20.

Received: 09 February 2009
Accepted: 01 March 2009

Richard E Moon, MD, FRCPC, PACP, FCCP, is Professor of Anesthesiology and Professor of Medicine, Division of Pulmonary, Allergy & Critical Care Medicine, and Medical Director, Center for Hyperbaric Medicine & Environmental Physiology
Chief, Division of General, Vascular, Transplant Anesthesia
Duke University Medical Center
Durham, NC, USA
E-mail: <*moon0002@mc.duke.edu*>

This article is based on a presentation at the SPUMS ASM, Kimbe, PNG, 2008.

Article reprinted from other sources

The effects of high altitude on relative performance of dive decompression computers [Abstract]

Buzzacott PL, Ruehle A

In this paper, dive-computer-generated no decompression limits (NDLs) in fresh water at high altitude were compared with low-altitude, single, repetitive and multilevel dives. All computer-generated high-altitude NDLs exceeded those published for the altitude dived. Computer rankings by conservatism for single dives at low altitude had negative correlations with rankings at high altitude (r = -0.81). Correlation between high-altitude, square-profile dives and low-altitude, repetitive, multilevel NDLs was significantly higher (r = 0.91, $P < 0.01$). We conclude sea-level, single-dive NDLs, such as those published in instruction manuals, are not reliable when gauging the conservatism of dive computers for use at high altitude. It is recommended that divers using dive computers for planning high-altitude dives consider computer-generated, real-time NDLs as experimental.

School of Population Health, University of Western Australia and Department of Chemistry and Biochemistry, University of Denver

Reprinted with kind permission from Buzzacott PL, Ruehle A. The effects of high altitude on relative performance of dive decompression computers. *Underwater Technology*. 2009;28(2):51-5.

Key words
Altitude, computers – diving, decompression, performance, reprinted from

Predicting performance in competitive apnoea diving. Part I: static apnoea

Erika Schagatay

Key words
Breath-hold diving, oxygen consumption, diving reflex, pulmonary function, physiology, review article

Abstract

(Schagatay E. Predicting performance in competitive apnoea diving. Part I: static apnoea. *Diving and Hyperbaric Medicine*. 2009;3(2):88-99.)
Ever since the first deep diving competitions were organized, there has been debate about when the ultimate limits of human apnoeic performance will be reached, and which factors will determine these limits. Divers have thus far surpassed all former predictions by physiologists in depth and time. The common factor for all competitive apnoea disciplines is apnoeic duration, which can be prolonged by any means that increase total gas storage or tolerance to asphyxia, or reduce metabolic rate. These main factors can be broken down further into several physiological or psychophysiological factors, which are identified in this review. Like in other sports, the main aim in competitive apnoea is to extend human performance beyond the known limits. While a beginner may extend apnoeic duration by getting closer to his or her personal limit, the elite diver can only extend the duration further by pushing the individual physiological limit further by training. In order to achieve this, it is essential to identify the performance predicting factors of apnoea sports and which factors can be affected by training, work that has only just begun. This is the first of two papers reviewing the main factors predicting performance in competitive apnoea diving, which focuses on static apnoea, while the following paper will review dynamic distance and depth disciplines. Great improvements have been made in all diving disciplines in recent years and the 10-minute barrier in resting 'static apnoea' has been broached. Despite this, current training methods and the strategies employed suggest that duration can be prolonged still further, and divers themselves suggest the ultimate limit will be 15 minutes, which appears physiologically possible, for example, with further development of techniques to reduce metabolic rate.

Introduction

During the past decade the depth, distance and duration of competitive apnoeic diving have increased at an astonishing rate. Records are set at nearly every competition and there is no sign of this development tapering off. At the time of writing, the current male world record for swimming to depth in the 'constant weight' (CWT) category is 120 metres' sea water (msw), for horizontal underwater swimming (DYN) the distance is 250 m, and duration for static underwater immersion (STA) is 10 min 12 s. The female records are not far from these, with 96 msw in CWT, 214 m in DYN and 8 min in STA, which equal the male records set only a few years ago, and appear to be improving at the same rate. Part of the explanation for these improvements is that participation in the sport is increasing and more talents are being discovered. Training methods and diving strategies have also developed immensely during the past decade. The emergence of systematic training in apnoea diving schools and communication among divers worldwide are undoubtedly important aspects in the spreading of effective training methods. But what methods and strategies can be used to further increase performance?

Different types of diving

There are two fundamentally different types of human diving: sustained, repetitive diving and single dives of maximum performance. Human apnoeic diving has likely existed since the emergence of mankind, and there are evolutionary theories that suggest swimming and diving activities among our earliest ancestors were responsible for some of the most unique human features.[1] The most natural human diving is probably making short, repeated dives within the aerobic dive limit spaced by short intervals. The goal is to spend as much time as possible per hour or day at the working depth to collect enough food. The excellent early work presented by Rahn and Yokoyama is still the best survey of typical human forage diving, which will not be discussed further.[2] Recent reviews have focused on pathology and safety issues concerning breath-hold diving.[3,4] Such issues will be largely excluded from this review, which focuses on predicting competitive apnoeic performance, where safety is provided by the organizers.

In competitive diving, where the aim is to perform one dive of maximal performance, the costs in terms of lactate accumulation and oxygen debt after the maximal dive are not important as the diver has unlimited time to recover, as long as consciousness is not compromised. Competitive diving is done for duration, distance or depth, including sub-disciplines, with or without fins.[4] These disciplines have varying key physiological features that determine individual performance. However, international competitions typically include all or several of these disciplines, and most athletes compete in all disciplines. This makes specialization difficult, requiring training for overall performance, rather than discipline-specific training. The expected influence

of different physiological factors on the ultimate limits for duration, distance and depth, requires separate discussion of these disciplines. This first of two papers reviews the factors setting the limits for static apnoea duration, that is, how long can voluntarily be spent without breathing during rest without compromising consciousness? For competitive apnoea this feature is central, as disqualification results should signs of hypoxic loss of muscle control or syncope occur.

Apnoeic duration – static apnoea

Sufficient apnoeic duration is a prerequisite for performance in all apnoea sports, expressed in its most pure form in STA, where no variations in work or depth influence the performance (Figure 1). The current STA record of over 10 minutes far exceeds the duration for record dives in the other disciplines, suggesting that the major determinant of duration is the ability to restrict metabolic rate to below typical resting levels.

Three factors determine the limits of apnoeic duration:
- total body gas storage capacity in lungs, blood and tissues
- tolerance to asphyxia
- metabolic rate.

These can be broken down into several further factors (Figure 2).

GAS STORAGE

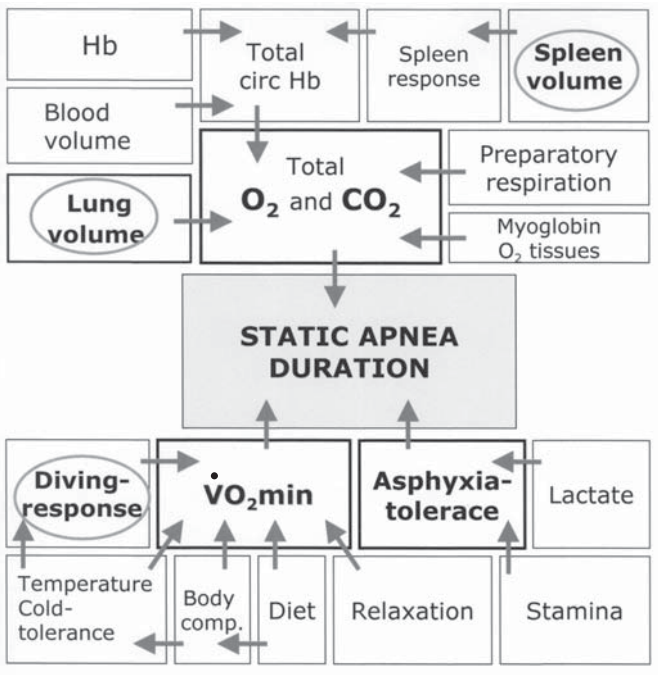
Lungs

Large lung volume has repeatedly been described as a factor distinguishing apnoea divers.[5] Benefits from having large lungs are obvious from both an apnoeic duration and depth perspective. With metabolic rate minimized, as in static apnoea, an extra litre of lung air could prolong an apnoea by up to one minute. We recently reported a mean (SD) vital capacity of 7.3 (0.9) L for 14 male elite divers, which was about two litres larger than in a control group matched for age and stature. The individual vital capacity of these divers correlated with their diving performance (total points from three disciplines) in the 2006 apnoea world championships.[6] Several divers had vital capacities of 8–9 L. This leads to the question whether expanded lung volume among divers is due to pre-selection or reflects training-induced changes.

Total lung capacity (TLC) is generally regarded as fixed in adults, but several studies suggest that specific training may increase it. Large lungs in elite divers could, aside from individual predisposition, be due to increased respiratory muscle strength, chest flexibility and or lung compliance, or, possibly training-induced lung growth. The classic study by Carey et al showed that lung volume can actually be increased reversibly by dive training.[7] Other longitudinal studies suggest an enlarging effect on the lungs by swimming and by high altitude exposure, but most likely not by other sports.[8] Stem cells are present in lung tissue, and after removal of lung lobes in children the lungs can regenerate to normal size within two years.[9,10] This suggests that lung growth may be induced in man, at least at an early age. Several elite divers, who have participated in our studies since 2003, state that their lung volume has increased since they started apnoea training (personal communications).

Figure 1
Static apnoea performance – Herbert Nitsch setting a world record of 9:04 in 2006

Figure 2
The major factors influencing performance in competitive static apnoea (apnoeic duration at rest); some factors are grouped together for reasons explained in the text; circled factors have been shown to correlate with apnoea competition performance



Breathing techniques

A frequently used method to increase lung volume just before diving is 'lung packing' or 'glossopharyngeal breathing' manoeuvres.[11] The breathing method was originally observed in paralyzed patients, and it does not involve normal respiratory muscles.[12] The normal TLC is determined by the maximal contraction of the inspiratory muscles, and the chest and lung recoil. By using the oral cavity and tongue as a pump to repeatedly press down small volumes of additional air into lungs already filled to TLC the diver can increase TLC by up to 4 L.[11,13] Apnoeic duration during rest will likely increase by the same number of minutes. One drawback of this manoeuvre is that the resulting increase in intrathoracic pressure will reduce venous return, resulting in syncope if the diver does not submerge in time.[14] A large inspired lung volume may also to some extent attenuate the development of the oxygen-conserving cardiovascular diving response discussed below.[15] Yet the likely net effect will be that the extra air volume will prolong apnoea, both by providing increased oxygen (O_2) storage, and by diluting the carbon dioxide (CO_2) received from the blood.

Lung packing can also be used as a training method to increase lung volume, and is often combined with specific stretching manoeuvres (personal communications from divers 2004−2008), but the resulting increase in vital capacity (VC) from a specifically-designed training programme of six weeks in non-divers was only 3%.[16] An effect of autoinflating the lungs is also that the alveolar surface will increase and the respiratory membrane will be thinner. Five minutes after lung packing, an increased TLC compared to before packing has been recorded suggesting a warm-up effect.[17] However, normal lung compliance in apnoea divers was recently reported, suggesting this may not be a major mechanism for long-term increases in TLC.[18]

Blood

Another major factor affecting total gas storage is the circulating volume of haemoglobin, which is a product of circulating blood volume and haematocrit or haemoglobin concentration [Hb]. The circulating haemoglobin will not only determine the available blood O_2 storage, but also increase the CO_2-buffering capacity, which is further enhanced when S_aO_2 is low (Haldane effect). Both factors will have a major influence on apnoeic duration.

Blood volume

Blood volume in diving mammals is greater than in other mammalian groups, accounting for 10–20% of body weight in seals and sea lions, compared to 7–8% in terrestrial mammals.[19,20] It further seems to correlate with the species' diving ability.[19] It would thus seem adaptive if human divers had greater blood volumes. Human blood volume can be increased through plasma volume expansion via heat adaptation or endurance training [21,22] Gas storage will only increase marginally unless this is accompanied by an increase in erythrocyte volume. In a study of moderately trained apnoea divers, scuba divers and triathletes, only triathletes were found to have higher Hb mass and increased blood volume.[23] Fat is much less vascular than lean tissue, and individual blood volume therefore varies with lean body mass.[24,25] There is no information regarding lean body mass in the study by Prommer et al.[23]

Haematocrit and total haemoglobin

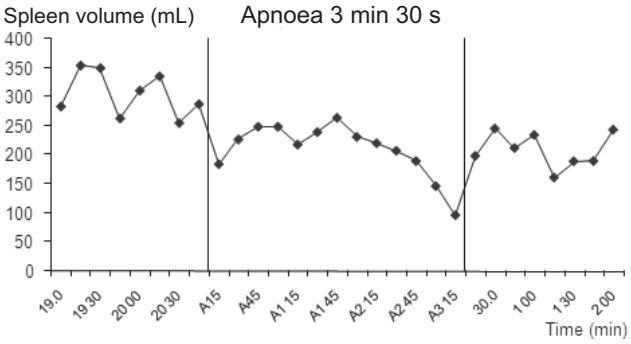
The circulating erythrocyte volume, normally slightly less than half of blood volume, can be increased by two separate mechanisms associated with apnoeic diving and altitude exposure. These two changes develop on different time scales: in the short term, by splenic contraction during apnoea or hypoxic breathing;[26,27] in the longer term, by enhanced erythropoietin production.[28–30]

Splenic contraction

The spleen of diving mammals is an extra storage site for erythrocytes when they are not needed for oxygen delivery. This supply can be ejected into the circulation during diving to temporarily enhance gas storage.[31,32] This effect is also present in mammals specialized in endurance running, e.g., horses and dogs, and also in humans during intense exercise.[33,34] The purpose of the storage is likely reduction of blood viscosity between these periods of activity. Splenic contraction in humans during apnoea diving was first observed in Ama divers.[35] The resulting Hb increase is associated with an increased apnoeic duration which is not present in splenectomized subjects.[26]

It was recently observed that the best performances in competitive apnoeic diving were associated with the largest spleens, with volumes of up to 600 ml.[36] The difference in splenic contraction between the smallest versus the largest spleens measured in the elite divers was equivalent to an increase of apnoeic duration by 30 s (unpublished observations). It is still unclear if this represents genetic diversity and pre-selection or training-induced changes, but the observed growth of a small accessory spleen after removal of the main spleen suggests a high regenerative ability.[37] The [Hb] elevation during apnoea is greater in divers than in untrained subjects and endurance athletes suggesting a training-induced promotion of the response.[38] Splenic contraction has been shown to be an active contractile process.[39] It is at least partly induced by hypoxia.[27,40] It was originally suggested to be part of the human diving response, but it now seems that it is not linked to the cardiovascular response, as the two responses are not induced by the same stimuli and occur on separate time scales.[26,41,42] Splenic contraction develops progressively across an apnoea and may need several apnoeas to develop fully (Figure 3).[41]

Figure 3
Progressive development of spleen contraction in a subject performing an apnoea at rest



Total haemoglobin

Higher baseline [Hb] in elite apnoea divers than in endurance skiers and untrained subjects suggests that either apnoea training or pre-selection favours this characteristic.[43] However, there are also reports of similar levels in divers and untrained subjects.[23] These differences could be related to the training status of the tested divers. The amount of erythrocytes in the blood is controlled via erythropoietin (EPO) produced by the hypoxic kidney.[28] While it is accepted that hypobaric hypoxia, i.e., through altitude exposure, enhances EPO production, it has only recently been shown that apnoeic episodes may also elevate EPO.[29,30] When non-divers performed 15 maximal duration apnoeas in sequence with short pauses, a mean increase in serum EPO of 24% was observed, equivalent to increases after six h at 1,800 m.[30] A follow-up study of 10 non-divers showed that 10 maximal effort apnoeas daily for two weeks also increased reticulocyte count, indicative of enhanced erythrocyte production (unpublished observations). However, [Hb] was not increased by this training, possibly due to its short duration or insufficient hypoxic exposure. When 15 apnoeas daily were performed rather than 10 for two weeks by three subjects, an increase in [Hb] of 3% resulted.

With more intense and specific apnoea training, and a diet sufficient in iron, it is likely that the Hb levels of apnoeists may increase further, leading to increased O_2 stores and increased CO_2 buffering capacity. The apnoea-related training of some elite divers reaches 20 hours per week, which by far exceeds these experimental protocols (personal communication with divers 2003–2008). The [Hb] in sleep apnoea patients appears to be correlated with the severity of the condition.[44] Apnoea diving, especially deep diving, may impose a more potent stimulus than altitude for EPO production, as there is a rapid reduction in tissue oxygen, after an initial phase of hyperoxia; it has been shown that the reduction is, in itself, an important stimulus and this occurs repeatedly during apnoea diving.[45]

Tissues

Local O_2 stores in the tissues are also important sources for aerobic metabolism. While there is a small amount of O_2 in solution in the tissues (2–3% of the total body storage), the main tissue stores are found in muscle myoglobin. In marine mammals, myoglobin concentration may be 10 times greater than in terrestrial species.[46,47] Myoglobin accounts for a considerable part of the total body O_2 stores in diving mammals and is correlated to maximum dive duration in toothed whales.[46,48] While potentially important in all apnoeic events, myoglobin content would be most important in dynamic apnoea disciplines (to be discussed in part II). It may seem less likely that myoglobin stores could be recruited for the central circulation during voluntary, conscious apnoea, if non-working areas are 'shut off' by the diving response, but depletion of myoglobin oxygen stores have been observed in sleeping elephant seal pups.[49] In addition to the ability of these hypoxia-adapted animals to withstand increased workload under hypoxic conditions, the enhanced level of myoglobin allows such animals to maintain lower critical pO_2 values, i.e., the lowest O_2 partial pressure where animals can sustain aerobic metabolism for prolonged periods of time. Thus, myoglobin is important for extending the aerobic dive limit, but the anaerobic capacity also influences diving ability in marine species.[50, 51]

Specific preparatory breathing techniques are used by divers to maximize total O_2 storage and to reduce lung, blood and slow-tissue CO_2. While O_2 is mainly stored in venous blood, CO_2 stores are predominantly in the tissues, especially in the muscles (about 10 L).[52] This muscle CO_2 storage can be mobilized with a time constant of 30 min, which is much slower than that of other tissue stores.[52] During prolonged yoga-breathing hyperventilation, often used by the divers, these CO_2 stores will be at least partially depleted, allowing a breath hold to continue for longer without excess CO_2 accumulation. The combination of increasing both the slow and major fast-equilibrating tissue storage sites may prolong apnoeic duration in divers.

TOLERANCE TO ASPHYXIA

With the presence of this enhanced O_2 supply and maximal storage capacity made available for CO_2, the next factor determining individual apnoeic duration is the tolerance to asphyxia, that is, the lowest tolerable level of hypoxia primarily for the brain without compromising consciousness, and the highest acceptable level of hypercapnia and acidosis for body functions to remain intact.

Respiratory drive and the phases of apnoea

The respiratory drive during apnoea depends on both hypoxia and hypercapnia (and acidosis; asphyxia) and their interactions, i.e., greater hypercapnia is tolerated with higher O_2 levels.[53,54] Long-term exposure to hypercapnia will reduce the ventilatory response.[55] A blunted ventilatory

response to CO_2 has been reported in submarine escape-training instructors, Ama divers and underwater hockey players.[56–58] While CO_2 is the dominant factor in inducing breathing in non-divers, the respiratory drive will be more dependent on the development of hypoxia in trained divers with a reduced ventilatory response to hypercapnia. At a given time into apnoea the P_ACO_2 is lower and the P_AO_2 higher in divers compared to non-divers, while at the end of maximal apnoeas the situation is reversed, showing that divers can tolerate higher levels of hypercapnia and more severe hypoxia.[59]

A dive or an apnoea is, according to the classical definition by Dejours, characterized by two phases, an initial "easy-going phase" without an urge to breathe, followed by a "struggle phase" where mainly the accumulating CO_2 will give rise to progressively more powerful involuntary breathing movements.[60] While the length of the first phase is determined mainly by the P_aCO_2, the duration of the second phase will also depend on psychological factors such as individual motivation and stamina. Long-term apnoea training has been shown to extend not only total apnoeic duration but also the duration of the "easy going phase".[61] The diving response is also increased after training, which may be involved in delaying the onset of the "struggle phase".[61] The 'breaking point' P_ACO_2 appears stable for a given individual at a given time despite different apnoeic conditions but can thus be varied through long-term training.[61,62]

While most inexperienced subjects interrupt the apnoea at the beginning of the struggle phase, elite apnoeists identify three phases of apnoea: the struggle phase will eventually lead to the "fighting phase", where the urge to breathe imposed by the combined stimuli of hypercapnia and hypoxia requires a strong effort not to resume breathing. During this phase the diver does not relax but uses muscle force to sustain the apnoea. The termination of apnoea often depends on learned sensory warning signs e.g., distortion of hearing or tunnel vision (personal communication with elite divers 2008). This enables the diver to interrupt the apnoea just in time to be able to perform the required 'surface protocol'. A developed psychological tolerance to the extreme discomfort during respiratory muscle contractions during the fighting phase is essential for performance in static apnoea. Part of the overall increase in apnoeic duration in divers is due to greater psychological tolerance built up through training.

Hyperventilation in various forms is used by divers to lower pre-apnoeic P_aCO_2 and extend the easy-going phase and the time before hypercapnia reaches apnoea-terminating levels.[63] However, this carries an increased risk of syncope as O_2 stores are not increased to the same extent. Some trained divers may terminate apnoea due to the hypoxic stimulus alone, while PCO_2 is still relatively low due to pre-apnoeic hyperventilation.[64] A strategy used by an increasing number of competitive divers, however, is to start the apnoea without prior hyperventilation or warm-up apnoeas, which is apparently successful and the underlying mechanisms deserve further study. Changes in lung volume and other stimuli derived from lung stretch receptors may also be involved in the integrated respiratory response to apnoeic diving, especially at depth.

Hypoxia and brain function

Several studies suggest that the minimum S_aO_2 level tolerable by the human brain may be lowered by apnoea training.[64,65] While an S_aO_2 of 50% is considered to threaten consciousness in untrained individuals, S_aO_2 levels of approximately 30 per cent have been recorded in apparently unaffected divers after maximal apnoeas, both in laboratory and field settings (unpublished observations). The diagram by Rahn and Fenn predicts how the combined effects of hypoxia and hypercapnia determine apnoeic duration,[53] and more recent data from several studies confirm that divers may resume breathing in a different range of pO_2 than non-divers.[59] During competition, divers frequently experience hypoxic loss of motor control (LMC) and sometimes syncope, but recover rapidly after assistance. While the short-term risk is obvious, the question arises as to whether such insults cause any long-term damage to brain function.

A study of extended apnoeas (mean 5 min 34 s) in divers without any signs of impaired consciousness revealed that brain damage markers increased by 37% within the first 10 min of recovery.[66] Although small compared with levels known to be associated with brain damage, this suggests there are effects on the brain which could potentially be harmful, considering the repetition of apnoeas in highly trained divers. Studies of neural function in apnoea divers, however, have revealed no long-term effects.[67] Hypoxia has been shown to prevent later ischaemic damage in experimental animal models.[68] This raises a counter-argument that apnoea training could be protective against later hypoxic events. It seems likely that frequent apnoea training involving periods of severe hypoxia may induce up-regulation of protective stress proteins.[69] Other potentially neuroprotective effects such as increased brain blood flow during apnoea have been attributed to hypercapnia and/or the diving response, as there appears to be an independent effect of face immersion.[70]

Thus, the threat of drowning due to syncope aside, there is currently no evidence that repeated syncopal events in voluntary apnoea are harmful to the human brain; it could likely be considered part of normal physiological responses to an imposed stress within the range allowed by human physiology. There is evidence that hypoxia is far less harmful than ischemia.[71] Many competitive divers probably already perform near their individual hypoxic limit, where improvements of results can only be made when training allows this limit to be moved forward. The tolerance to asphyxia was reported to be similar in amateur

and expert divers, while inferior in non-divers, suggesting that the factor may already have been exploited among most elite divers.[59]

Acidosis

The accumulation of CO_2 and lactic acid during prolonged apnoea leads to progressive acidosis.[72] One of the effects of a more powerful diving response with face immersion during apnoeas with exercise was an increased blood lactate accumulation, suggesting an increased anaerobic metabolism during these apnoeas.[73] There is also lactate accumulation during resting apnoeas, although less pronounced.[42] The diving response is known to be more powerful in divers compared to untrained individuals suggesting that divers should accumulate more lactate in underperfused tissues.[74] Interestingly, Joulia and associates obtained lower blood lactate levels in divers compared to non-divers after resting or working apnoeas and also after eupneic exercise, suggesting the presence of specific adaptive mechanisms to reduce lactate accumulation.[75]

Whether such differences were induced by the diving training is not clear, but surprisingly the non-divers had lower lactate accumulation after hand grip exercise with apnoea than without apnoea.[75] This 'lactate paradox of apnoea' deserves further study. One of the essential features for enduring prolonged apnoea may be the blood-buffering capacity for hydrogen ions from accumulating CO_2 and lactic acid. The overall CO_2 storage in divers was estimated to be twice that of non-divers.[59] The increased total Hb resulting from apnoea training noted above may increase buffering capacity, and splenic contraction will contribute to this effect, and lung-volume expansion will have a diluting effect on CO_2.

METABOLIC RATE

The third component setting the limits for apnoeic duration is metabolic rate, determining how well the 'space' between the first two factors is managed. In resting apnoeas, the main factor limiting metabolic rate may be the cardiovascular diving response.[76] When no working muscles compete with the prioritized central circulation, the selective vasoconstriction may be most efficient. Other important factors include reaching a relaxed state before the apnoea, e.g., by meditation, and the influence of thermal factors and dietary status on metabolism.[77,78] While maximum O_2 uptake is a crucial factor for performance in aerobic endurance sports, during apnoea the individual minimum O_2 uptake ($\dot{V}O_{2\ min}$) is probably one of the most important features determining performance.

The cardiovascular diving response

The diving response was first observed in humans by Irving,[79] and has later been shown to have an oxygen-conserving and dive-prolonging effect.[74,80] The response is initiated by apnoea, and transmitted either via decrease in lung stretch receptor activity or by direct contact between respiratory and cardiovascular control centres.[76] A full response is only developed with simultaneous facial chilling, mainly of the forehead and eye region.[81] The main effects are a selective vasoconstriction in areas tolerant to hypoxia, and a vagally-mediated bradycardia which develops during the initial 30 s of apnoea.[76,82] There is a corresponding reduction in cardiac output.[83] In divers, the response may involve a lowering of resting heart rate to half.[74] It does not appear to depend on hypertension as the response often develops during the transient hypotension associated with inspiration at apnoea onset (unpublished observations). It is essentially a hypoxia-prevention system, developing earlier than any signs of asphyxia.

Responsible for the oxygen-conserving effects of this 'central priority system' are the reduced oxygen consumption in underperfused areas, which may temporarily rely on anaerobic metabolism, local O_2 stores or possibly hypometabolism, and the heart rate reduction that reduces the O_2 demand of the cardiac muscle.[84] This leads to slower depletion of lung oxygen stores.[85] The diving response does not change across series of apnoeas but long-term apnoea training is known to enhance it.[61,86] Some other factors modifying the diving response development are lung volume, with a more pronounced response with low volumes such as at depth (Figure 4).[15] To some extent hypoxia will modify the response with a more pronounced bradycardia at low O_2 saturations.[87] As cold-receptors are involved in triggering the response, thermal factors during diving are important for the outcome. Diving bradycardia develops even in warm water when ambient air temperature is higher, i.e., when there is a gradient between air and water (Figure 4). However, the simultaneous chilling of other areas than the face, which normally leads to tachycardia, will not

Figure 4
Heart rate of a diver going to 68 msw in 28°C water, demonstrating that bradycardia occurs in warm water (see text); dive duration approximately 2.5 min

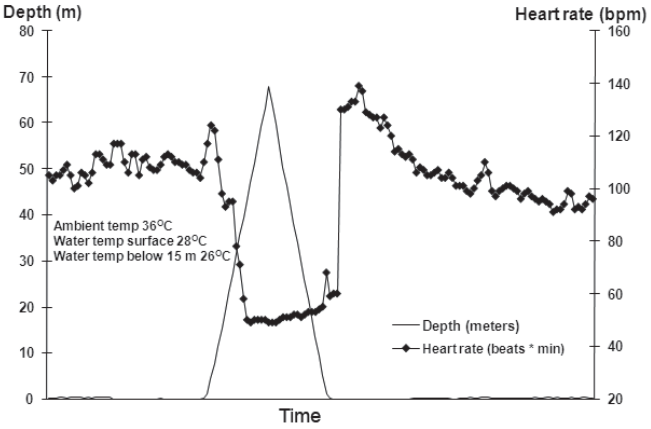
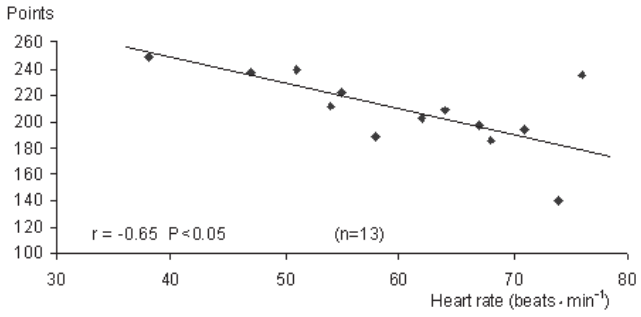


Figure 5
Correlation between apnoeic heart rate during two min apnoeas and total competition points in the world championship in apnoea in 2006



interfere with the response during apnoea.[88] Experimental two-minute apnoeas during rest in 13 male participants in the world apnoea championship in 2006 revealed an inverse correlation between apnoea heart rate and points in the competition (Figure 5).

Temperature

The diving response is dependent on both water and ambient air temperatures.[89] When apnoeas were performed in different ambient air temperatures, a standard water temperature for facial immersion evoked a diving response of varying magnitude, and within a certain temperature range, the change in temperature was the key stimulus.[89] This explains why tropical divers possess a powerful diving response despite diving in relatively warm water: as long as the air is even warmer the response will be initiated (Figure 4). When diving in cold water, the response would be expected to be attenuated in the immersed diver who is already cold before submerging fully. The peripheral vasoconstriction would be expected to be active even before diving, possibly compromising an O_2-conserving effect. However, when comparing dry-body simulated apnoeic diving with immersed dives, we recently found that the thermal stimulus during face immersion will generate a diving response powerful enough to conserve O_2 in divers even with the body immersed in cool water.[90]

The second effect of temperature on metabolism is the direct cooling effect on the body. While 'cold-blooded' animals have a metabolic rate and oxygen consumption correlated to body temperature, mammals generally respond to a threat to body temperature by increased metabolic rate, mainly through shivering thermogenesis. At water temperatures below approximately 33^{O}C, the human body will either chill down or expend energy to keep core temperature constant.[91] The muscle work associated with shivering typically increases metabolism two- to three-fold, more in thin than in fatter individuals as fat provides better insulation due to its low perfusion.[91] This shortens the apnoeic duration and may also lead to an increased superficial blood flow. However, a reduced local temperature without inducing shivering would lower tissue O_2 consumption. The responses to environmental temperature are known to vary between different populations and there are also considerable inter-individual differences. Anyone tolerating skin and core temperature reductions without shivering would likely be able to perform longer apnoeas. Regional hypothermia is a strategy contributing to the minimization of energy expenditure in marine mammals.[51]

In the 1960s, Ama divers were described as some of the most cold-adapted humans, but more recent studies reveal that the use of wetsuits has led to deacclimatization.[92] Their O_2 consumption during diving without wetsuits was clearly higher in the winter.[93] Non-shivering thermogenesis (NST) may also influence O_2 consumption during immersion and this heat-generating system increases after training, e.g., cold-water swimming.[94,95] NST allows less peripheral circulation compared to shivering, which will likely increase the efficiency of the diving response. In winter swimmers during cold water (13^{O}C) immersion, shivering started much later, heart rate was lower and the elevation of metabolic rate was slower.[95] Metabolic rate was increased by only half as much during 60 min immersion in the acclimatized swimmers, and their body temperature was lower compared to unacclimatized subjects.

Thus, better insulated individuals use less energy on shivering and cold-acclimatized individuals allow a lowering of body temperature instead of wasting energy on keeping it constant. Even repeated immersions in one day may produce some of these effects with a lowering of core temperature due to less shivering but with maintained peripheral vasoconstriction.[96] This means there could be a 'cooling-down' effect of using warm-up dives, which would likely lead to reduced metabolism and prolonged apnoeic duration. Such short- and long-term cold acclimatisation would be beneficial, and long-term changes could be one explanation as to why Scandinavian divers tend to perform well in championships, despite a shorter training season.

Thus, thermal input influences the cardiovascular responses relevant to apnoeic performance, and may have beneficial effects on metabolism during mild non-shivering cooling but with a negative effect once shivering is induced.

Anthropometrics and body composition

The surface area to mass ratio determines energy transfer between the body and the environment, and in cold water this is particularly relevant as the conductive capacity of water is approximately 25 times that of air. This means, that when large and small individuals with similar body shape are immersed, the smallest chill the fastest, and among subjects with the same weight, the one with the smaller surface area will sustain longer immersion before chilling.[97,98] With two individuals of the same weight and shape, the one with a thicker layer of subcutaneous fat will endure longer, and

a smaller person will need more subcutaneous fat to be immersed the same time without chilling.[91,97,98] One would therefore expect the ideal diver to be a tall, reasonably muscled person, as this allows for a larger blood volume, with some subcutaneous fat and thereby increased insulative capacity, and female divers to have more subcutaneous fat to compensate for being smaller.

In certain groups of marine mammals, body mass in itself has been found to vary with maximum dive duration among species.[48] The enormous variation in body size in cetaceans make size-related differences in metabolic rate and surface-to-mass ratio important, but with the limited variation in size among humans this effect is probably relatively minor.

Fasting and diet

Fasting is a strategy often used by divers in order to enhance apnoeic performance, and fasting during extended periods is observed in marine mammals without any inhibiting effect on physiological performance.[99] During fasting, the body relies mainly on fat metabolism, which requires 8% more O_2 per unit energy produced than during purely carbohydrate metabolism. Therefore, if only taking this factor into account, a fasting diver will likely produce shorter apnoeas than a fed one. However, fasting also involves decreased CO_2 production (by 30%) which will postpone the respiratory stimulus.[63] In inexperienced divers, this could prolong apnoeas but lead to an increased risk due to a later warning signal to end the apnoea.[78] In experienced divers, the P_aCO_2 at a given time is largely determined by pre-apnoeic respiration, and effects of hypoxia may be more important for ending the apnoea. Extended physical work causes the same shift towards increased fat metabolism when glycogen is depleted.[100] However, exercise is carefully avoided by the divers before competitions and fasting is preferred.

There may be several reasons why experienced divers fast during competition. Caloric restriction has been shown to reduce resting metabolic rate by up to 17%.[101,102] Thus fasting may switch the body to a starvation mode restricting energy expenditure and oxygen consumption, irrespective of the substrate, with conceivably positive net effects for apnoeic performance. With a full stomach, the diving response may be compromised by ongoing digestive processes requiring considerable circulatory resources (unpublished observations). Restricted energy supply leading to hypoglycaemia may also delay the onset of shivering thermogenesis (discussed above).[103] While the best results in STA may be produced during fasting, this factor is not likely to enhance performance much further as most divers already exploit this strategy during both training and competitive performance.

In contrast to this, some divers take carbohydrate supplements just before competitions to enhance performance, but the net effect is questionable. As in most sports, a well-composed diet with sufficient iron intake will, in the long run, most likely be relevant to achieving the best result from training but competition results could depend on dietary modifications. Strikingly, while many divers are extremely conscious of what they eat, other top divers disregard this factor altogether.

Figure 6
Diver meditating just before starting a dynamic apnoea

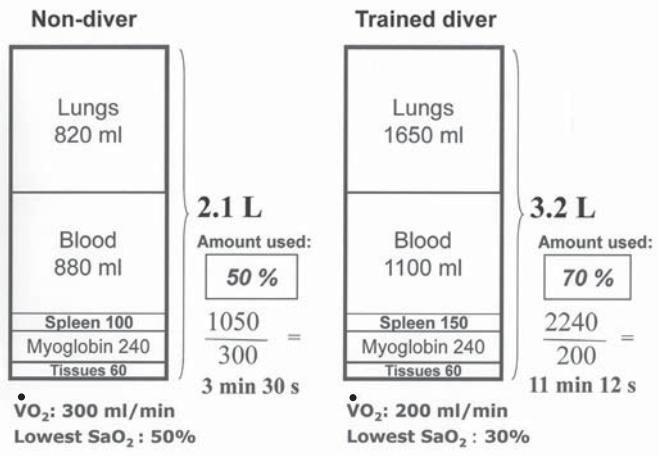
Relaxation techniques

In a sport where $\dot{V}O_{2\ min}$ is more important than $\dot{V}O_{2\ max}$, it is obvious that relaxation techniques have a significant impact on results. This is specifically evident in static apnoea performance, but used in all disciplines. Specialized relaxation borrowed from Yoga meditation, with breathing techniques adapted to apnoeic performance, is used by nearly all elite divers before competition (Figure 6). Cyclic yoga relaxation techniques have been reported to lower O_2 consumption by 32 per cent.[104] This will likely be developed further and yield longer static apnoeas. Slow yoga breathing may also affect slow-tissue gas stores, and, by lowering their CO_2 content, there will be an increased storage space made available for accumulation during apnoea.[52]

Calculations of duration of static apnoea – where are the limits?

The total O_2 store in a 70 kg person with TLC of 5.5 L, was calculated by Rahn to be 1,996 ml, with 820 ml of the storage in the lungs, 880 ml in the blood, 240 ml in the myoglobin, and 56 ml in physical solution in tissues.[105] With a consumption rate of 300 $ml.min^{-1}$, and if the total amount could be consumed, this would allow for roughly 6 min 40

Figure 7
Calculations of maximal voluntary apnoeic duration in a standardized non-diver and in an elite diver with maximized gas storage and asphyxia tolerance and reduced metabolic rate. Values for divers are based on data measured in individual divers and for non-divers on values adopted from Rahn.[105]



s of apnoea. Only about half of this amount, however, can be consumed before the normal person loses consciousness, thus a more realistic figure for the allowed voluntary apnoeic duration with these O_2 stores is 3 min 20 s. Based on roughly the same figures, but adding the amount of O_2 possibly provided by a normal-sized spleen emptying by two thirds during apnoea[33] (100 ml) the total amount would be 2,100 ml and the time with 50% usage about 3 min 30 s (Figure 7). This may well describe the situation in non-divers, but obviously does not fit with the current record dive beyond 10 min or the fact that the majority of world-class competitive free divers can produce static apnoeas in the five to seven minute range.

In a hypothetical diver with a TLC after lung packing of a good 11 L (1,650 ml O_2), a blood O_2 supply of 1,100 ml due mainly to elevated [Hb] and blood volume, a spleen volume of 600 ml (150 ml O_2) and with a similar myoglobin concentration and physical solution in tissue (60 ml), the total stores would instead amount to 3,200 ml (Figure 7). With a low metabolic rate of 200 ml.min^{-1} allowed by specific relaxation techniques and a powerful diving response, and the lowest possible S_aO_2 in conscious divers set to 30%, the maximum duration would be 11 min 12 s, one minute beyond the current record (Figure 7).

These anthropometric values represent the upper range of values measured on elite divers in our laboratory (partly unpublished), but we have not identified all these factors in the same individual at any one time. It should be noted that not all factors evident in marine mammals, with the potential to increase O_2 storage, e.g., increased muscle myoglobin, have been included in the model. Also not included are the effect of lung packing on lung oxygen concentration and pressure, thus the duration is likely underestimated. Discussing the limits of static apnoea duration with elite free divers revealed that they expect the limit to be extended to around 15 minutes before record setting will level off. It seems likely that the addition of some O_2 stores, a further lowering of metabolic rate below resting levels and anaerobic metabolism in vasoconstricted areas could prolong apnoeas even further.

Conclusions

Gas storage and tolerance to asphyxia can be significantly increased in divers compared to non-divers and metabolism can be reduced by specific meditation techniques. We can explain the current record of 10 min 12 s by including values of gas storage and asphyxia tolerance measured in elite divers. Several of these factors can be enhanced by training and possibly further developed by increased discipline specialization. However myoglobin increases or tissue gas storage modifications have not been included in these calculations. Current knowledge about human apnoeic capacity is incomplete and several potentially contributing factors known from marine mammals have yet to be studied in human divers.

Acknowledgements

I wish to thank the divers for their participation in the many experiments reported here and for valuable discussions on the limiting factors of apnoeic duration. I am also indebted to the co-workers in the studies and to Dr Matt Richardson and Mr Christian Ernest for feedback on the manuscript.

References

1 Hardy A. Was man more aquatic in the past? *New Scientist*. 1960;7:642-5.
2 Rahn H, Yokoyama T, editors. *Physiology of breath-hold diving and the ama of Japan*. Washington: Natl Acad Sci, Natl Res Council; 1965. 1341. p. 227-36.
3 Lindholm P, Lundgren CEG. Review: The physiology and pathophysiology of human breath-hold diving. *J Appl Physiol*. 2009;106(1):284-92.
4 Pollock NW. Review: Breath-hold diving: performance and safety. *Diving and Hyperbaric Medicine*. 2008;38(2):79-86.
5 Ferretti G, Costa M. Review: diversity in and adaptation to breath-hold diving in humans. *Comp Biochem Physiol*. 2003;136:205-13.
6 Schagatay E, Lodin A, Richardson M. Lung volume and diving performance in elite apnoeists. (Abstract) *33rd Annual Scientific Meeting of the European Underwater and Baromedical Society*, Sharm el Sheikh, Egypt; 2007.
7 Carey CR, Schaefer KE, Alvis HJ. Effect of skin diving on lung volumes. *J Appl Physiol*. 1956;8(5):519-23.
8 Gaultier C, Crapo R. Effects of nutrition, growth hormone disturbances, training, altitude and sleep on lung volumes. *Eur Respir J*. 1997;10(12):2913-9.
9 Kotton DN, Fine A. Lung stem cells. *Cell Tissue Res*. 2008;331(1):145-56.
10 Nakajima C, Kijimoto C, Yokoyama Y, Miyakawa T, Tsuchiya

Y, et al. Longitudinal follow-up of pulmonary function after lobectomy in childhood – factors affecting lung growth. *Pediatr Surg Int*. 1998;13:341-5.
11 Wittaker LA, Irvin CG. Going to extremes of lung volume. *J Appl Physiol*. 2007;102(3):831-3.
12 Dail CW. Glossopharyngeal breathing by paralyzed patients: preliminary report. *Calif Med*. 1951;75:217-8.
13 Örnhagen H, Schagatay E, Andersson J, Bergsten E, Gustafsson P, Sandström S. Mechanisms of "buccal pumping" ("lung packing") and its pulmonary effects. *24th Annual Scientific Meetin of the European Underwater and Baromedical Society*, Stockholm, Sweden; 1998. p 80-3.
14 Andersson J, Schagatay E, Gustafsson P, Örnhagen H. Cardiovascular effects of "buccal pumping" in breath-hold divers. In: Gennser M, editor. *24th Annual Scientific Meeting of the European Underwater and Baromedical Society*. Stockholm: National Defence Research Establishment. FOA Report: FOA-B--98-00342--721--SE:103-6; 1998.
15 Andersson J, Schagatay E. Effects of lung volume and involuntary breathing movements on the human diving response. *Eur J Appl Physiol*. 1998;77:19-24.
16 Nygren-Bonnier M, Lindholm P, Markström A, Skedinger M, Mattsson E, Klefbeck B. Effects of glossopharyngeal pistoning for lung insufflation on vital capacity in healthy women. *Am J Phys Med Rehabil*. 2007;86:290-4.
17 Seccombe LM, Rogers PG, Mai N, Wong CK, Kritharides L, Jenkins CR. Features of glossopharyngeal breathing in breath-hold divers. *J Appl Physiol*. 2006;101:799-801.
18 Tetzlaff K, Scholz T, Walterspacher S, Muth CM, Metzger J, et al. Characteristics of the respiratory mechanical and muscle function of competitive breath-hold divers. *Eur J Appl Physiol*. 2008;103:469-75.
19 Costa DP, Crocker DE, Gales NJ,. Blood volume and diving ability of the New Zealand sea lion, Phocarctus hookeri. *Physiological and Biochemical Zoology*. 1998;71(2):208-13.
20 Courtice FC. The blood volume of normal animals. *J Physiol*. 1943;102:290-305.
21 Bass DE, Kleeman DR, Quinn M, Henschel A, Hegnauer AH. Mechanisms of acclimatization to heat in man. *Medicine*. 1955;34:323-80.
22 Convertino VA, Brock PJ. Keil LC, Bernauer EM, Greenleaf JE. Exercise training-induced hypervolemia: role of plasma albumin, renin, and vasopressin. *J Appl Physiol*. 1980;48(4):665-9.
23 Prommer N, Ehrman U, Schmidt W, Steinacker JM, Radermacher P, Muth CM. Total haemoglobin mass and spleen contraction: a study on competitive apnoea divers, non diving athletes and untrained control subjects. *Eur J Appl Physiol*. 2007;101:753-9.
24 Wennesland R, Brown E, Hopper J Jr, Hodges JL Jr, Guttentag OE, et al. Red cell, plasma and blood volume in healthy men measured by radiochromium (Cr51) cell tagging and hematocrit: influence of age, somatotype and habits of physical activity on the variance after regression of volumes to height and weight combined. *J Clin Invest.* 1959;38:1065-77.
25 Feldschuh J, Enson Y. Prediction of the normal blood volume: relation of blood volume to body habitus. *Circulation*. 1977;56(4):605-12.
26 Schagatay E, Andersson J, Hallén M, Pålsson B. Physiological and genomic consequences of intermittent hypoxia. Selected contribution: role of spleen emptying in prolonging apnoeas in humans. *J Appl Physiol*. 2001;90(4):1623-9.
27 Richardson M, Lodin A, Reimers J, Schagatay E. Short-term effects of normobaric hypoxia on the human spleen. *Eur J Appl Physiol*. 2008a;104(2):395-9.
28 Erslev A. Humoral regulation of red cell production. *Blood*. 1953;8(4):349-57.
29 Eckardt KU, Boutellier U, Kurtz A, Schopen M, Koller EA, Bauer C. Rate of erythropoietin formation in humans in response to acute hypobaric hypoxia *J Appl Physiol*. 1989;66(4):1785-8.
30 de Bruijn R, Richardson M, Schagatay E. Increased erythropoietin concentration after repeated apnoeas in humans. *Eur J Appl Physiol*. 2008;102(5):609-13.
31 Qvist J, Hill RD, Schneider RC, Falke KJ, Liggins GC, et al. Hemoglobin concentrations and blood gas tensions of free-diving Weddell seals. *J Appl Physiol*. 1986;61(4):1560-9.
32 Hurford WE, Hochachka PW, Schneider RC, Guyton G.P, Stanek KS, et al. Splenic contraction, catecholamine release, and blood volume redistribution during diving in the Weddell seal. *J Appl Physiol*. 1996;80(1):298-306.
33 Laub M, Hvid-Jacobsen K,.Hovind P, Kanstrup IL, Christensen NJ, Nielsen SL. Spleen emptying and venous hematocrit in humans during exercise. *J Appl Physiol*. 1993;74(3):1024-6.
34 Stewart IB, McKenzie DC. The human spleen during physiological stress. *Sports Med*. 2002;32:361-9.
35 Hurford, WE. Hong SK, Park YS, Ahn DW, Shiraki K, et al. Splenic contraction during breath-hold diving in the Korean Ama. *J Appl Physiol*. 1990;69(3):932-6.
36 Schagatay E, Richardson M, Lodin A. Spleen size and diving performance in elite apnoeists. (Abstract) *33rd Annual Scientific Meeting of the European Underwater and Baromedical Society*, Sharm el Sheikh, Egypt; 2007.
37 Voet D, Afschrift M, Nachtegaele P, Delbeke MJ, Schelstraete K, Benoit Y. Sonographic diagnosis of an accessory spleen in recurrent idiopathic thrombocytopenic purpura. *Pediatr Radiol*. 1983;13:39-41.
38 Richardson M, de Bruijn R, Holmberg HC, Björklund G, Haughey H, Schagatay E. Increase of hemoglobin concentration after maximal apnoeas in divers, skiers and untrained humans. *Canadian J Appl Physiol*. 2005;30:276-81.
39 Bakovic D, Valic Z, Eterovic D, Vukovic I, Obad, et al. Spleen volume and blood flow response to repeated breath-hold apnoeas. *J Appl Physiol*. 2003;95(4):1460-6.
40 Richardson M, deBruijn R, Schagatay E. Hypoxia augments apnoea-induced increase in hemoglobin concentration and hematocrit. *Eur J Appl Physiol*. 2008b;105(1):63-8.
41 Schagatay E, Haughey H, Reimers J. Speed of spleen volume changes evoked by serial apnoeas. *Eur J Appl Physiol*. 2005;93:447-52.
42 Schagatay E, Andersson J, Nielsen B. Hematological response and diving response during apnoea and apnoea with face immersion. *Eur J Appl Physiol*. 2007;101:125-32.
43 de Bruijn R, Richardson M, Haughey H, Holmberg HC, Björklund G, Schagatay E. Hemoglobin levels in elite divers, elite skiers and untrained humans (Abstract). *33rd Annual Scientific Meeting of the European Underwater and Baromedical Society*, Ajaccio, Corsica, France; 2004.
44 Choi JB, Loredo JS, Norman D, Mills PJ, Ancoli-Israel S, et al. Does obstructive sleep apnoea increase hematocrit? *Sleep Breath*. 2006;10(3):155-60.
45 Balestra C, Germonpre P, Poortmans JR, Marroni A. Serum erythropoietin levels in healthy humans after a short period of normobaric and hyperbaric oxygen breathing: the "normobaric oxygen paradox". *J Appl Physiol*. 2006;100(2):512-8.
46 Kooyman GL. *Zoophysiology: diverse divers*. Berlin:

Springer-Verlag; 1989.
47 Kooyman GL,Ponganis PJ. The physiological basis of diving to depth: birds and mammals. *Annu Rev Physiol*. 1998;60:19-32.
48 Noren SR, Williams TM. Body size and skeletal muscle myoglobin of cetaceans: adaptations for maximizing dive duration. *Comp Biochem Physiol A Mol Integr Physiol*. 2000;126:181-91.
49 Ponganis PJ, Kreutzer U, Sailasuta N, Knower T, Hurd R, Jue T. Detection of myoglobin desaturation in *Mirounga angustrirostris* during apnoea. *Am J Physiol Regulatory Integrative Comp Physiol*. 2002;282:267-72.
50 Costa DP, Gales NJ, Goebel ME. Aerobic dive limit: how often does it occur in nature? *Comp Biochem Physiol A Mol Integr Physiol*. 2001;129:771-83.
51 Butler PJ. Review: metabolic regulation in diving birds and mammals. *Respir Physiol Neurobiol*. 2004:141:297-315.
52 Linér MH, Linnarsson D. Tissue oxygen and carbon dioxide stores and breath-hold diving in humans. *J Appl Physiol*. 1994;77(2):542-7.
53 Rahn H, Fenn WO. *A graphical analysis of the respiratory gas exchange. The O_2-CO_2 diagram*. Washington DC, Am Physiol Society; 1955.
54 Hesser CM. Breath holding under high pressure. In: Rahn H, Yokoyama T, editors. *Physiology of breath-hold diving and the Ama of Japan*. Washington: Natl Acad Sci, Natl Res Council; 1965. p. 165-81.
55 Schaefer KE, Hastings BJ, Carey CR, Nichols JR. Respiratory acclimatization to carbon dioxide. *J Appl Physiol*. 1963;18(6):1071-8.
56 Schaefer KE. Adaptation to breath-hold diving. In: Rahn H, Yokoyama T, editors. *Physiology of breath-hold diving and the Ama of Japan*. Washington: Natl Acad Sci, Natl Res Council; 1965. p. 237-52.
57 Masuda Y, Yoshida A, Hayashi F, Sasaki K, Honda Y. The ventilatory responses to hypoxia and hypercapnia in the Ama. *Jap J Physiol*. 1981;31:187-97.
58 Davis FM, Graves M, Guy H, Prisk GK, Tanner TE. Carbon dioxide responses and breath-hold times in underwater hockey players. *Undersea Biomed Research* 1987;14(6):527-34.
59 Ferretti G. Review: Extreme human breath-hold diving. *Eur J Appl Physiol*. 2001;84:254-71.
60 Dejours P. Hazards of hypoxia during diving. In: Rahn H, Yokoyama T, editors. *Physiology of breath-hold diving and the Ama of Japan*. Washington: Natl Acad Sci, Natl Res Council; 1965. p. 183-93.
61 Schagatay E, van Kampen M, Emanuelsson S, Holm B. Effects of physical- and apnoea training on apnoeic time and diving response in humans. *Eur J Appl Physiol*. 2000;82:161-9.
62 Andersson J, Schagatay E. Effects of lung volume and involuntary breathing movements on the human diving response. *Eur J Appl Physiol*. 1998;77:19-24.
63 Lin YC, Lally DA, Moore TA, Hong SK. Physiological and conventional breath-hold break points. *J Appl Physiol*. 1974;37(3):291-6.
64 Lindholm P, Lundgren CEG. Alveolar gas composition before and after maximal breath-holds in competitive divers. *Undersea Hyperb Med*. 2006;33:463-7.
65 Ferretti G, Costa M, Ferrigno M, Grassi B, Marconi C, Lundgren CEG. Alveolar gas composition and exchange during deep breath-hold diving and dry breath-holds in elite divers. *J Appl Physiol*. 1991:70:794-802.
66 Andersson JPA, MH Liner, H Jonsson. Increased levels of the brain damage marker S100B after apnoeas in competitive breath-hold divers. In: Lindholm P, Pollock NW, Lundgren CEG, editors. *Breath-hold diving*. Proceedings of the Undersea and Hyperbaric Medical Society/Divers Alert Network 2006 Workshop. Durham, NC: Divers Alert Network; 2006.
67 Ridgway L, Mc Farland K. Apnoea diving: long term neurocognitive sequelae of repeated hypoxemia. *Clin Neuropsych*. 2006;20:160-76.
68 Gidday JM, Fitzgibbons JC, Shah AR, Park TS. Neuroprotection from ischemic brain injury by hypoxic preconditioning in the neonatal rat. *Neurosci Lett*. 1994;168:221-4.
69 Tanaka S, Uehara T, Nomura Y. Up-regulation of protein-disulfide isomerise in response to hypoxia/brain ischemia and its protective effect against apoptotic cell death. *J Biol Chem*. 2000;275(14):10388-93.
70 Palada I, Obad A, Bakovic D, Valic Z, Ivancev V, Dujic Z. Cerebral and peripheral hemodynamics and oxygenation during maximal dry breath-holds. *Respir Physiol Neurobiol*. 2007;157:374-81.
71 Miyamoto O, Auer RN. Hypoxia, hyperoxia, ischemia, and brain necrosis. *Neurology*. 2000;54:362.
72 Olsen CR, Fanestil DD, Scholander PF. Some effects of apnoeic underwater diving on blood gases, lactate and pressure in man. *J Appl Physiol*. 1962;17:938-42.
73 Andersson JPA, Linér MH, Fredsted A, Schagatay E. Cardiovascular and respiratory responses to apnoeas with and without face immersion in exercising humans. *J Appl Physiol*. 2004;96(3):1005-10.
74 Schagatay E, J Andersson. Diving response and apnoeic time in humans. *Undersea Hyperb Med*. 1998;25:13-9.
75 Joulia F, Steinberg JG, Wolff F, Gavarry O, Jammes Y. Reduced oxidative stress and blood lactic acidosis in trained breath-hold human divers. *Resp Physiol Neurobiol*. 2002;133:121-30.
76 Elsner R, Gooden B. *Diving and asphyxia: a comparative study of animals and man*. Physiological Society Monograph 40. Cambridge: Cambridge Univ Press; 1983. p. 1-175.
77 Schagatay E, Holm B. Effects of water and ambient air temperatures on human diving bradycardia. *Eur J Appl Physiol*. 1996;73:1-6.
78 Lindholm P, Conniff M, Gennser M, Pendergast D, Lundgren C. Effects of fasting and carbohydrate consumption on voluntary resting apnoea duration. *Eur J Appl Physiol*. 2007;100:417-25.
79 Irving L. Bradycardia in human divers. *J Appl Physiol*. 1963:18:489-91.
80 Andersson J, Schagatay E. Arterial oxygen desaturation during apnoea in humans. *Undersea Hyperb Med*. 1998;25(1):21-5.
81 Schuitema KE, Holm B. The role of different facial areas in eliciting human diving bradycardia. *Acta Physiol Scand*. 1988;132:119-20.
82 Jung K, Stolle W. Behaviour of heart rate and incidence of arrhythmia in swimming and diving. *Biotelem Patient Monit*. 1981;8:228-39.
83 Ferrigno M, Hickey DD, Linér MH, Lundgren CEG. Cardiac performance in humans during breath holding. *J Appl Physiol*. 1986;60(6):1871-7.
84 Lin YC. Breath-hold diving in terrestrial mammals. *Exercise Sport Sci Rev*. 1982;10:270-307.
85 Andersson J, Biasoletto-Tjällström G, Schagatay E. Pulmonary gas exchange is reduced by the cardiovascular diving response in resting humans. *Resp Physiol Neurobiol*. 2008;160:320-4.

86 Schagatay E, van Kampen M, Andersson J. Effects of repeated apnoeas on apnoeic time and diving response in non-divers. *Undersea Hyperb Med*. 1999;26:143-9.
87 Gooden BA. Mechanism of the human diving response. *Integr Psychol Behav Sci*, 1994:29(1):6-16.
88 Andersson J, Schagatay E, Gislén A, Holm B. Cardiovascular responses to cold water immersions of the forearm and face, and their relationship to apnoea. *Eur J Appl Physiol*. 2000;83:566-72.
89 Schagatay E, Holm B. Effects of water and ambient air temperatures on human diving bradycardia. *Eur J Appl Physiol*. 1996;73:1-6.
90 deBruijn R, Richardson MP, Schagatay E. The function of the diving response in the immersed human. *Diving and Hyperbaric Medicine*. 2009;39; (in press).
91 Cannon P, Keatinge WR. The metabolic rate and heat loss of fat and thin men in heat balance in cold and warm water. *J Physiol*. 1960;154(2):329-44.
92 Park YS, Rennie DW, Lee IS, Park YD, Paik KS, et al. Time course of deacclimatization to cold water immersion in Korean women divers. *J Appl Physiol*. 1983;54(6):1708-16.
93 Kang DH, Kim PK, Kang BS, Song SH, Hong SK. Energy metabolism and body temperature of the Ama. *J Appl Physiol*. 1965;20(1):46-50.
94 Jansky L. Non-shivering thermogenesis and its thermoregulatory significance. *Biol reviews*. 1972;48:85-132.
95 Vybíral S, Lesná I, Jansky L, Zeman V. Thermoregulation in winter swimmers and physiological significance of human catecholamine thermogenesis. *Experimental Physiol*. 2000;85(3):321-6.
96 Castellani JW, Young AJ, Sawka MN, Pandolf KB. Human thermoregulatory responses during serial cold-water immersions. *J Appl Physiol*. 1998;85(1):204-9.
97 McArdle WD, Magel JR, Gergley TJ, Spina RJ, Toner MM. Thermal adjustment to cold-water exposure in resting men and women. *J Appl Physiol*. 1984;56(6):1565-71.
98 Tikuisis P, Jacobs I, Moroz D, Vallerand AL, Martineau L. Comparison of thermoregulatory responses between men and women immersed in cold water. *J Appl Physiol*. 2000;89(4):1403-11.
99 Yochem PK, Stewart BS, Mazet JA, Boyce WM. Hematological and serum biochemical profile of the northern elephant seal (*Mirounga angustrirostris*) variation with age, sex, and season. *J Wildl Dis*. 2008;44(4):911-21.
100 Lindholm P, Gennser M. Aggravated hypoxia during breath-holds after prolonged exercise. *Eur J Appl Physiol*. 2005;93(5-6):701-7.
101 Connolly J, Romano T, Patruno M. Effects of dieting and exercise on resting metabolic rate and implications for weight managements. *Family Practice*. 1999;16(2):196-201.
102 Apfelbaum M, Bostsarron J, Lacatis D. Effect of caloric restriction and excessive caloric intake on energy expenditure. *Am J Clin Nutr*. 1971;24(12):1405-9.
103 Passias TC, Meneilly GS, Mekjavic IB. Effect of hypoglycaemia on thermoregulatory responses. *J Appl Physiol*. 1996;80(3):1021-32.
104 Telles S, Reddy SK, Nagendra HR. Oxygen consumption and respiration following two yoga relaxation techniques. *Appl Psychophysiol Biofeedback*. 2000;25:221-7.
105 Rahn H. Oxygen stores of man. In: Dickens F, Neil E, editors. *Oxygen in the animal organism*. New York: Pergamon Press Macmillan; 1964. p. 609-18.

Received: 05 April 2009
Accepted: 08 May 2009

Erika Schagatay, PhD, is Professor in the Environmental Physiology Group, Department of Engineering and Sustainable Development, and at the Swedish Winter Sports Research Centre, Mid Sweden University, Östersund, Sweden

Address for correspondence:
Environmental Physiology Group
Department of Engineering and Sustainable Development
Akademigatan 1, Mid Sweden University
83125 Östersund, Sweden
Phone: +46-(0)63-165512
Fax: +46-(0)63-165700
E-mail: <Erika.Schagatay@miun.se>

The world as it is

Problems of a preclinical treatment algorithm for diving accidents: analysis of the Swiss hyperbaric situation

Jürg Wendling, Peter Nussberger and Christian Wölfel

Key words
Diving accidents, decompression illness, first aid, hyperbaric facilities, policy

Abstract

(Wendling J, Nussberger P, Wölfel C. Problems of a preclinical treatment algorithm for diving accidents: analysis of the Swiss hyperbaric situation. *Diving and Hyperbaric Medicine*. 2009;39(2):100-3.)
Following recent changes in the medical infrastructure in Switzerland, the number of hyperbaric facilities fell from ten to two, and the existing algorithms for telemedical assistance of divers required review. A retrospective study of decompression illness (DCI) from 2004 to 2008 showed that many divers were asymptomatic at the start of hyperbaric treatment. Because of this and recent publications, we may need to modify in the future the decision to recompress a diver. The descriptive nomenclature for DCI was used to determine the probability that symptoms represent DCI. Secondly we evaluated the probability that symptoms may be due to other pathologies or of psychosomatic origin, and thirdly the potential of symptoms for sequelae and their degree of invalidism was estimated. This process will result in one of three therapeutic decisions:

- An absolute indication for hospital-based hyperbaric treatment
- Immediate recompression if available or normobaric oxygen (NBO) with telemedical supervision and adequate monitoring; in exceptional situations, on-site chamber recompression or in-water recompression may be considered.
- NBO as a preventive measure for two hours and 'bends watch' for 24 h.

A prospective study should confirm evidence of good treatment practice. The key for further improvements will be to better understand ambiguous symptoms and to differentiate so-called 'mild' neurological symptoms. As a consequence of the reduction in hyperbaric facilities, longer transfer and treatment delays have resulted. Therefore, there is a need for careful evaluation of the indications for hyperbaric treatment of injured divers and also of the need to install on-site chambers for professional diving operations.

Introduction

Switzerland is a small country in the middle of Europe, full of mountains but isolated from the seashores. There are numerous natural fresh-water lakes mostly originating from the glacial period. Some of them are very deep. Most have a sediment bottom; however, many have rocky drop offs, caves or colourful underwater springs. While the 'big lakes' are at about 400 m altitude, many smaller ones in the Alps are higher, up to 2,500 m. Local diving, therefore, is generally drysuit diving with water temperatures around 4°C, and depths up to 50 metres' fresh water (mfw) are very common. Extreme deep diving (down to 180 mfw) is popular in a small but active technical diver community.

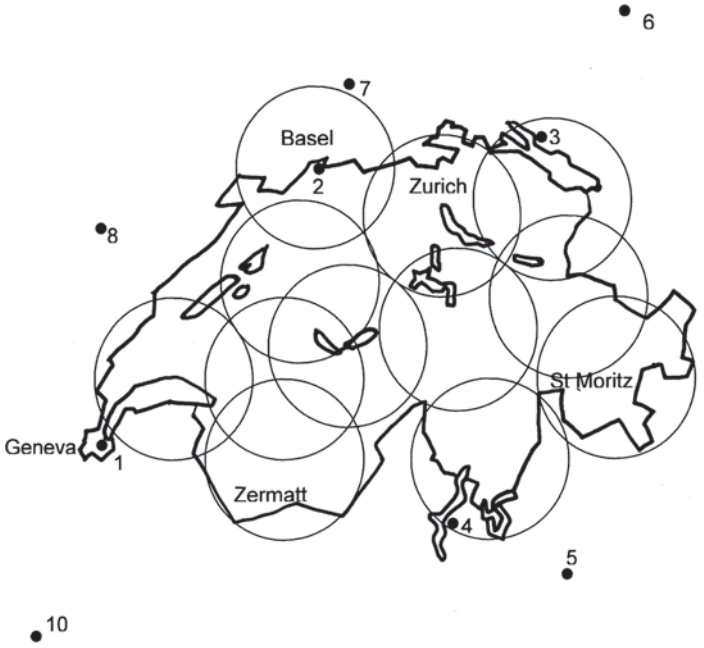
The commonest problem in emergency situations, therefore, is difficulty of access in spite of helicopter availability, particularly in bad weather conditions. The second problem is that the technical and cave divers tend to assume that they have access to a full recompression service when they call the hotline, not understanding that treatment in quasi saturation conditions needs preparations and presents logistical challenges.

Historical perspective

Switzerland, though a land-locked country, was a leader in the development of deep diving physiology and practice over many years.[1] By the 1980s a network of hyperbaric chambers had been established, reflecting an appropriate acceptance of hyperbaric medicine by the community and amongst medical specialties. Eighteen years ago, together with the establishment of the DAN Hotline for Europe in Zurich, a medical team of diving physicians started to advise divers in need and to organise and coordinate appropriate treatment: first-aid normobaric oxygen (NBO), recompression if indicated and medical follow up for up to two years after a diving incident.

The Swiss Air Ambulance, REGA, provided the telephone hotline whereby doctors were called by pager and divers were transferred rapidly by helicopter to the closest chamber. Eleven REGA helicopter bases and a few from cooperating local groups were able to get to anywhere in Switzerland within 15 minutes and to bring the patient to a recompression chamber within less than 20 minutes (Figure 1). All divers with symptoms compatible with decompression illness

Figure 1
Availability of HBOT chambers and helicopter-based medevacuation in Switzerland



Dots ● Hyperbaric treatment centres
1 Geneva (University Hospital), Switzerland
2 Basel (private), Switzerland
3 Überlingen (County Hospital), Germany, → 12km
4 Laveno-Mombello (Verbania) (private), Italy, → 16km
5 Zingonia-Bergamo (private), Italy, → 60km
6 Ulm (Military Hospital), Germany, → 106km
7 Freiburg (County Hospital), Germany, → 47km
8 Besançon, France, → 45km
9 Innsbruck (University Hospital) Austria, → 80km
10 Grenoble, France → 108km

Circle O 15 minutes around a helibase of REGA
Arrow → Distance to Swiss national border

(DCI) were brought to one of the ten hyperbaric chambers for assessment and recompression treatment (HBOT) if indicated.

Changes in Swiss health services since 2004

Treatment for DCI was paid for by a fixed daily 'hospital fee' by private health insurance (mandatory in Switzerland, controlled by Government audit) and the considerable extra costs of recompression were covered by the 'deficit guarantee' of the Canton (State of Switzerland). In 2004, new public health insurance regulations came into force; hospitals now had to be managed like a private commercial company. Due to budget restrictions, many specialist services were shut down, including hyperbaric facilities; four of five university hospital-based chambers (in Berne, Basel, Zurich and Lausanne) and four of five private chambers. The remaining two chambers still available for outpatient treatments are in Geneva and Basel. This has resulted in an increase in treatment delays of between 30 and 90 minutes and helicopters may not always be available for long-distance transfer. Also, patients have now to contribute to their costs, as the 2004 public health insurance regulations only provide a much reduced coverage for HBOT, and indications for recompression must be clearly justified. Switzerland suddenly became a 'remote' country from a diving perspective. As a result, Swiss policies for the telemedical management of diving accidents became necessary. A three-pronged approach was adopted for this.

Retrospective review of DCI cases from 2004 to 2008

Using the Divers Alert Network (DAN) Europe Suisse case reports databank, 198 possible cases of presumed DCI were identified from January 2004 to December 2008. The cases (142 men, 56 women) show a typical age distribution: nine were aged 8–14, 106 were 25–39, 77 were 40–69 and six were more than 60 years old. Severity categories and treatment are shown in Table 1. Only seven had residua (more than minor sensory impairment), all but one survived;

Table 1
Annual incidence and severity of DCI cases in Switzerland; HBOT – hyperbaric oxygen; NBO – normobaric oxygen
*** mostly not DCI after assessment**

	Symptoms				
Year	Nil	Mild	Moderate	Severe	Fatalities
2004	5	7	16	5	5
2005	4	13	11	4	4
2006	8	12	14	5	2
2007	4	23	22	4	5
2008	6	11	17	7	3
Total	27	66	80	25	19
%	14	33	40	13	10
HBOT	1	23	42	14	
NBO	20	31	33	11*	
Bends watch	6	12	5	-	

that death being from an intracerebral haemorrhage while diving, not DCI). Diving fatalities are added to the table from another source (National Statistics Board). From these cases we note some interesting findings:

- Twenty divers (10%) were asymptomatic at the start of HBOT (the main indication being omitted decompression).
- Six divers (3%) reported having experienced similar symptoms after previous dives, which resolved spontaneously.
- Twenty-nine divers (15%) had patterns of paraesthesia and sensory impairment not typical for specific spinal or cerebral lesions.

Review of international recommendations

A review of recommendations in the world literature was undertaken. Since there are no evidence-based criteria for the need for therapeutic recompression in divers with suspected DCI,[2] only 'expert opinion' could be obtained:

- US Navy diving manual: always recompress to 2.4 MPa (in-water recompression if no chamber available)[3]
- Société de Médecine et de Physiologie Subaquatiques et Hyperbares de langue française: all types of neurological symptoms may be spinal, so recompress[4]
- *Bennett and Elliott's physiology and medicine of diving*: always recompress[5]
- Comex medical book: immediate recompression in all cases[6]
- Australian recommendations: Type I DCS is self-healing; many neurological symptoms are 'peripheral'; many symptoms are of psychosomatic origin[7]
- DAN: Distinguish between mild versus severe neurological symptoms; paraesthesia and pain are the most frequent symptoms, spinal or cerebral symptoms rarer; mild symptoms (sensory, pain, rash) appear generally after one hour, while severe neurological symptoms appear within minutes (however, may appear later in some cases)[8]
- Undersea and Hyperbaric Medical Society/DAN Workshop, Sydney, 2004: in simplified summary, a clearly defined set of 'mild' symptoms behaves differently from severe symptoms, therefore allowing a flexible decision upon HBOT according to their natural prognosis, versus probability of complications due to transport and other remote conditions.[9]

Survey of hyperbaric physicians' opinions

A short questionnaire was sent to diving accident hotline experts in Barcelona, Marseille, Aberdeen, Plymouth, UK Royal Navy, Bergen, Sharm-el-Sheik and Bandos inviting their opinions on a series of scenarios and their actual practice on their diver hotline:

Scenario 1: Omitted decompression, no symptoms, delay to call 30 min – HBO versus NBO versus 'wait and see'?

- 6:1 for NBO, but 'neurocheck' necessary; duration 1–6 h, consider use of rebreather; management to be based on DOLA score.[10]

Scenario 2: Pain-only DCI; delay more than 24 h post dive – HBO versus NBO?

- 3:3 HBO, if possible, and if not more than 4 h away from a chamber

Scenario 3: mild neurological symptoms (sensory), acute – HBO versus NBO?

- 5:2 HBO (one proposal: if lower limb, give HBO – suspect spinal lesion; if upper limb, give NBO – suspect peripheral lesion or other pathology)
- when rapidly improving: 5:2 NBO
- when lasting more than 24 h: 4:3 NBO
- Atypical symptoms (unlikely DCI): 4:3 HBO if possible.

Scenario 4: Use of on-site chamber with tender and technician versus referral to clinical chamber?

- 5:2 for on-site treatment, but first call hotline; recompression on medical advice only; O_2 only (generally US Navy treatment table 6).

Scenario 5: Is an algorithm used by 'hotline' doctors?

- 6:1 No; case-by-case assessment preferred; Royal Navy uses algorithm from their diving manual.

Similar opinions were expressed by Australian, New Zealand and South African hotline doctors. It seems that transport times of up to four h for recreational diving accidents are typical worldwide and accepted as appropriate, be it only to obtain a competent, medically qualified assessment.

Discussion and conclusions

The loss of therapeutic resources in Switzerland has been considerable in recent years, but, using neighbouring countries' recompression facilities, transport times can be kept to an acceptable level (Figure 1). Greater flexibility when faced with mild symptoms compatible with DCI has been adopted. However, this needs an intensive information campaign for diving providers, training organisations and emergency department physicians in order to understand the need for external consultation and shared decision making. If the change to discretional decisions is not fully understood at all levels, divers will be placed at risk.

The Swiss medical hotline team now uses a revised guideline (Table 2) based on the above reviews and a prospective study is being conducted to ensure that the new protocols improve the overall outcome for DCI and that the reduction of indications for HBO is associated with patient satisfaction and good cost effectiveness.

The Swiss Underwater Hyperbaric Medicine Society needs to be active in providing family and hospital doctors with information about the medical indications for HBOT, not only for diving incidents. For the time-being, the approximately 40 DCI cases in recreational divers per year in Switzerland can be managed successfully with currently available resources. However, professional diving operators and tunnelling projects will need an on-site chamber and

Table 2
Guideline for Swiss diving accidents hotline 2009

1) Make working diagnosis using descriptive nomenclature for DCI
a) Classify symptoms according to severity (neurocheck via phone, differentiate light and severe neurological symptoms)
b) Type of onset (time after surfacing)
c) Type of symptom evolution (progression? Additional symptoms?)
d) Note probability and severity of barostress
e) Note gasload and probable decompression stress

2) Check probability of non-DCI origin
a) Concomitant diseases known?
b) Other immersion- or pressure-related pathologies possible?
c) Possiblity of psychosomatic symptoms (stress, social)

3) Estimate potential for progression and/or invalidating sequelae
a) Severe neurological symptoms (type II DCS), particularly medullary pathologies, progressive evolution

4) Triage decision (what treatment procedure)
a) If instability of vital functions (cardio-respiratory, unconscious) – immediate transport to next emergency medical centre (with ICU)
b) If severe type II symptoms (high potential for sequelae) medevac and recompression in hospital-based centre
c) Type I DCS with light neurological symptoms ('peripheral' type), some ambiguous signs, omitted decompression >20 min – immediate recompression in local chamber if available, otherwise transport to regional chamber. In exceptional remote situations, NBO and monitoring 24 h (telemedical supervision) may be considered.
d) Omitted decompression (no symptoms, <20 min), rapid ascent (no symptoms), pain only or skin symptoms >24 h: NBO + bends watch 24 h, repeat neurocheck.

---

medical support through a hotline to support commercial diving operations in Switzerland.

Acknowledgements

We thank DAN EUROPE and REGA, the Swiss air ambulance service, for their excellent cooperation and support over many years.

References

1 J Wendling, P Nussberger, B Schenk. Milestones of the deep diving research laboratory Zurich. *SPUMS Journal*. 1999;29(2):91-8.
2 Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness (Cochrane Review). In: *The Cochrane Library* (Issue 2, 2006). CD005007. Chichester, UK: John Wiley & Sons, Ltd; 2006.
3 *US Navy Diving Manual*, Revision 6, SS521-AG-PRO-010, Published by Direction of Commander, Naval Sea Systems Command. Flagstaff, AZ: Best Publishing Company; 2008.
4 B Brousolle, J Méliet, M Coulange. *Physiologie et médecine de la plongée*. 2nd edition. Paris: Ellipses Edition Marketing SA; 2006.
5 Moon RE, Gorman DF. Treatment of decompression disorders. In: Brubakk AO, Neumann TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th edition. Edinburgh: Saunders; 2003. p. 600-50.
6 *Comex Medical Book*, revised edition 1986. Aubagne (France): Comex SA, Imprimerie Louis Lartigot; 1986.
7 Walker R. Decompression sickness. In: Edmonds C, Lowry C, Pennefather J, Walker R, editors. *Diving and subaquatic medicine*, 4th edition. London: Hodder Arnold; 2002. p. 111-66; 414; 418-32.
8 *DAN annual diving report, 2006 edition*. Durham, NC: Divers Alert Network DAN; 2006.
9 Mitchell S, Doolette D, Wachholz C, Vann R. *Management of mild or marginal decompression illness in remote locations*. Workshop Proceedings. Durham, NC: Undersea and Hyperbaric Medical Society/DAN; 2005.
10 Desola J, Sala-Sanjaume J, Garcia A. A score index for assessment of decompression sickness risk after omitted decompression (DOLA or ADOL index). *Proceedings International Joint Meeting on Hyperbaric and Underwater Medicine*. Milan: European Underwater and Baromedical Society/International College of Hyperbaric Medicine; 1996.

Received: 31 March 2009
Accepted: 04 May 2009

Jürg Wendling, MD, is a consultant general and hand surgeon at Hospital Center Biel-Bienne, is a diving and hyperbaric medicine specialist, Director of DAN Europe Suisse, member of the European Committee for Hyperbaric Medicine, Swiss Delegate to European Diving Technology Committee (EDTC) and Chairman, Medical Subcommittee, EDTC.
Peter Nussberger, MD, is a general surgeon and Head of Department of Surgery, Riehen Hospital, Basel, member of DAN Europe Suisse Medical Team, President of Swiss Underwater and Hyperbaric Medical Society, and a past assistant to Professor A Bühlmann, Zurich.
Christian Wölfel, MD, is an anesthesiologist at Kantonsspital Luzern, Diving Medicine Physician, member of DAN Europe Suisse Medical Team, and the Training Committee of Swiss Underwater and Hyperbaric Medical Society.

Address for correspondence:
Jürg Wendling, MD
Faubourg du Lac 67, CH-2502 Bienne
Switzerland
Phone: *+41-(0)32-322-3876*
Fax: *+41-(0)32-322-3839*
E-mail: *<mail@wendling.ch>*

This paper is based on a presentation by Dr Wendling at the SPUMS ASM, Kimbe, PNG, 2008.

Opinion

Health surveillance of employed divers

Diving Medical Advisory Committee statement on health surveillance of commercial divers[1]

Personnel working in the offshore diving industry are exposed to various hazards, including pressure exposure, toxic exposures, accidents, noise, vibration and illness in remote locations. Some of these result in well-established diseases, such as decompression illness, barotraumas, noise-induced hearing loss (NIHL), dysbaric osteonecrosis, etc. It has also been demonstrated that divers are more likely to complain of symptoms, particularly musculo-skeletal pain and forgetfulness, and many demonstrate changes in lung function, although no definite disease entity had been found to explain these.

These data justify health surveillance as a risk control measure within this population. Some of these diseases have effective surveillance methods established, e.g., audiometry for NIHL. For others, methods are available but considered to carry an inappropriate risk, e.g., radiological screening for dysbaric osteonecrosis. For symptoms without a defined disease entity, questionnaire methods using techniques which can weight the importance of symptoms are appropriate.

The most critical step is to gather exposure data for individual divers covering pressure exposure, toxic chemical exposure, noise, accidents, and other incidents to assist in determining whether detected health effects are related to specific exposures.

In principle, it should be possible to establish a health surveillance programme using a combination of audiometry, lung function measurement and questionnaire methods.

Significant logistical difficulties exist, given that health status is critical for divers' employment and because many divers are self-employed (or on short-term contract) rather than being salaried employees and because it is not possible to make health surveillance mandatory. Hence it should be anticipated that divers may be reluctant to release any data indicating a health problem, particularly if that data may be accessible by their potential employers, or affect their annual medical fitness certification exercise. Furthermore, the specification of statutory annual medical examination includes some aspects of health surveillance.

In order to overcome these problems it is recommended that a health surveillance programme should be:

- conducted completely separately from annual fitness assessments, such that results cannot influence fitness decisions;
- conducted by an independent body capable of examining the health data and comparing it with exposure data;
- voluntary;
- preceded by a public relations exercise designed to reassure divers that the programme does not represent a threat to their continued diving and demonstrate the potential benefits to the diver population;
- extended to cover non-diving personnel within the offshore diving industry;
- conducted in such a way that it does not itself induce health effects.

As divers may move between different employers during a career it would be greatly advantageous if an industry-wide format for the collection of exposure and health surveillance data be agreed.

Dysbaric osteonecrosis remains the most well established long-term health effect of diving. While routine radiological screening is no longer considered appropriate the development of effective alternative methods such as MRI require further evaluation.

Footnote: The views expressed in any guidance given are of a general nature and are volunteered without recourse or responsibility upon the part of the Diving Medical Advisory Committee, its members or officers. Any person who considers that such opinions are relevant to his circumstances should immediately consult his own advisers.

Reference

1 *DMAC statement on health surveillance of commercial divers*; April 2008. <www.dmac-diving.org/guidance/DMAC-Statement-200804.pdf> (accessed 12 May 2009).

Key words

Occupational diving, occupational health, medicals – diving, health surveillance, reprinted from

---

Editorial note:

The International Marine Contractors Association (IMCA) accepts the training objectives in <http://www.dmac-diving.org/guidance/DMAC29.pdf> now being revised with the European Diving Technology Committee (EDTC) medical subcommittee.

Medical Examination of Divers – Asia-Pacific Initiative Information note D 09/07

Information note IMCA D 20/01, issued in July 2001, advised members of the requirements for doctors who undertake annual medical examination of divers, in light of the UK Health & Safety Executive (HSE) withdrawal of recognition from doctors operating outside the UK.

Subsequently, four members in Singapore agreed to jointly recognise a number of doctors to conduct diver medicals to the standard cited in IMCA D 20/01 and to be contacted with regard to any hyperbaric medical emergency. This was publicized via information note IMCA D 21/01. Diving contractors in the wider Asia-Pacific region have now agreed an updated list of jointly recognised doctors and have asked IMCA to publicise the attached note.

While IMCA is not in a position to approve or recognise doctors (for clarity, no doctor is 'IMCA approved'), it welcomes this contractor initiative and is pleased to publicise the agreement.

Editorial - health surveillance

In a recent diving magazine article, Michael Glenn, an instructor at the North Carolina (NC) Justice Academy, put his case for more extensive screening to monitor and protect employed divers' long-term health.[1] His particular concern was the apparent inability of correlating chronic or delayed illness in divers to acute and or long-term exposure to hazardous environments during their diving careers. Specific examples were anti-fouling paints containing tributylin, and the exposure of a diving team in NC to the toxic dinoflagyllate *Pfiesteria*. Whilst some exposures may present medical problems acutely as did this one, he points out that "*if the ailment does not present itself in the traditional manner you would expect, how can you link it to the initial exposure or to a specific dive itself?*".

The *Pfiesteria* issue is interesting. Dr John Ross tracked Glenn's statement regarding *Pfiesteria* exposure leading to a realistic possibility of Alzheimers back to a NC laboratory that was researching *Pfiesteria* at the time. The laboratory accidently released a *Pfiesteria*-contaminated aerosol, which was inhaled by several laboratory workers who developed various symptoms including what was described as an "*Alzheimer-like short-term memory loss*" lasting several months. The NC dive team were briefed and this information was relayed to Glenn whose article feeds this misinformation into the diving community. Dr Ross's interest here is in how workers' perception of risk can be skewed, and the level of unnecessary health anxiety that this generates.

An example of a successful occupational screening programme is given by Glenn from the US Bureau of Alcohol, Tobacco and Firearms (BATF). BATF noted that arson investigators had a higher incidence of renal cancer than the general population. The toxic fumes released at fire scenes were the suspected cause, and respiratory protection and an on-going monitoring programme were introduced to mitigate the risk. He goes on to say "as *with the BATF, the dive community has already addressed exposure issues by categorising water quality and implementing recommended standards for equipment to be utilised. Having removed the first obstacle, the second would be the adaptation of the medical screening and record keeping*".

Dr Ian Millar was invited to comment in the Journal on Glenn's article. Meanwhile, Dr Ross wrote to the Journal, and the IMCA statement came to our attention. The IMCA list excludes many diving physicians on the SPUMS Diving Doctors List, qualified to examine employed divers, and this has raised concerns within the Antipodean diving medical community. As the New Zealand model is specifically mentioned by Elliott and Millar, the Diving Medical Directorate in Auckland, responsible for a centralized database for employed divers in NZ, was also invited to comment. The 'Pisa initiative' statement was released in February and is published here to complete a range of opinions, certainly not exhaustive, from diving physicians.

Professor Gorman and colleagues are a dissenting voice in the international community on health surveillance of divers. Most importantly their perspective is one based on actual published data derived from an employed diver community within a single nation. Also, the ELTHI study from the North Sea suggests that divers within the offshore oil industry are little different from their topside colleagues in terms of long-term health issues. Clearly more research is needed on the long-term health effects of professional diving and this will require equally substantial funding support. Whether this will be forthcoming remains a debatable issue.

Readers involved in the medical clearance and health surveillance of employed divers are invited to contribute to this debate.

Reference

1 Glenn M. Screenings need a boost. *H_2Ops*. 2008;5(5):36.

Michael Davis

Key words
Occupational diving, occupational health, medicals – diving, health surveillance, editorials

Is it enough to be 'Fit to dive'?

David H Elliott and Ian L Millar

The requirement for working divers to pass annual 'fit-to-dive' medical examinations is generally agreed to be important and appropriate, although what is implemented differs around the world. Some medicals are basic and others elaborate. Requirements are usually determined by the government that has jurisdiction over the diving to be done and they typically represent the legal minimum that the diver must attain in order to work in those waters.

For divers who regularly work around the world, such as those in the offshore industry and some scientific divers, international reciprocity and a consistent medical standard for all locations would be of benefit. The industry is seeking this and also has an expectation that the doctor who provides the medical examinations should have and maintain an appropriate level of expertise. There is, however, no formal international standard to enforce this. In the South East Asian region, frustration with the lack of consistency and international reciprocity has led an informal grouping of companies to publish names of doctors it considers appropriate to conduct medical examinations on offshore industry divers.

The International Marine Contractors Association (IMCA) is a peak body for the majority of employers of divers working in the offshore industry. It has accepted the medical standards of the European Diving Technology Committee (EDTC) for use worldwide. The EDTC has undertaken substantial work towards international standardisation, including outlining the training objectives for the medical examiners of divers (Level I Med) and it has also specified the additional requirements for those who provide routine and emergency medical support for diving (Level IIa / DM Occ). Specifically these include an appropriate knowledge of occupational health and safety for the medical management of hazards to health in diving.

These worldwide objectives were reviewed recently,[1] and courses aiming to meet EDTC requirements continue to be audited by the Diving Medical Advisory Committee (DMAC), a body that has been active in the North Sea region for some 35 years. It is promising that North American, South East Asian, Australasian and South African observer members have been invited to join EDTC and that this has led to improved momentum for internationalisation.

Glenn's article from the lay diving press recognises the importance of medical screening for finding any aspect of medical, mental and physical fitness that might affect the diver's safety at depth.[2] Glenn's particular concern, however, is with conditions that present themselves later in a diving career, possibly even after its completion. The writer is an instructor at the North Carolina Justice Academy and quotes the example of cumulative exposure to biological hazards with a possibility of delayed complications from diving in the Chesapeake.

Any possibility of 'workman's compensation' requires that the original exposure and its consequences have been properly recorded. Glenn concludes that each diver should ask his diving physician to establish a baseline of screening to be added to his annual assessment and, although his example may not be widely appropriate, the objective of promoting health surveillance is good. Much has been written on the consequences to the diver of exposure to physical, chemical and biological hazards and the need for surveillance that has been highlighted by several international workshops on the long-term health effects of diving.

Health surveillance is, however, a difficult and often poorly implemented aspect of the diving medical examination. Most recreational divers never have an examination subsequent to their initial review. Even for those occupational divers who have annual reviews out of choice or requirement, the degree of long-term health trend tracking that occurs is very variable and there is no clear separation in the medical examination between those aspects that are about fitness to dive and those that evaluate long-term health effects. Much of the standard examination aims to do both, of course, but many would argue that health surveillance should be conducted independently of fitness evaluations, particularly where there may be an incentive for divers to conceal problems in case they 'fail' the diving medical.

This is the clear view of DMAC, which, in April 2008, published a *Statement on the Health Surveillance of Commercial Divers*.[3] Outside of the diving industry, well-developed health surveillance programmes have usually had either a research base or have been enabled by individual employers needing to monitor for potential effects of specific hazards. For the highly mobile diver, this will never be satisfactory. New Zealand has addressed diver mobility within that country by establishing a centralised occupational diver-fitness registry that incorporates a health monitoring element into the annual fitness re-assessment process.

There have been a number of well-run, independent diving health surveillance programmes, such as the ELTHI study,[4] but these have limited geographical and temporal coverage. Some would argue that the diver's personal medical practitioner is in the best position to be responsible for long-term health surveillance, but this is generally unsatisfactory due to lack of diving and occupational medicine knowledge and the fact that many divers do not have a regular personal physician. In many cases, the diver's medical examiner is their most regularly visited single doctor.

Although Glenn argues for each individual's diving medical examiner to undertake the health surveillance role at the request of the diver, we believe that the need for health surveillance and its content should be determined by a suitably trained diving physician (Level IIa / DM Occ) or occupational physician. As recommended by DMAC, it should be conducted separately from the diving fitness examination.

In an ideal world, each diver would be able to have their long-term health data held securely in a register with worldwide accessibility and the potential to also use data for research and new hazard identification purposes. Information technology and the internet is making this logistically possible and health registries are increasingly being established in other fields. However, there are many legal barriers around health information privacy, funding is lacking and no suitable candidate organisations are apparent to undertake the task for divers and the diving industry. It is our hope that developments in international co-operation with respect to diving medicine training and the fitness to dive certification process will, in time, also extend to improving the health surveillance process for those who enter the special environment of the underwater world.

References

1 L'Abbate A, Elliott D. The Pisa inter-university initiative for the medical and physiological support of complex and deeper diving. Concensus statement. *Diving and Hyperbaric Medicine*. 2009;39:110-1.
2 Glenn M. Screenings need a boost. *H_2Ops*. 2008;5(5):36.
3 *DMAC statement on health surveillance of commercial divers*; April 2008. <www.dmac-diving.org/guidance/DMAC-Statement-200804.pdf> (Accessed 26 April 2009).
4 Macdiarmid JI, Ross JAS, Taylor CL, Watt SJ, Adie W, et al. Co-ordinated investigation into the possible long term health effects of diving at work. Examination of the long term health impact of diving: *The ELTHI diving study.* Sudbury: HSE Books, Research Report 230; 2004. <www.hse.gov.uk/research/rrhtm/rr230.htm>

David Elliott, OBE, DPhil, FRCP, FFOM, is the Civilian Consultant in Diving Medicine to the Royal Navy, Technical Adviser to IMCA and is hoping shortly to retire completely.
Ian L Millar, MB,BS, FAFOM, DipDHM, is the Unit Director, Hyperbaric Medicine, The Alfred, Melbourne, Australia

E-mail: *<Davidelliott001@aol.com>*

Key words
Occupational diving, occupational health, medicals – diving, health surveillance, medical database, questionnaire

Diving or commuting?

John A S Ross

Underwater diving became a practical reality in the first half of the nineteenth century with improved air pumping technology and the development of a watertight diving dress. The reason for these developments was to allow men to go to work underwater. Nevertheless, the potentially drastic consequences of returning to the surface, the intricacies of human physiology in adapting to the underwater environment and, more recently, the high-pressure environment, have led us, as physicians, somewhat to forget that the diver is there, first and foremost, to do a job of work and not to just dive. This attitude has been fostered by the nature of surface-orientated diving where a relatively brief sojourn underwater is followed by the dangers of decompression and a risk of decompression illness that is repeated for each dive performed. In other words, the risks of the dive have been greater than the risk of the job undertaken.

The same consideration has also been true for compressed air workers but arguably to a lesser degree, perhaps because the shift spent at work is so much longer than is usual for a surface-orientated diver. Indeed, in one of the early medical narratives of caisson work, regarding the construction of the Forth Rail Bridge in Scotland, the first problem faced by the site doctor was a case of enteric fever caused by digging out a shoal of rotting fish trapped by the immersion of a caisson.[1]

For commercial diving, as decompression procedures have improved and with the realization of the importance of limiting the duration of exposure to depth in air diving,[2] decompression illness has become an infrequent and generally mild event for commercial divers in UK waters and elsewhere. As the risk of the dive itself decreases, perhaps we, as physicians, need to consider more the risks associated with the underwater tasks that divers undertake. While a survey of UK divers in 2002 detected a reported career-long prevalence of decompression illness of 37%, 47% of the same work-force reported having had one or more industrial accidents leading to time off work, 13% had been subject to underwater explosion, 35% had been exposed to contaminated breathing gas and 19% reported one or more drilling-mud burns.[3] Underwater explosions in diving were most commonly caused by blowback from the ignition of pockets of hydrogen and oxygen during underwater oxy-electric arc cutting and this technique has fallen into disuse after some fatal accidents.

Drilling-mud burns are a form of occupational dermatitis caused by contact with petrochemicals and their presence is an indicator of dermal contact with and, very probably, dermal uptake of the same petrochemicals.[4] These data are a reminder that oilfield divers, while diving in a contaminated environment, do not use a diving dress that reliably protects them from toxic exposure. Approximately 15% of divers had worked as a welder under pressure and more than 90% of these welders had sustained one or more episodes of major electric shock, burn, metal fume fever or eye damage. In addition, hyperbaric welding and grinding generates a potential exposure to high levels of a nanoparticulate fume with a mean aerodynamic diameter of 20–30 nm.[5]

About 50% of a random sample from this population showed some signs of noise-induced hearing loss on audiometry. A further indication that factors other than diving were important was the observation that subjective health perception in oilfield divers may be open to psychosocial influences.[6] This has implications for the overall long-term health of these workers and the management of any future health surveillance package as it is important that divers have an accurate and undistorted perception of the occupational risks that they run.

As a consequence of the questionnaire study in 2002, and the recognition by the diving industry that health surveillance might be improved, a health surveillance questionnaire is being developed for oil industry divers. In the work up for the questionnaire, six oilfield saturation divers were interviewed by an exposure scientist and a hyperbaric physician.[7] These divers annually spent 60–140 days in saturation. All divers performed shift work and were exposed to vibrating hand tools to a level that would trigger a statutory requirement for a formal health surveillance programme in the UK. One diver had recently experienced hand arm vibration syndrome. All divers reported a period of fatigue after each saturation dive that was suggestive of a shift work effect. Five divers reported regular exposure to a level of noise that impeded conversation. Four divers reported smelling petrochemicals at work and for two of these it was a significant exposure incident. Chlorine and hydrogen sulphide exposures were also reported. All divers reported mud inside their diving suit after diving and one had suffered a drilling-mud burn. All divers did underwater construction and inspection and three were hyperbaric welders.

These observations confirmed those of the year 2000 study and the health surveillance questionnaire under development will include sections on vibration, noise, chemical exposure and dermatitis in addition to just diving. Identification of problems should lead to the application of appropriate occupational hygiene measures and will improve the divers' working environment and safety. Although these observations and the health surveillance questionnaire are confined to oilfield divers, a number of these issues, such as diving in contaminated water and exposure to noise and vibration, are undoubtedly areas of concern also for the coastal and inshore diver.

For the professional diver, diving has always been just another way to go to work. In the past, the act of diving has been so dangerous as to distract from the risks and hazards of the underwater job. The use of increasingly safe diving techniques and, in the oil industry, the switch to saturation diving perhaps now mean that the hyperbaric physician can and should pay more attention to the health consequences of the divers' underwater tasks. Failure to do so would evoke the analogy of an occupational physician whose only interest is the commute to work.

References

1 Hunter J. *General features of caisson work in: Compressed air: its physiological and pathological effects*. MD Thesis, University of Edinburgh; 1887. p. 53.

2 Health and Safety Executive. *Exposure limits for air diving operations*. DVIS5 C20 1998. Sudbury: Health and Safety Executive Books; 1998. <http://www.hse.gov.uk/pubns/dvis5.htm>

3 Macdiarmid JI, Ross JAS, Taylor CL, Watt SJ, Adie W, et al. Co-ordinated investigation into the possible long term health effects of diving at work. Examination of the long term health impact of diving: *The ELTHI diving study*. Sudbury: HSE Books, Research Report 230; 2004. <www.hse.gov.uk/research/rrhtm/rr230.htm>

4 Ormerod AD, Dwyer CM, Goodfield MJ. Novel causes of contact dermatitis from offshore oil-based drilling muds. *Contact Dermatitis*. 1998;39:262-3.

5 Ross JAS, Semple S, Duffin R, Kelly F, Feldmann J, Raab A. Characterisation of fume from hyperbaric welding operations. *J Phys: Conf Ser*. 2009,151 012042 <http://iopscience.iop.org/1742-6596/151/1/012042/pdf?ejredirect=.iopscience> (Last referenced 12 May 2009)

6 Ross JAS, Macdiarmid JL, Osman LM, Watt SJ, Godden DJ, Lawson A. *Health status of professional divers and offshore oil industry workers. Occup Med*. 2007;57:254-61.

7 Ross JAS, Semple S. Designing a health surveillance questionnaire for North Sea oil and gas industry divers. *Proceedings 34th Annual Meeting of the European Underwater and Baromedical Society*, Graz; 2008. p. 74.

John A S Ross, MB ChB, PhD, FRCoA, Honorary FFOM, is Senior Lecturer in Environmental and Occupational Medicine at the University of Aberdeen Medical School.

Address for correspondence:
Liberty Safework Research Centre
Foresterhill Road
Aberdeen AB25 2ZP, United Kingdom
Phone: *+44-(0)1224-558197*
E-mail: *<j.a.ross@abdn.ac.uk>*

Key words
Occupational diving, injuries, occupational health, medicals – diving, health surveillance, medical database, questionnaire

Routine occupational dive medical examinations

Des Gorman, Chris Sames and Simon Mitchell

Surveillance of the health of working divers is justified by the nature of the work and acknowledged short- and long-term health consequences.[1,2] There is frequent confusion between what is more properly practical or physical competency testing and what is germane to the 'medical fitness' for occupational diving. In respect of the latter, a recent diving industry publication proposed "*more extensive screening and record-keeping in order to monitor and protect divers' long-term health*".[3] The suggestions included blood gas testing, examination for specific chemicals and toxins, tissue screening and the recording of baselines against which future illness could be measured in cases of employment-related compensation.

Most jurisdictions mandate an annual comprehensive medical examination of occupational divers to determine their 'fitness to dive', but for New Zealand occupational divers this comprehensive approach was shown to be of doubtful validity at even the initial evaluation.[4] A system of a five-yearly comprehensive interview and examination plus an annual health status questionnaire was consequently instituted over five years ago and has been recently evaluated.[5]

To undertake this evaluation, we examined the records of all registered occupational divers who had completed a second comprehensive medical assessment after a five-year interval. Three hundred and thirty six divers (23% of the total occupational diver population) qualified. We found that only ten (3%) had an assessment outcome of this second comprehensive review that had a career impact. One was considered permanently unfit, four were temporarily unfit, and five were issued with conditional certification. Two were identified by respiratory function testing and eight by way of their responses to the questionnaire; none was found independently by the medical interview and examination process. This poor sensitivity is not surprising in a healthy worker group and where there is both extensive pre-screening and a rigorous and demanding training process.

We conclude that five-yearly medical examinations have a low detection rate for important health problems, but may be useful for ongoing discussions of risk understanding, acceptance and mitigation. Importantly, the questionnaire system did not 'miss' any divers who had developed a critically important health problem, and detected most of those who had less important problems.

Critics of self-reporting health questionnaires claim that they depend on the honesty of the diver, but such self-reporting of health issues by workers is an integral part of health surveillance regardless of the nature of the process. Based on our anecdotal experience, when we changed the system in New Zealand, misreporting is most likely if the worker feels their livelihood is at stake and that they cannot influence the outcome other than by way of the manner in which their health is represented. That is, the veracity of health reporting is probably more related to the nature of the outcome of the process (discretionary versus prescribed approaches to determining fitness) than it is to the conduct of the evaluation (questionnaire versus history taking by interview and physical examinations and investigations). Regardless of the drivers of these behaviours, the study we have cited above shows that there is no gain in sensitivity in this context when a self-reported questionnaire is supplemented by an interview, a physical examination and conventional investigations.[5]

There is no basis in evidence or logic then to compel us to unnecessarily frequent or extensive medical examinations and investigations. For occupational divers, a so-called comprehensive medical examination without honest disclosure by the diver is unlikely to discover epilepsy, drug addiction, or psychological and other conditions that may compromise diving safety.

We endorse most of the recommendations of the DMAC statement (p. 104 this issue) regarding health surveillance of commercial divers, but we disagree that health surveillance and fitness for work evaluations should be separate. This will increase compliance costs. A critical goal of health surveillance is to enable timely intervention to prevent further illness/injury, such that our current system of concurrent collection of health surveillance data and the fitness for work assessment seems justified both pragmatically and in terms of good health and safety practice.

A robust system for data collection in regard to exposure is required as our recent study found that only 15.5% of divers responded explicitly to the question "*how many dives in the past 12 months*?". In an attempt to address the non-reporting of such information, an electronic (internet-based) form of the annual health questionnaire will be introduced in New Zealand this year. Questions such as those relating to exposure will be 'required fields'. It is possible that in the future such a system could operate internationally.

In summary, we believe that there is a need to distinguish between issues that are best addressed through practical and physical competency assessments and those that are properly elements of 'medical fitness' for occupational diving. Secondly, those elements of fitness that are assessed, need to be so from an evidence base. Thirdly, emphasis needs to be on system design that addresses the drivers of reporting behaviour and hence that encourages veracity. Simply put, an iterative and comprehensive assessment of 'medical fitness' for diving that is either evidence-free and or is low in integrity is not only expensive but also delusory.

References

1 Macdiarmid JI, Ross JAS, Taylor CL, Watt SJ, Adie W, et al. Co-ordinated investigation into the possible long term health effects of diving at work. Examination of the long term health impact of diving: *The ELTHI diving study*. Sudbury: HSE Books, Research Report 230; 2004. <www.hse.gov.uk/research/rrhtm/rr230.htm>
2 Hope A, Lund T, Elliott DH et al. *Long term health effects of diving*. An international consensus conference. Godøysund 6-10 June 1993. Bergen: John Grieg forlag A/S; 1994:387-391, cited in: Brubakk AO, Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th ed, Edinburgh: Saunders; 2003.
3 Glenn M. Screenings need a boost. H2Ops. 2008;5(5):36.
4 Greig P, Gorman D, Drewry A, Gamble G. The predictive power of initial fitness-to-dive procedures for occupational divers in New Zealand. *SPUMS Journal*. 2003;33(4):182-7.
5 Sames C, Gorman D, Mitchell S, Gamble G. The utility of regular medical examinations of occupational divers. *Internal Medicine Journal*. 2009; in press.
6 DMAC statement on health surveillance of commercial divers; April 2008. <www.dmac-diving.org/guidance/DMAC-Statement-200804.pdf> (Accessed 12 May 2009).

Professor Des Gorman, PhD, MD, FACOM, CertDHM (ANZCA), is Head of School, and
Dr Simon Mitchell, PhD, FANZCA, CertDHM (ANZCA), is Senior Lecturer in Anaesthesia in the Faculty of Medicine and Health Sciences, The University of Auckland.
Chris Sames, FRNZCGP, MMedSci, is Medical Director, The Slark Hyperbaric Medicine Unit, RNZN Hospital, Devonport, Auckland.

Address for correspondence:
Dr Chris Sames,
RNZN Hospital, HMNZS Philomel
Devonport, Auckland, New Zealand
Phone: *+64-(0)9-445-5922*
E-mail: *<CHRISTOPHER.SAMES@mil.govt.nz>*

Key words
Occupational diving, occupational health, medicals – diving, health surveillance, medical database, questionnaire

The Pisa Inter-University Initiative for the medical and physiological support of complex and deeper diving

Antonio L'Abbate and David Elliott

The Pisa Initiative is a forum established as a result of a consensus meeting of individuals ('the group') which was convened in Pisa, Italy, in February 2009 by Prof. L'Abbate of the Scuola Superiore Sant'Anna of Pisa (SSSA). This university has a Master's degree course in hyperbaric medicine that is in the English language. Significantly the course not only covers the physiological and medical aspects of recreational diving but also incorporates the training objectives required by the European Diving Technology Committee (EDTC) for more complex and deeper diving. These aspects include the needs of the international offshore diving industry and other working divers and are audited by the Diving Medical Advisory Committee (DMAC). The only other university post-graduate course audited and approved to these standards is held at Stellenbosch University, South Africa.

The group acknowledges the support that enabled the Pisa meeting and which was provided by the Scuola Superiore Sant'Anna of Pisa, the Centro Iperbarico in Ravenna, and the Centro Formazione Offshore also of Ravenna.

The group has common concerns. There is a need for succession planning for the generation of experts who have in the past undertaken research and/or provided medical and physiological support for complex and deeper occupational diving operations, in particular but not exclusively those associated with the offshore oil and gas industry. The international nature of complex and deep offshore diving and its future development has also shown the need for a greater geographic distribution of appropriate medical support worldwide.

The Pisa Initiative group has agreed to undertake further work in order to facilitate these aims, but recognises that it is not in itself either an education provider or a body with any formal status in determining standards of education or competency. The members came as individuals from universities and other organisations that historically are associated with deeper diving and its offshore development in the recent decades.

The group that met in Pisa has developed the Consensus Statement below with respect to the medical and physiological support of complex and deeper occupational diving.

CONSENSUS STATEMENT OF THE ATTENDEES

There is a need to advance the quantity, quality and availability of training and education for those personnel who may become involved in providing medical or physiological support for occupational diving involving deeper and more complex techniques.

Internationally defined standards of education and competency are useful to enable diving contractors and their employing companies and government agencies to select the most appropriate individual or group service provider and/

or the most appropriate expert in biomedicine to provide medical and/or physiological oversight, specific policy advice, emergency support or occupational health input.

The group recognises and supports the extremely valuable progress made by the EDTC, The European Committee of Hyperbaric Medicine (ECHM) and DMAC in working towards international standardisation of diving medicine training standards. There is a need, however, to extend the aims of this work to a wider multinational community in view of the worldwide nature of the diving industry and the international mobility of divers.

The group supports the EDTC medical subcommittee's proposal to define an additional level of proficiency, which sees short courses achieving the minimum standards (previously 'Level IIa') being retitled 'Diving Medicine Basic' with a next level of 'Diving Medical Advisor' which is more consistent with the level of proficiency developed by graduates of the Master's courses offered by the SSSA of Pisa and the University of Stellenbosch. Certification should include the need for appropriate audits of training quality and subsequent requirements for Continuing Professional Development.

Exposure to the practicalities of both recreational and occupational diving is recommended as a pre-requisite to attending courses in Diving Medicine at all levels. Because graduates may then become involved in the support of offshore and other deep and complex diving operations, they should obtain first-hand experience of the realities of such diving. This is best achieved by practical in-water experience using relevant occupational diving equipment preferably at a diver training facility, and enhanced by site visits to relevant diving operations. This should extend to the procedures, facilities and logistics of complex and deeper diving operations and the culture of the divers, other workers and organisations involved.

Diving contractors, oil companies, navies and other organizations that utilise diving operations, should be encouraged to continue making their facilities and operations available for site visits by all those who may become involved in providing practical medical or physiological support to occupational diving.

Specific aspects of biomedicine that the group feels require additional emphasis in many courses include these examples:

- The operational and legal differences between recreational and occupational diving
- The principles of occupational health and safety and occupational medicine with emphasis on minimising exposure to chemical, physical and biological hazards
- The complex physiology and practicalities of deeper diving, including saturation diving
- The interface between physiology and engineering as in the functional requirements for breathing apparatus and for hyperbaric evacuation.

There would be great value in sharing both historical and current case reports and incident investigations but it is acknowledged that it is essential to ensure that data security, copyright and confidentiality issues are addressed appropriately.

The Pisa Initiative group has agreed to work in support of ongoing developments worldwide in providing advanced levels of training in Biomedicine associated with diving, based upon the existing university programmes developed by the SSSA of Pisa and the University of Stellenbosch and their willingness to share their experience and elements of their course material.

It is noted that research into the medical and physiological aspects of diving does not presently have the support or breadth that it had in the past. In addition to supporting diving researchers, it would be useful to identify potential synergies and collaborative projects between those organizations that can undertake research. The group has agreed to assist in identifying future research priorities in diving medicine and physiology, in particular those aspects relevant to deeper and more complex occupational diving operations.

There is also a need to identify individuals with subspecialty expertise in the diving medicine and physiological aspects of their primary specialty, such as ENT (ORL), orthopaedics, neurology etc., who can contribute to difficult fitness to dive determinations, incident investigations, policy and research. Such individuals should be encouraged to contribute summary documents to the materials available to the diving medicine and physiology community.

ATTENDING PARTICIPANTS:
Antonio L'Abbate, Pisa, Italy
Remo Bedini, Pisa, Italy
Marco Brauzzi, Grosseto, Italy
Alessandro Bosco, Ravenna, Italy
Giuseppe De Iaco, Pisa, Italy
Pasquale Longobardi, Ravenna, Italy
David Elliott, Haslemere, England
Jack Meintjes, Cape Town, South Africa
Ian Millar, Melbourne, Australia
Stephen Watt, Aberdeen, Scotland
Jürg Wendling, Biel Bienne, Switzerland

UNABLE TO ATTEND:
Marc Borgnetta, Marseilles, France
Jan Risberg, Bergen, Norway

Footnote: The consensus views of the participants do not necessarily reflect those of their institutions.

Key words
Occupational diving, occupational health, medicals – diving, health surveillance, medical database, meetings

Book review

Physiology and medicine of hyperbaric oxygen therapy

Neuman TS, Thom SR, editors

Hard cover, 606 pages
ISBN 978-1-4160-3406-3
Philadelphia, PA: Saunders Elsevier; 2008
Price: US$179 plus postage
Copies may be ordered through Best Publishing Company, 2355 North Steves Boulevard, PO Box 30100, Flagstaff, AZ, USA 86003-0100
<www.bestpub.com>
Phone: +1-928-527-1055
Fax: +1-928-526-0370

This is the third English-language textbook dealing with hyperbaric medicine to be published in recent years, the other two being the *Handbook on hyperbaric medicine* (2006) and *Hyperbaric medicine practice*, 3rd edition (2008).[1,2] Reviews of both of these have appeared in this journal.[3,4] Comparing these tomes is obviously important, but let us deal with this new book on its intrinsic merits first.

The intention of the book, as described in the Preface is "*to try to assemble all of the important physiological information, as well as the carefully conducted clinical investigations of HBOT* [hyperbaric oxygen therapy]. *The text is meant to be a reference tool for researchers and clinicians to help them gain a better understanding of the fundamental mechanisms of HBOT; it also serves as a critical review of the indications for HBOT. It is not intended to be a simple recitation of the virtues of hyperbaric medicine but rather an academic approach to the subject.*" Given these premises, the editors have succeeded very well in their aims, and some of the basic and applied science reviews here are outstanding in bringing together difficult and complex phenomena in a logical, well-written and understandable manner. On this basis alone, this new text adds usefully to the existing literature.

The format is that of a series of invited chapters from experts, divided into five sections – history, technical aspects, physiology, indications, side effects and complications; the section on indications occupying half the book. Each chapter starts with an outline of the topics to be covered and multiple subheadings make reference back to specific items easy. There is a nicely written historical perspective by Dick Clarke to get things going, followed, in the technical section, by chapters on monoplace and multiplace chambers, physics, paediatric and critical care for HBOT. Of these chapters, the one on physics is disappointing and neither its writer nor the editors can be excused for allowing the statement "oxygen itself is flammable" to escape correction. Also in this section is an outstanding chapter by Mitchell and Bennett on clearance to dive and fitness for work, which represents the most commonsense approach to this subject that I have seen in print. The editors rightly draw specific attention to this chapter in their preface.

Whilst Weaver does an excellent job on critical care in monoplace chambers, the single paragraph on patient ventilator use in multiplace chambers is totally inadequate. What is needed here is a discussion of the underlying physical principles and problems of ventilator function under pressure. Also, the discussion of intravenous infusion pumps is out of date and no reference is made to the considerable range of existing literature on patient care equipment for multiplace chambers that particularly has appeared in this journal and the European literature. Interestingly the paper on transmural endotracheal cuff pressures using saline-filled cuffs is also not referenced.[5] In any subsequent edition, these imbalances need to be redressed by a co-author with experience in these areas.

The physiology section starts with an elegantly written chapter by Piantadosi that should help the many clinicians who have always regarded respiratory physiology as a 'black box' to come to grips with a subject that is particularly important in the hyperbaric setting. The two chapters on ischaemia-reperfusion injury and oxygen and the basic mechanisms of wound healing are equally well written. What both achieve is a sense of the whole in describing complex cellular, subcellular and biochemical interactions.

Section four consists of nine chapters on widely accepted indications for HBOT, and a tenth chapter summarizing the evidence base for other potential indications. This excellent, latter chapter by Bennett looks at many of the Cochrane reviews for which the Sydney group has largely been responsible and is essential reading. Each of the nine chapters consists of an explanation of the underlying pathophysiology and review of the laboratory research evidence. This is followed by a discussion of the clinical presentation and treatment and review of the clinical evidence for HBOT in the condition. Naturally the balance of these components varies somewhat with each indication, but this consistent, structured format lends to much easier reading and comprehension than do some past publications. Feldmeier addresses delayed radiation injury in a manner that will be familiar to many readers and there is nothing particularly new in this chapter as its publication obviously preceded some important recent papers in this field.

I was disappointed by the chapter on gas embolism, which is primarily a rehash by the senior editor of his previous chapters in diving medical texts. There should have been greater focus on iatrogenic gas embolism, the difficulties of diagnosis in the clinical setting, and a better review of the non-diving literature. Anyone who writes that air is used during laparoscopy (page 258) has clearly not stepped foot inside an operating theatre for many a day! Reproduction of chest radiographs in this chapter is uniformly poor and unhelpful. Finally, the reproduction of US Navy treatment tables 6 and 6a in both this and the following chapter on

decompression sickness is unnecessary repetition.

The chapter on decompression sickness (DCS) is well written, meeting the needs of the clinician with a general or hyperbaric rather than a diving medicine background. Remarkably the only randomised clinical trial (on non-steroidal anti-inflammatory medication) in DCS is not mentioned, nor is the 2006 Cochrane review on decompression illness.[6] I am unclear as to why return to diving after pulmonary barotrauma is discussed in this chapter rather than in the chapter on gas embolism.

Carbon monoxide poisoning is addressed in a superbly written chapter by Stephen Thom. The explanation of the underlying pathophysiology, which is much more complex than the simple concept of an hypoxic injury, is by far the best I have read, and the review of the clinical evidence for HBOT places our current understanding in useful perspective. Interestingly there is no mention of the extensive animal studies of Gorman and his group, an omission that seems surprising.

The remaining chapters in this section are all worth reading and again the consistent format of all but one (see below) makes for easy comprehension. However, some are largely a rendering of previously published work. For instance, the chapter on crush injury by Strauss adds nothing new to material already in the literature and the section on ischaemia-reperfusion injury in it unnecessarily duplicates the material in the third section. Whilst the chapter on the management of chronic wounds is well written, patient centred and has a 'best practice' approach, there is hardly a mention of HBOT and its format differs from that of the other chapters in this section. For some reason, the clinical rationale and evidence for HBOT appears in the wound-healing chapter in the physiology section. I feel this separation should be corrected in subsequent editions. The advantage of the format adopted for the other chapters on clinical indications is that each chapter provides a comprehensive one-stop summary for clinicians (e.g., vascular, plastic and reconstructive, maxillo-facial and orthopaedic surgeons) to assist them in understanding why and how HBOT might contribute in a planned manner to patient care. This is not possible for chronic wound care, for instance, in discussing the role of HBOT for a patient with a diabetic foot ulcer with a diabetologist.

The final section on side effects and complications is rather patchier in quality. There is another outstanding, must-read chapter on oxygen toxicity by James Clarke, that should be in the third section as it is predominantly focused on the underlying pathophysiology. Of the chapter on cardiovascular aspects, only one page is devoted to complications, the rest also being better suited to the physiology section. A five-page chapter on ocular complications, when half of this has already been covered in Clarke's chapter seems rather superfluous. The chapter on non-pulmonary barotrauma is useful, but the elegant drawing of the ear on page 514 has not reproduced well. The final chapter on contra-indications is basic, but perhaps is a little out of date if the statement about hypoglycaemia during HBOT in diabetics – "*there are…no experimental studies and only a few, scant clinical reports on this problem*" – is allowed to be printed and none of several useful references on this subject has been included.

Presentation is excellent throughout, with a clear, very readable font and well laid out chapters divided into many subsections. Typographical errors are few, but some do confuse meaning (e.g., page 137 "PAO_2 of 90 mm Hg…" rather than "PaO_2" and page 155 caption to Figure 8.23 refers to CO rather than CO_2). Every chapter is extensively and contemporaneously referenced. A disappointing feature is the many black and white photos, and the X-rays mentioned earlier, that are often too small and rather washed out in appearance from the printing. Fortunately many of the clinical photos are also presented in a block of colour plates in the centre of the book, though I wonder how much they contributed to my understanding. There is an adequate index.

How does this text compare with the other two books? I have not read Kindwall and Whelan, but judging by Richardson's review, this has a clinical emphasis like the previous editions, in which case the two texts are likely to complement each other. The European text ranges over more topics than Neuman and Thom, but this new text advances our understanding of the pathophysiology by comparison. All three have their merits and deserve to sit side by side on the hyperbaric physician's bookshelf. This is another valuable contribution to the growing hyperbaric literature.

References

1 Mathieu D, editor. *Handbook on hyperbaric medicine*. Dordrecht: Springer; 2006.
2 Kindwall EP, Whelan HT, editors. *Hyperbaric medicine practice*. Philadelphia PA: Saunders Elsevier; 2008.
3 Davis M. Mathieu D, editor. Handbook on hyperbaric medicine. Dordrecht: Springer; 2006. *Diving and Hyperbaric Medicine*. 2006;36(4):233-4.
4 Richardson K. Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice. Philadelphia PA: Saunders Elsevier; 2008. *Diving and Hyperbaric Medicine*. 2009;39(1):49-50.
5 Bennett MH, Isert PR, Cumming RG. Postoperative sore throat and hoarseness following tracheal intubation using air or saline to inflate the cuff – a randomized controlled trial. *Anaesth Intens Care*. 2000;28:408-13.
6 Bennett MH, Lehm JP, Mitchell SJ, Wasiak J. Recompression and adjunctive therapy for decompression illness (Cochrane Review). In: *The Cochrane Library* (Issue 2, 2006). CD005007. Chichester, UK: John Wiley & Sons, Ltd.

F Michael Davis
Editor, Diving and Hyperbaric Medicine

Key words
Hyperbaric oxygen therapy, textbook, book reviews

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates (updated October 2008)

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

1 The candidate must be medically qualified, and be a current financial member of the Society of at least two years' standing.
2 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.
3 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.
4 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.
5 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.
6 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, published in *Diving and Hyperbaric Medicine* 2008; 38(2): 117-9.
7 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.
8 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 accepted or published in other journals will 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 <http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm>) 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 2008, the SPUMS Academic Board consists of:
Associate Professor David Smart, Education Officer
Associate Professor Mike Davis
Dr Simon Mitchell.

All enquiries and applications to the Education Officer:
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

Australian and New Zealand College of Anaesthetists

Certificate in Diving and Hyperbaric Medicine

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 4 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 Diploma in Diving and Hyperbaric Medicine.

3 **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including 6 months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS
   b **and** Completion of a further 12 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 18 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA
   d **and** 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:
<www.anzca.edu.au/edutraining/DHM/index.htm>

Dr Margaret Walker, FANZCA, Cert DHM (ANZCA)
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine

---

Diving and Hyperbaric Medicine Trainees

Please be advised that examinations for the ANZCA Certificate in Diving and Hyperbaric Medicine will be held as follows:

Written Exam	Monday 9 November, 2009
Oral Exam	Friday 27 November 2009

All eligible candidates are encouraged to sit. For further information or to register, please contact Cherie Wilkinson,
Phone: +61-(0)3-8517-5312 or
E-mail: <cwilkinson@anzca.edu.au>

Certificate in Diving and Hyperbaric Medicine

Please be advised that the following recommendations were recently passed by Council.
Certificate holders will now be advised

- that certificate holders of the Diving and Hyperbaric Medicine Certificate be permitted to use this qualification as a post-nominal
- the post-nominal be "Cert DHM (ANZCA)"
- the College use this post-nominal in correspondence with these doctors
- that the post-nominal may only be used by practitioners who are up to date with their annual Certificate fee.

Janet Devlin
Administrative Officer, Facility Accreditation
ANZCA
Phone: +64-(0)3-8517-5325
Fax: +64-(0)3-9510-6786

EUBS European Underwater and Baromedical Society

35th Annual Scientific Meeting

International Conference on Diving and Hyperbaric Medicine Aberdeen, August 25 - 28, 2009

Combined with the

British Hyperbaric Association Annual Scientific Meeting

Venue:

King's College Conference Centre, University of Aberdeen

Hosts:

University of Aberdeen and Aberdeen Royal Infirmary Hyperbaric Medicine Unit

Key topics will include:

- Health technology assessment and hyperbaric oxygen therapy
- Diving research and treatment of decompression illness
- Treatment of ORN and diabetic foot

Accommodation:

Accommodation for delegates has been reserved until 10 August.
The University has its own purpose built hotel-style accommodation, King's Hall on site.
Other accommodation is further away. You can book and pay for your accommodation online at:
https://www.conferencebookings.co.uk/delegate/ACBEUBS2009

Zetterström award and travel grants for students:

More information about these two initiatives of the EUBS
are available on the conference website below.

Registration and fees:

EUBS 2009
c/o Environmental & Occupational Medicine
Liberty Safe Work Research Centre,
Foresterhill Road, Aberdeen, AB25 2ZP
Phone: +44-(0)1224-558188
Fax: +44-(0)1224-551826
E-mail: <hyperbaric@abdn.ac.uk>
Website: <www.hyperchamber.com/EUBS2009>

EUBS European Underwater and Baromedical Society

EXECUTIVE COMMITTEE (as of October 2008)

PRESIDENT
Professor Alf O Brubakk
NTNU, Dept Circulation & Imaging
N-7089 Trondheim, Norway
Phone: +47-73-598904
Fax: +47-73-597940
E-mail: <alf.o.brubakk@ntnu.no>

VICE PRESIDENT
Dr Peter Germonpré
Centre for Hyperbaric Oxygen Therapy
Military Hospital Brussels
B-1120 Brussels, Belgium
Phone: +32-2-2644868
Fax: +32-2-2644861
E-mail: <peter.germonpre@gmail.com>

IMMEDIATE PAST PRESIDENT
Dr Noemi Bitterman
Technion, Israel Institute of Technology
Technion City
Haifa 32000, Israel
Phone: +972-4-8294909
Fax: +972-4-8246631
E-mail: <noemib@tx.technion.ac.il>

PAST PRESIDENT
Dr Ramiro Cali-Corleo
Hyperbaric Unit, St Luke's Hospital
G'Mangia, Malta
Phone: +356-(0)21-234765
Fax: +356-(0)21-372484
E-mail: <irocali@daneurope.org>

HONORARY SECRETARY
Dr Joerg Schmutz
Foundation for Hyperbaric Medicine
Kleinhuningerstrasse 177
CH-4057 Basel, Switzerland
Phone: +41-61-631-3013
Fax: +41-61-631-3006
E-mail: <joerg.schmutz@hin.ch>

MEMBER AT LARGE 2008
Dr Peter Knessl
Steinechtweg 18
CH-4452 Itingen/BL
Switzerland
Phone: +41-44-716-7105
E-mail: <pknessl@bluewin.ch>

MEMBER AT LARGE 2007
Dr Phil Bryson
DDRC, The Hyperbaric Medical Centre
Tamar Science Park, Research Way
Derriford, Plymouth
Devon, PL6 8BU, United Kingdom
Phone: +44-1752-209999
Fax: +44-1752-209115
E-mail: <phil.bryson@ddrc.org>

MEMBER AT LARGE 2006
Professor Maide Cimsit
Department Underwater and Hyperbaric Medicine
Istanbul Faculty of Medicine
80620 Istanbul, Turkey
Phone: +212-5313544
E-mail: <mcimsit@istanbul.edu.tr>

HONORARY TREASURER & MEMBERSHIP SECRETARY
Ms Patricia Wooding
16 Burselm Avenue
Hainault, Ilford
Essex, IG6 3EH, United Kingdom
Phone & Fax: +44-20-85001778
E-mail: <patriciawooding@btinternet.com>

NEWSLETTER EDITOR
Dr Peter HJ Müller
Dudenhofer Str. 8C
D-67346 Speyer, Germany
Phone: +49-6232-6865866
E-mail: <pete@ejuhm.de>

The EUBS website is at
www.eubs.org
Members are encouraged to log in

Nomination for election as EUBS Executive Committee Vice-President 2009

Costantino BALESTRA

Born 13 February 1964 in Udine (Italie)
Nationality: Belgian (2nd nationality, Italian)
Married, with three children

Education

Master in sciences (Sports Sciences)
1995 PhD «*Contribution to the understanding of the neurophysiological mechanisms of fatigue in humans*." (grande distinction)
Annexed Thesis : "Scuba Diving and Disabilities: Doppler study in the field."
2004 Rehabilitation medicine (CAPAES)
2004 Second PhD thesis : *Is the cardiac foramen ovale an increased risk for dysbaric injuries?*

Diving education

DAN Oxygen First Aid Trainers Examiner (1994)
IDC Staff Instructor (PADI) (1998)
CMAS 3-star instructor, Instructor trainer IDEA; CEDIP 3-star

Professional background

Since 2001 Lecturer in the pedagogic habilitation studies (Université Libre de Bruxelles) (Pôle Universitaire de Bruxelles Wallonie
Since 2002 Professor, Haute Ecole Paul Henri Spaak (Pôle Universitaire de Bruxelles Wallonie) Director of the Environmental & Occupational Physiology Laboratory
Since 2000 Special Projects Director for Europe for the Diving Safety Lab (DAN Europe)
Since 2000 Lecturer for the Belgian Osteopathic College (applied research methods)
Since 1999 International consultant on dysbaric problems in astronauts
Since 1998 Visiting Professor, School of Medicine, University of Belgrade, for post graduate studies in baromedicine
Since 2004 Expert for the Belgian Rehabilitation Commission C.A.P.A.E.S.
Since 2004 Part-time Professor, Padua University, in Motor Sciences
Since 2004 Legal expert for national and international scuba diving problems
Since 2005 Part-time professor, Scuola Superiore Sant'Anna (Pisa)
Since 2006 Expert pour la commission Européenne pour l'évaluation des crédits à octroyer.
Since 2006 Member of the Belgian National Scientific Board for Motor Sciences

Scientific awards and distinctions

2006 Winner of the 'Spin Off' Contest in Brussels; winner with the Flomedi project on flow-mediated dilation.
2004 Zetterström Award, EUBS Annual Meeting (Ajaccio)
2002 DAN-EUBS 2002 Award: fostering hyperbaric medicine and physiology together with P.Germonpré
1996 Winner of the KINE 2000 Prize for the study of the patency of the cardiac foramen ovale in handicapped divers
1995 Meritorious Educational Excellence Award for innovation, leadership and professionalism in diving education for the disabled person (PADI Europe)
1988 Winner of the Van Goethem-Brichant Foundation Prize for the project : "*Pour une plus grande mobilité des personnes handicapées se déplaçant en chaise roulante*.

Scientific publications and activities

Author and co-author of more than 150 scientific publications
Co-author of two books:
Balestra C, Germonpre P, Marroni A, Cronje FJ. *PFO and the Diver*. Flagstaff, AZ: Best Publishing; 2007.
Balestra C, Boancheaux E, Balestra C. *Introduction à la communiCaCtion*. Bruges: Die Keure/La charte; 2008.
Contributor to several book chapters
Member of the Editorial Board, European Journal of Applied Physiology
Reviewer for : Journal of Applied Physiology; British Journal of Sports Medicine; European Journal of Applied Physiology; European Journal of Neurophysiology; International Journal of Ultrasound Medicine; Applied Physiology & Nutrition; Journal of Sport Sciences

Nominations for election as EUBS Executive Committee Member-at-Large 2009

For the term 2009–2011, the following two nominations have been accepted by the EUBS Executive Committee. Ballot forms will be circulated by mail. Please return the ballot form using the enclosed envelope.

Andreas Møllerløkken

Address: Rosenborggt 33b, N-7014 Trondheim, Norway
Phone: +47 97 68 40 36
E-mail: <andreas.mollerlokken@ntnu.no>
Date of birth: 15.05.1974
Marital status: Married, one child

Work experience:

2008	Researcher, Department of Circulation and Medical Imaging, Faculty of Medicine, NTNU
2003–2008	PhD student, Medical Technology, Department of Circulation and Medical Imaging, Faculty of Medicine, NTNU
2003	Teacher, Lesjaskog skule
2002	Teacher Education NTNU, part 2, Anna van Rijn College, The Netherlands
2001	Teacher Education NTNU, part 1, Sunnland Ungdomsskole, Trondheim
1997–2003	Axel Bruun AS, Trondheim

Education:

2003–2008	PhD Medical Technology
2001–2002	Programme for Teacher Education, NTNU
1995–2001	Cand. Scient, Biology, NTNU
1994	Officer's training school, Trandum
1993	University of Trondheim
1990–1993	College, Trondheim Cathedral school

Awards:

2007	FRIDA award.
2005	Zetterstrom Award for best poster, EUBS, Barcelona

Publications:

1 Møllerløkken A, Gutvik C, Berge V, Jørgensen A, Løset A, Brubakk AO. Recompression during decompression and effects on bubble formation in the pig. *Aviat Space Environ Med*. 2007; 78: 557-60.
2 Møllerløkken A, Gutvik C, Måsøy S-E, Brubakk AO. Where do bubbles form?. *Eur J Underwater Hyperb Med*, 2007; 8:26-7.
3 Møllerløkken A, Nossum V, Hovin W, Gennser M, Brubakk AO. Recompression with oxygen to 160 kPa eliminates vascular bubbles, but does not prevent endothelial damage. *Eur J Underwater Hyperb Med*. 2007; 8 (1&2): 11-16.
4 Løset A, Møllerløkken A, Berge V, Wisløff U, Brubakk AO. Post-dive bubble formation in rats: Effects of exercise 24 h ahead repeated 30 min before the dive. *Aviat Space Environ Med*. 2006; 77: 905-8.
5 Møllerløkken A, Berge V, Jørgensen A, Wisløff U, Brubakk AO. Effect of a short-acting NO donor on bubble formation from a saturation dive in pigs.. *J Appl Physiol*. 2006; 101: 1541-5.
6 Møllerløkken A, Gutvik C, Tunstad H. Trygt opp fra dypet. Gemini [Avis] 14.06.2006
7 Møllerløkken A, Brubakk AO, Wisløff U. The effect of hyperbaric oxygen on aerobic capacity in rat. *Eur J Underwater Hyperb Med*. 2005; 6: 5-9.

Lectures:

1 Invited lecture EUBS 2007: Exercise and diving
2 Where do bubbles form? EUBS 2007
3 Ultrasound for animal studies. Ultrasound for monitoring decompression: Theory and practice. NTNU
4 Bubblegrade vs. number of bubbles. 32nd Annual Scientific Meeting of the EUBS 2006.
5 Who am I and what do I do? Mid-Scandinavian winter meeting in extreme environmental physiology.
6 Recompression during decompression reduce bubble formation in the pig: further support for a gas phase model of decompression. 31 Annual Meeting of the European Underwater & Baromedical Society.
7 Amis 2000, our new research tool? R&D diving, December 2005.
8 In-water recompression as first aid. Dykkeseminaret 2005.
9 Pigs without bubbles. Dykkeseminaret 2004.

Poster presentations:

1 Gutvik, Christian; Møllerløkken, Andreas; Brubakk, Alf O. Difference in bubble formation using deep stops is dependent on bottom time: experimental findings and theoretical support. Dykkeseminaret; 14–15.11.2007
2 Møllerløkken, Andreas; Gutvik, Christian; Måsøy, Svein-Erik; Brubakk, Alf O. Where do bubbles form? Dykkeseminaret; 14–15.11.2007
3 Gutvik, Christian; Møllerløkken, Andreas; Brubakk, Alf O. Parameter Estimation on the Copernicus Bubble Model Using Human Doppler Data. UHMS ASM 2006.
4 Møllerløkken, Andreas; Nossum, Vibeke; Hovin, Wenche; Brubakk, Alf O. Recompression with oxygen to 160 kPa eliminates vascular gas bubbles in the pulmonary artery, but does not prevent injury to the endothelium and the cns. 31st Annual Meeting of the European Underwater & Baromedical Society 2005.
5 Møllerløkken, Andreas; Brubakk, Alf O; Lundsett, Nina. The effect of nitric oxide on vascular bubble formation in pigs. EUBS 2004.

Jean-Michel Pontier

Born 28 February 1968 in Aix-en-Provence (France)

Since 2002 Licensed physician in diving medicine with the French Armed Forces, and working at the Diving School (Ecole de Plongée) in St Mandrier.
2002-2004 St. Anne Hospital hyperbaric centre (Toulon, France). During that period, participated as on-call physician for the hyperbaric centre of the Font-Pré Hospital.
2005 Master's Degree in physiology and extreme environments.
Certified specialist in sports medicine and emergency medicine.
Member, French Society of Diving and Hyperbaric Medicine (MedSubHyp).

Author or co-author of 8 peer-reviewed scientific papers and 15 scientific conference presentations on the specific aspects of military diving (more particularly navy 'seals'), military rebreather diving (epidemiological study) and factors increasing the risks for decompression pathology (clinical study).

Participated in the experimental studies demonstrating the protective effect of physical activity on decompression bubble formation.

Currently investigating, for a Master Thesis, the mechanisms of platelet activation during decompression, using an animal model of decompression sickness.

2005 served as on-board physician for the Clipperton Atoll expedition, with Dr J-L Etienne. For this mission, an in-water recompression protocol for treating decompression disorders in remote areas was developed.

Open letter from the EUBS ExCom

Dear EUBS member,

Over the past years, the Executive Committee of your Society has made efforts to modernise and improve EUBS's membership services. One of the most visible changes has been the merger of our Journal, EJUHM, into the SPUMS Journal *Diving and Hyperbaric Medicine* to become a joint publication between the two societies. We have also updated our website, offering new functions and 'members only' benefits.

However, the financial costs of these improvements are weighing hard on the Society. This is a temporary situation, due to the fact that last year, membership fees did not go up as they needed to, despite the increased costs. Our bank accounts need your input now. We would therefore urge you to pay your membership fees for the next membership year (July 1st 2009 - June 30th 2010) immediately.

You can log on to your membership page on the EUBS website: <www.eubs.org> and make this payment securely online using the PayPal system. You will receive a receipt immediately and a confirmation from the Society.

To log in, use your e-mail address (the one we sent this message to) and your password. In case you have forgotten your password, click the "I have forgotten my password" link, fill in your e-mail address and your password will be mailed to you automatically within seconds. If you experience any difficulties, please send a quick message to <webmaster@eubs.org> and we will try to sort out the problem as soon as possible.

Your Society needs you now! Thank you for responding promptly.

Alf O. Brubakk, President
Peter Germonpré, Vice-President
Maide Cimsit, Phil Bryson, Peter Knessl, Members at Large
Noemi Bitterman, Iro Cali-Corleo, Past and Immediate Past Presidents
Peter Mueller, European Editor, DHM
Joerg Schmutz, Secretary
Patricia Wooding, Membership Secretary

A message from the EUBS 2009 Annual Meeting conveners

The EUBS 2009 Annual Meeting will be held in King's College, University of Aberdeen, Scotland 25–28 August 2009. Further details, including a provisional programme and how to book are on the website:
<http://www.hyperchamber.com/EUBS2009/>

Abstracts should be submitted as soon as possible.
The early booking deadline is the end of June.
Accommodation can be difficult in Aberdeen and rooms are being held until 10th August.
Come and join us in the Granite City!

EUBS Annual Scientific Meeting 2010
Preliminary Announcement

Dates: 14–18 September 2010
Earlier dates were not possible because of Ramadan (6 August–7 September) followed by a religious feast.
Venue: The Marmara Hotel, Istanbul, Turkey

The Registration Desk opens from 14 September 2009.

Preliminary enquiries to:
Prof. Maide Cimsit, Istanbul University
Secretary General, EUBS ASM 2010
E-mail: <mcimsit@istanbul.edu.tr>
or
Ms. Mine Cuca, Figur Congress
Ayazmaderesi Cd. Karadut Sk. No: 7
34394 Dikilitas, Istanbul, Turkiye
Phone: + 90 212 381 46 88
Fax:+ 90 212 258 60 78
Gsm: +90 533 955 34 81
E-mail: minecuca@figur.net

Royal New Zealand Navy Hospital
Introductory Course in Diving Medicine

Dates: 11–14 September 2009
Venue: RNZN Hospital, HMNZS Philomel, Devonport, Auckland
Cost: NZ$850

This course is designed for GPs with an interest in diving medicine. Emphasis is on the assessment of fitness to dive. Those who successfully complete the course can register with the NZ Department of Labour as a Designated Diving Doctor and conduct occupational diving medicals.

The course is registered with RNZCGP for 22 hours of CME credits.

Further details and registration form available at:
<www.navyhyperbaric.mil.nz>
or contact Dr Chris Sames
Phone: +64-(0)9-445-5922

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

The poetry doctor

Oh, Siren Narcosis

Oh, Siren of the Seven Seas,
Seductress of the deep,
Calling me to come to you
And in your arms to sleep.

As I part the surface waves
And sink towards your door,
I'm thunderstruck by your beauty great.
I'm rapt in wondrous awe.

You steer me true by your sweet song
That lures me down to you,
But your silence tempts me too
Whose stillness I pursue.

The waters buoy my spirits high.
I'm weightless in your womb
Unaware that in my trance
It may transform to tomb.

As I descend the spell is cast
I'm held in your abyss.
My mind is fully narcotized
I float in heaven's bliss.

I am yours to do your will
And be at your command.
To serve your wish for evermore
Captive in Neptune's land.

Please, Siren of the Seven Seas
Take mercy on my soul.
Release me from your magic spell
And free of your control.

Let me return to nature's air
And reach my land alive.
I promise I will honour you
On each and every dive.

I'll tell all of your beauty great
And of the promises divine.
So they gaze down in respect of you
From the shallows of the brine.

John Parker
<www.thepoetrydoctor.com>

The Hyperbaric Research Prize

The Hyperbaric Research Prize encourages the scientific advancement of hyperbaric medicine and is awarded annually whenever a suitable nominee is identified. It will recognise a scholarly published work or body of work(s) either as original research or as a significant advancement in the understanding of earlier published science. The scope of this work includes doctoral and post-doctoral dissertations. The Hyperbaric Research Prize is international in scope. However, the research must be available in English. The Hyperbaric Research Prize takes the form of commissioned art piece and US$10,000 honorarium.

For detailed information please contact:
Baromedical Research Foundation
5 Medical Park, Columbia, SC 29203, USA
Phone: +1-803-434-7101
Fax: +1-803-434-4354
E-mail: <samir.desai@palmettohealth.org>

Asian Hyperbaric & Diving Medical Association

5th Annual Meeting

Dates: 25 to 27 September 2009
Venue: The Bogmallo Beach Resort, Goa, India
<www.bogmallobeachresort.com>
Guest speaker: Dr Simon Mitchell, New Zealand

For further information, registration & to submit a free poster/presentation:
E-mail: <ahdma.goa@gmail.com>
Website: <www.adhma.org>
USD $75 for early registration (before 30 June)

Note: This conference overlaps the 10th International Maritime Health Conference, 23–26 September. Details available on their website: <www.ismh10.com>

XIth International Meeting on High Pressure Biology

Dates: 31 August – 1 September, 2009
Venue: Faculty of Sciences, University of Brest, Brest, France
Organizing Committee:
Philippe Sebert (Brest, France)
Yoram Grossman (Beer-Sheva, Israël)
Steve Daniels (Cardiff, UK)

Registration: €80
Contact: <philippe.sebert@univ-brest.fr>

2009 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' UNDERWATER MEDICINE COURSE

Dates: 10 to 21 November 2009
Venue: HMAS Penguin, Sydney
Cost: tbc

The Medical Officers' Underwater Medicine 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.
For information and application forms contact:
The Officer in Charge, Submarine & Underwater Medicine Unit, HMAS PENGUIN,
Middle Head Road, Mosman, 2088 NSW, Australia
Phone: +61-(0)2-9960-0572
Fax: +61-(0)2-9960-4435
E-mail: <Scott.Squires@defence.gov.au>

ROYAL ADELAIDE HOSPITAL DIVER MEDICAL TECHNICIAN (DMT) COURSES 2009

Full DMT Courses:
16 November – 4 December

DMT Refresher Course
26 – 30 October

For more information contact:
Lorna Mirabelli
Senior Administrative Assistant
Hyperbaric Medicine Unit, Royal Adelaide Hospital
Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: <Lmirabel@mail.rah.sa.gov.au>

Situation vacant

The Alfred Hospital, Australia

Registrar/Fellow in Diving and Hyperbaric Medicine

Applications are sought for 2010 appointment as full-time Registrar in Hyperbaric Medicine at The Alfred Hospital, Melbourne, Australia. Fellowship durations are 6–12 months (February to July and/or August to January inclusive). The Alfred is a major academic teaching hospital and operates a large, modern, rectangular, multiplace hyperbaric chamber. It provides around 4–5,000 treatments per annum to a range of ambulatory through to critically ill patients each year.
For detailed information contact:
Dr Ian Millar, Unit Director
Phone: *+61-(0)3-9076-2269*
E-mail: *<i.millar@alfred.org.au>*

Instructions to authors

(revised March 2009)

Diving and Hyperbaric Medicine welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to *Diving and Hyperbaric Medicine*, unless clearly authenticated copyright exemption accompanies the manuscript. All manuscripts, including SPUMS Diploma theses, will be subject to peer review. Accepted contributions will be subject to editing.

Contributions should be sent to:
The Editor, Diving and Hyperbaric Medicine,
C/o Hyperbaric Medicine Unit, Christchurch Hospital,
Private Bag 4710, Christchurch, New Zealand.
E-mail: <spumsj@cdhb.govt.nz>

Requirements for manuscripts
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Microsoft® Office Word 2003. Paper submissions will also 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 supplied. The text should generally be subdivided into the following sections: an 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.

The text should be double-spaced, using both upper and lower case. Headings should conform to the current format in *Diving and Hyperbaric Medicine*. All pages should be numbered. Underlining should not be used. Measurements are to be in SI units (mmHg are acceptable for blood pressure measurements) and normal ranges should be included. Abbreviations may be used once they have been shown in brackets after the complete expression, e.g., decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is up to 3,000 words. Including more than five authors requires justification, as does more than 30 references. Case reports should not exceed 1,500 words, with a maximum of 15 references. Abstracts are required for all articles. Letters to the Editor should not exceed 500 words with a maximum of five references. Legends for figures and tables should generally be less than 40 words in length.

Illustrations, figures and tables must NOT be embedded in the wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image.
Table data may be presented either as normal text with tab-separated columns (preferred) or in table format. No gridlines, borders or shading should be used.
Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPG or BMP format. If figures are created in Excel, submit the complete Excel file. Large files (> 10 Mb) should be submitted on disk.
Photographs should be glossy, black-and-white or colour. Colour is available only when it is essential and may be at the authors' expense. Indicate magnification for photomicrographs.

References
The Journal reference style is the 'Vancouver' style (Uniform requirements for manuscripts submitted to biomedical journals, updated May 2007. Website for details: <http://www.nlm.nih.gov/bsd/uniform_requirements.html>). References must appear in the text as superscript numbers at the end of the sentence after the full stop.[1,2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used (<http://www.nlm.nih.gov/tsd/serials/lji.html>). Examples of the exact format for a 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.

Place a full stop after the journal name and at the end of the reference. Titles of books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying with the above requirements will be returned to the author before being considered for publication.

Consent
Studies on human subjects must comply with the Helsinki Declaration of 1975 and those using animals must comply with National Health and Medical Research Council Guidelines or their equivalent. A statement affirming Ethics Committee (Institutional Review Board) approval should be included in the text. A copy of that approval should be available if requested.

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' can be found on the EUBS and SPUMS websites (revised March 2009).

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)

SOUTH-EAST ASIA
+852-3611-7326 (China)
010-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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

EUROPE
+39-06-4211-8685 (24-hour hotline)

UNITED KINGDOM
+44-07740-251-635

USA
+1-919-684-8111
+52-5-629-9800 (America-Mexico))

LATIN AMERICA
+1-919-684-9111 (may be called collect; Spanish and Portuguese)

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. Information, all of which is treated as being confidential in regard to identifying details, is utilised in reports on fatal and non-fatal cases.

Such reports can be used by interested people or organisations 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>

DIVING INCIDENT MONITORING STUDY (DIMS)

DIMS is an ongoing study of diving incidents. 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.

Diving Incident Report Forms (Recreational or Cave and Technical)
can be downloaded from the DAN-AP website: <www.danasiapacific.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

CONTENTS

Diving and Hyperbaric Medicine Volume 39 No. 2 June 2009

Editorials

Original articles

Review articles

The world as it is

Opinion

Articles reprinted from other sources

Book review

SPUMS notices & news

EUBS notices & news

Diving and Hyperbaric Medicine is indexed on SCIE and EMBASE

Printed by Snap Printing, 166 Burwood Road, Hawthorn, Victoria 3122
hawthorn@snapprinting.com.au