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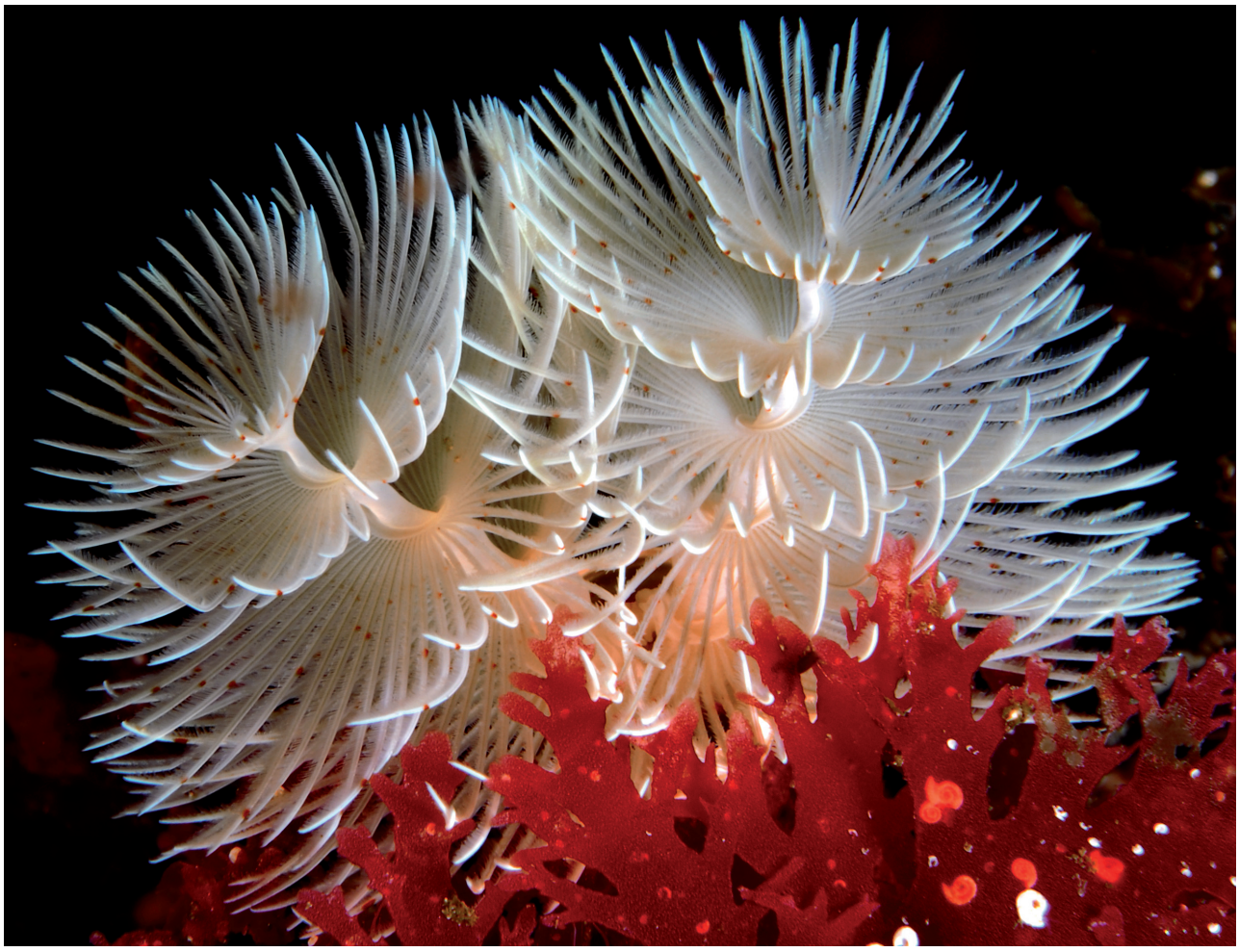

Best wishes to our readers for 2009

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

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

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The Editor's offering

Much of the epidemiological literature on sports injury suffers from insufficient information or poor study design or analysis. Such studies try to answer a series of questions.

- What is the incidence of injury? In diving, this has often been hampered by lack of reliable data on the size of the population at risk. Figures have been available for only a few specific at-risk groups and for scientific divers in the USA.[1]
- What are the nature and circumstances of injury? Descriptive studies such as Project Stickybeak and the Diving Incident Monitoring Study (DIMS) are good examples of the analysis of individual diving accidents and incidents.[2,3] Such studies do not provide incidence rates, but focus on the process of error, and in the case of incident reporting, regardless of outcome.
- What risk factors contribute to injury? As well as Stickybeak and DIMS, the reports of the Divers Alert Network (DAN) and the British Sub-Aqua Club (BSAC) have provided much information on those factors that contribute to diving accidents, and some clear patterns have emerged over the years.[4,5] Another example is a report from the Professional Association of Diving Instructors (PADI) looking at the incidence of arterial gas embolism during emergency ascent training, leading to PADI abandoning buoyant ascents.[6]
- What are the effects of preventive measures to minimize injury? By introducing changes, the efficacy of new medical screening, training methods, equipment or diving practices can be assessed prospectively. Because the main end points for scuba diving – death, decompression illness and drowning – are rare events, large numbers are required for this type of interventional investigation, and no such studies have been reported.

DAN has been collecting USA data since about the late 1980s, after the contract held by the University of Rhode Island with the Undersea Research Program expired. Whilst annual reports have been published, DAN has been criticised in some quarters for the lack of in-depth analysis of the data collected. Now, with a denominator of over a million membership years, DAN have been able to reliably analyse their database, giving a fatality rate for their recreational diving membership of 16.4 per 100,000 divers per year. A number of factors that have long been suspected are confirmed for DAN divers, namely that men are at greater risk than women and that the fatality rates for both sexes increase with age. Some interesting additional figures emerge for elderly divers that suggest several factors are at play here, not only cardiac risk. This raises important issues regarding on-going assessment of physical fitness and retention of diving skills in the older diver that need to be addressed by the recreational diving industry.

Studies from Australia, New Zealand, the UK and USA, have now reported little or no change in the annual numbers of diving deaths over the past decade or more. About a quarter of these divers had concomitant medical problems that, had they been identified, in many cases might have precluded involvement in scuba activities or that may have contributed in some way to the diver's death.

Divers who have had a long layoff from diving then return are thought to be over-represented in diving fatality statistics. A postal survey is reported in this issue that attempts to look at the reasons for such layoffs. Unfortunately its value is limited by a low response rate (always a problem with such surveys), but a few useful indicators are provided that could also be worth addressing by the recreational industry. More data are needed in this area.

Two established diving research laboratories, in Norway and Israel, add to the slowly growing list of basic science research papers coming to *Diving and Hyperbaric Medicine*. The idea that vigorous exercise roughly a day before diving may reduce the risk of decompression sickness has been rather over-enthusiastically taken up by the diving community, long before the scientific basis for this observation is fully understood. Since heat shock proteins are produced by both stresses, Medby and colleagues hypothesise that prior heat stress works in the same way as exercise in a rat model, but find that this may not be so. The high-pressure physiology group at Ben-Gurion University of the Negev continue their work elucidating the neuropharmacological mechanisms involved in the high pressure nervous syndrome (HPNS) associated with helium breathing by examining the expression of the glutamatergic *N*-methyl-D-asparate receptor, which is thought to be linked to the development of HPNS symptoms.

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Michael Davis

The front-page photo was taken in Fiordland, New Zealand by Dr Martin Sayer; red coral, *Errina novaezelandiae*, and serpulid (tube) worm, probably *Protula bispiralis*.

Original articles

Scuba injury death rate among insured DAN members

Petar J Denoble, Neal W Pollock, Panchabi Vaithiyanathan, James L Caruso, Joel A Dovenbarger and Richard D Vann

Key words
Accidents, age, cardiovascular, deaths, diving accidents, DAN – Divers Alert Network, epidemiology

Abstract

(Denoble PJ, Pollock NW, Vaithiyanathan P, Caruso JL, Dovenbarger JA, Vann RD. Scuba injury death rate among insured DAN members. *Diving and Hyperbaric Medicine*. 2008; 38: 182-188.)
We calculated the annual rates of diving-related deaths among DAN-insured members in the period from 2000 to 2006 and investigated the effects of age and sex on death rate by logistic regression. We determined relative risks for divers < 50 and ≥ 50 years of age for drowning, arterial gas embolism, and cardiac incidents, the three most common disabling injuries associated with diving death. There were 1,141,367 insured member-years and 187 diving-related deaths. Males made up 64% of the members. Individuals ≥ 50 years of age constituted 31% of the fatalities. Insured mean age increased from 40 ± 12 to 43 ± 13 years over the seven-year study period. Annual fatality rates varied between 12.1 and 22.9 (average 16.4, 95% confidence intervals 14.2, 18.9) per 100,000 persons insured. The relative risk for male divers in their thirties was six times greater than the risk for female divers in the same age range. Fatality rates increased with age for both sexes, but the higher relative risk for males progressively decreased until the rates became similar for both sexes after age 60. Death associated with cardiac incidents was 12.9 times more likely in divers ≥ 50 years of age. We recommend that older divers adjust their participation in diving according to health status and physical fitness, maintain fitness with regular exercise, and abstain from diving in conditions likely to require unaccustomed physical activity.

Introduction

Recreational scuba diving is associated with hazards inherent to all water-related activities as well as hazards specific to underwater breathing and environmental pressure changes. Injuries from these hazards can be fatal and occur unpredictably. Recreational dive training organizations set standards for safe diving practices to prevent injuries, but the responsibility for safety is ultimately that of the individual diver. A lack of understanding of the risks may make divers less compliant with safe practices and undermine risk mitigation initiatives.

The definition of risk includes both the probability of an adverse event and the severity of its consequences, but the popular perception of risk seems to be affected only by severity, not probability.[1] In a survey of 444 subjects, for example, scuba diving was ranked as more risky than snow skiing but less risky than bungee jumping, rock climbing, motorcycle racing, hang gliding, cliff jumping and sky diving.[2] In fact, the actual likelihood of injury in open water recreational diving seems to be 100 times less than the likelihood of injury in snow skiing.[3] However, injuries in diving may involve higher mortality due to the associated hazard of drowning, the most common cause of diving-related deaths.[4]

Unlike subjective perception, objective risk estimation is based on knowledge of past occurrences of injuries, their frequencies, severity, numbers of persons exposed and measures of their exposure. Complete information is rarely available in recreational scuba diving. The annual count of injury deaths in the United States (USA) and Canada has been surveyed since the 1960s. Peaking at 150 in the mid 1970s, the death count has been fairly stable for more than a decade at 84 ± 5 (range 77–91).[5] Without knowing the number of persons exposed, however, it is impossible to compare the risk of drowning for scuba divers and non-divers or to establish the absolute risk of death associated with diving. Attempts to calculate rates using projected numbers of divers and dives have produced estimates ranging from 3.2 to 34 per 100,000 divers and from 0.37 to 4 per 100,000 dives.[6–12]

The British Sub-Aqua Club (BSAC) has reported death rates on a per-diver basis for its membership since 1959. The count of BSAC members varied between 3,000 and 42,000 and the annual number of deaths from 1 to 10. Annual rates varied between 6.0 and 58 per 100,000 members.[8] With so few deaths annually, random variations may substantially affect the death rate independent of changes in external causes or specific risk factors. The BSAC estimates do not address the incidence per exposure since the individual frequency of diving is reported only for subgroups of divers, such as those with diabetes.[13]

Frequency data have been available for some specialty areas like cave diving and scientific diving. Diving in caves

with the associated hazards of becoming lost or entrapped or exhausting gas supplies before reaching the surface, is known to be a higher-risk endeavour than diving in open water.[14,15] On the other hand, scientific diving, with strong organizational safety policies, appears to be less risky than open-water recreational diving. In 2003 for example, the American Academy of Underwater Sciences (AAUS) recorded 104,921 dives and no fatalities.[16]

Recent trends find an increasing number of older persons participating in recreational activities.[17] While complete diver population records are not generally available, Divers Alert Network (DAN) data indicate that the age of divers is increasing.[5] Cardiovascular disease has also become increasingly recognized as a factor in the death of older divers.[18] Given the low incidence of fatalities and missing denominators, associations of dive-related deaths with age and health issues have not been tested. Large data sets are required for sufficient power to address such effects.

DAN is a not-for-profit organization with a large membership and good access to scuba injury and fatality data for the USA and Canada. Member benefits include a 24-hour emergency line and dive accident and travel insurance. DAN receives information on incidents and accidents through direct involvement in cases and by collecting data through active surveillance programmes. We used data for insured DAN members to calculate the annual rates of diving-related deaths, the influence of age and sex on the rates, and the relative risks of divers above and below 50 years of age for the disabling injuries of drowning, arterial gas embolism, and cardiac incidents.

Methods

Since 2000, the DAN membership database has included information about age and sex. Data about deaths came from insurance claims and DAN's fatality surveillance programme. Each case reported through the surveillance programme was cross-checked with the DAN membership database. The study was approved by the Duke University Medical Center Institutional Review Board. We extracted de-identified information from the membership database for all living divers and merged it with fatality information. The final dataset consisted of all insured DAN members for the period of 2000 through 2006 with age, sex, and fatality indicators.

A scuba fatality was defined as the death of a diver equipped with scuba who had entered the water with the intent to dive and had died in the water or had left the water with a disabling injury and consequently died. In our analysis of 947 diving fatalities, the medical examiner specified drowning as the cause of death (COD) in 70% of the cases.[17] For the majority of divers who drowned, however, there was evidence that a prior disabling injury was directly responsible for death or for incapacitation followed by death due to drowning. Thus, we elected to focus on the disabling injury as more relevant to diving safety than the subsequent COD, drowning. We found the three most common disabling injuries to be drowning, AGE, and cardiac incidents, and in the present paper, we investigated the association of these injuries with age above and below 50 years.

The mean ages of DAN-insured members were calculated for each calendar year. Annual fatality rates per 100,000 insured members were calculated based on the number of fatalities and the number of insured members. We adopted the rate per 100,000 exposed people as this unit is used in most national injury statistics. Sex and age-specific mortality rates were calculated using aggregated data for the entire observation period. Mortality rates for the three most common injuries – drowning, arterial gas embolism (AGE), and cardiac events – were calculated separately for insured members of < 50 and ≥ 50 years of age.

Data are shown as mean ± standard deviation where applicable. The difference in mean age between sexes was tested using a two-sample Wilcoxon test with significance accepted at $P < 0.05$. Trends in mean age over time were tested by linear regression. Mortality rates were calculated by dividing the number of injury deaths by the number of insured members and multiplying by 100,000. Rates were shown with 95% confidence limits (CI). Effects of age and sex on fatality rates were tested with logistic regression using backwards elimination of non-significant variables ($P < 0.05$). Differences in the disabling injury-specific fatality rates were calculated as a relative risk (RR) with 95% CI. If the lower confidence bound was less than one, the difference was considered not to be significant.[19]

Results

There were a total of 1,141,367 insured member-years and 187 scuba diving-related deaths among them in the seven-year period studied. The age distribution by sex is shown in Figure 1. Males represented 64% of all insured members in this period. Divers in their forties were the largest subpopulation (30%) while divers ≥ 50 years of age represented 31% of all diving-related fatalities.

Figure 1
Age distribution of insured member-years
males – black; females – white

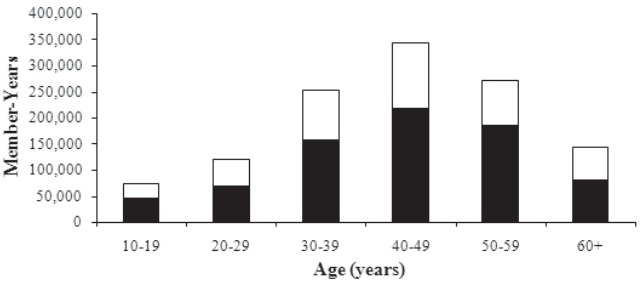
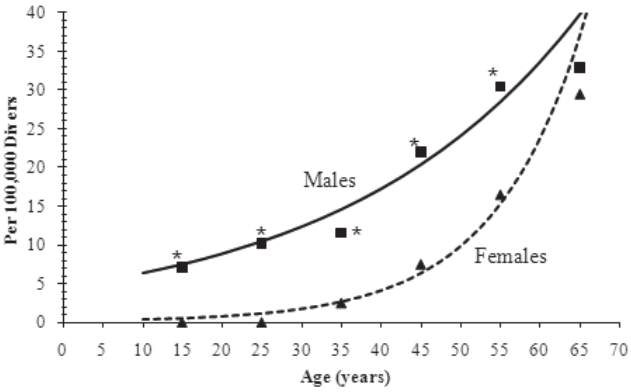


Figure 2
Fatality rates by gender and age. Lines indicate logistic regression-predicted rates, and squares and triangles represent mean values by sex for 10-year age groups; * indicates significant gender contrasts



The mean age of insured divers increased significantly from 2000 to 2006; from 41 ± 12 to 44 ± 14 years for males and from 39 ± 11 to 42 ± 13 for females. The pooled age increased from 40 ± 12 to 43 ± 13 years (median from 41 to 45, respectively). The percentage of divers 24 to 44 years of age decreased and the percentage of divers ≥ 45 years increased. The largest change was in age group 55–64, which increased from 9.2% (2000) to 18.1% (2006) of the total.

The overall rate for the seven-year period was 16.4 (95% CI 14.2, 18.9) per 100,000 persons, with annual rates varying from 12.1 to 22.9. The overall rate for males was 21.1 (95% CI 18.0, 24.7) and for females 7.6 (95% CI 5.4, 10.8). The relative risk for males was 2.8 (95% CI 1.9, 4.5) times the risk for females.

Raw composite data and logistic regression model results for rate by sex and age are shown in Figure 2. Logistic regression was conducted using continuous age data. The squares (male) and triangles (female) in Figure 2 represent mean values by sex for 10-year age groups. Fatality rates on a per-diver basis increase with age for both males and females. The rate for males was significantly higher for age groups < 60 years. The male fatality rate increased from 7.0 in teenagers to 33.0 per 100,000 divers in the ≥ 60 year age group. There were no recorded fatalities in females up to age 30 despite the fact that the participation rate was similar to that in the ≥ 60 year group. The female fatality rate increased from 3.0 in the 30–39 year age group to 29.1 per 100,000 divers in the ≥ 60 year group.

Logistic regression indicated there was an interaction between sex and age as shown by the convergence of the male and female risk lines in Figure 1 with increasing age. This is also reflected in Table 1 for the relative risks of males and females. The relative risk was significantly greater for men up to age 59 and similar thereafter. Values for the 10 to 29-year age range were extrapolated from the regression model.

There were sufficient data to investigate the probable disabling injury resulting in death in 129 cases of insured fatalities. The overall death rates as well as disabling injury-specific rates were compared in insured divers of ages up to 49 years and ≥ 50 years (Table 2). Presumed cardiac events were most common in the older group (37%), 13 times more likely (95 % CI 5.0–33.5) as a disabling injury than in the younger group. In addition, older divers had a greater relative risk both for AGE (RR 3.9) and drowning (RR 2.5).

Discussion

AGE OF INSURED DIVERS

The overall mean age of insured DAN members increased through the observation period. The representative portion was unchanged in the 15–24 year age group, decreased in the 25–44 year age groups, and increased in the 45 and older groups. This may indicate that young people still show the same initial interest for scuba diving but do not stay active for a long time. The largest increase in representation was in the 55–64 age group, reflecting the ageing diving population. We do not know how the age distribution of DAN members compares to other groups of divers.

Table 1
Relative risks of dive-related death by age and gender, [1] values based on regression model; * indicates significant sex contrasts

Age group (years)	Rate		Relative risk for males		
	Males	Females	RR	95% CI	
10–19	7	0	17[1]		
20–29	10	0	9.8[1]		
30–39	11	2	5.5*	1.3	23.6
40–49	22	7	3.1*	1.5	6.2
50–59	30	16	1.9*	1.1	3.4
≥ 60	33	29	1.1	0.5	2.8

Table 2
Cause-specific frequencies of death and age-dependent relative risks

Cause of death	< 50 years n=788,489	≥ 50 years n=352,878	RR	Lower limit	Upper limit
Cardiac	5	29	12.9	5.0	33.5
AGE	8	14	3.9	1.6	9.3
Drowning	15	17	2.5	1.3	5.1
Unknown	15	16	1.9	0.9	4.1
Other	7	3	1.0	0.2	3.7
Total	50	79			

OVERALL FATALITY RATE

In the current study based on a known number of insured members and number of dive-related deaths, we have established the death rate at 16.4 per 100,000 insured members for all ages and both sexes. The reliability of the rate depends on the validity of both the denominator and the number of deaths. In the case of DAN-insured members, it is likely that most deaths were captured.

A summary of previously published fatality rate estimates is given in Table 3. Estimates are presented both as the rate per 100,000 members and the rate per 100,000 dives, when available. BSAC and DAN fatality rates are computed from large datasets with measured denominators. Most of the published fatality rates are based either on small data sets,[11] surrogate measures,[9,10,12] or estimated denominators.[9,11,15]

The fatality rates per 100,000 divers derived from DAN Insured and BSAC data, 16.4 and 14.4 respectively, are reasonably similar given the expected variability and small total number of events. Despite variability, annual rates for the period 2000–2006 were not statistically different either. Conversely the rate of 3.4–4.2 per 100,000 divers rate from a study commissioned by the Divers Equipment and Marketing Association (DEMA) is much smaller, but the numerator and denominator were drawn from different sources.[20] The number of fatalities (the numerator) came from DAN data while the population at risk (the denominator) was estimated in a marketing study at between 2.1 and 2.7 million. Inaccuracy is likely when numerator and denominator are drawn from different populations.

A similar rate was estimated recently for Australia. Based on a survey of diving activities of overnight visitors to Queensland between April 2006 and March 2007 and the annual "participation in exercise, recreation and sport survey," the rate for combined local population and overnight visitors was established at 3.7 per 100,000 divers or 0.57 per 100,000 dives.[9] The erroneous use of overnight visitors as a denominator to calculate the annual rate per population probably led to underestimation of the actual annual fatality rates since for most visitors the outcomes were known only for the time of visiting rather then for the entire year. The estimated rate per 100,000 dives was the same as one in the study ordered by DEMA, possibly due to the same bias.

Using the reported number of tank fills as a proxy for the number of dives and fatality information provided by Coroner Services, the fatality rate per 100,000 dives in British Columbia, Canada was established with only three fatalities reported for the 14-month period. The validity of the estimate was further limited by the fact that only 65% of the identified filling stations participated.[12] The calculated rates were within the 95% confidence limits of those established by similar methods in Japan, and Victoria,

Table 3
Overview of recorded and estimated scuba injury death rates

Group	Denominator	Time period	Rate (95%CI) per 100,000 divers	Rate (95%CI) per 100,000 dives	Reference
Cave Divers, UK	Measured	1957–1979	–	138 (65, 300)	14
Cave Divers, UK	Measured	1980–2006	–	24.6 (12. 50)	15
USA	Estimated	1986	3.4–4.2	–	6
USA	Estimated	1989	16.7	0.8–1.6 (1.6, 12.7)	6
Orkney, Scotland	Measured	1999–2000	–	4	11
Australia	Estimated	1989	34	1.7–3.4	7
Victoria, Australia	Estimated by tank fill count	1992–1996	–	2.5	9
Australia	Estimated by survey	2000–2006	3.57	0.57	9
British Columbia, Canada	Estimated by tank fill count	1999–2000	–	2.04 (0.8, 6)	12
Japan	Estimated by tank fill count		(8.8, 33.8)	1 to 2.4 (0.5, 2)	10
BSAC	Measured	2000–2006	14.4 (10.5, 19.7)	–	8
DAN Insured	Measured	2000–2006	16.4 (14.2, 18.9)	–	Current study

Canada as well as the rates calculated from limited Orkney data.[9–11]

Fatality rates based on the number of participants do not take into account frequency of participation among individual divers. Per-dive-based rates established using known number of dives and accidents are rare in recreational diving. The AAUS reports no deaths in 104,921 logged and reported scientific dives.[16] Computing the upper 95% confidence limit it is expected that the mortality rate in scientific diving is less than 3 per 100,000 dives.

A British cave diving group has tracked its membership diving activity and accident data since 1957. Despite their small numbers, multi-year data indicate a significant decrease in the fatality rate from 138 (95% CI 60, 300) before 1980 to 24.6 (95% CI 18, 50) per 100,000 dives after 1990.[14,15] While improved, cave diving appears to be much riskier than recreational open water diving.

COMPARISON WITH OTHER INJURY DEATHS

According to the National Center for Health Statistics (NCHS) unintentional injury was the fifth leading cause of death in 2004. There were 117,809 fatal injuries making up 4.7% of all deaths in the United States. The overall death rate from injuries in the general population was 35.5 and from traffic accidents 16 per 100,000 persons annually.[17] The annual death rate of 16.4 per 100,000 divers is similar to the death rate due to heart attack while jogging (13 per 100,000 joggers).[21] It is important to remember, however, that these rates do not reflect exposure time. For example, the vast majority of persons will have much greater exposure to traffic situations than diving situations. The risk of diving-related death for scuba divers has been described as 13 times greater than the risk of drowning for the general population.[18] In this case, however, the rate for drowning was calculated based on the entire population rather than just those who were exposed to water hazards. This is very different from the diving-related death rates calculated for a group in which all members are exposed to diving hazards.

EFFECT OF AGE AND SEX ON DEATH RATE

The possible effects of age on risk of injury and death in recreational activities are of special interest due to the ageing of the general population and an increased participation of older people in recreational activities. For example, emergency medicine department reports indicate a substantial number of participants ≥ 65 years of age among those injured in recreational sports. They represented 17% of the injuries in golf, 15% in tennis, 9% in fishing and 4% of injuries in scuba diving (again with no exposure duration information).[17]

Our data indicate a clear effect of age and sex on the risk of death in diving. DAN-insured members ≥ 60 years of age have a relative risk four times greater than that of male teenagers. Young adult males have a four-fold greater risk than young adult females. Differences in risk associated with sex disappeared by 60 years of age. Youthful differences between sexes may reflect greater risk-taking by males, which may be related to testosterone.[22] However, the rates increased with age despite decline of testosterone levels.

The risk of dying during physical activity for older persons is associated with a high prevalence of heart disease. Persons 66–74 years are 27 times more likely to die from acute myocardial infarction than persons 35–44 years of age.[23] Responses to diving stress and exertion in persons with diagnosed and undiagnosed heart disease may cause death or weakness that is complicated by the possibility of drowning.[24,25]

Differences in fatality rates for males and females and their change with age may be partially explained by the different prevalence of heart disease in the two sexes. Oestrogen has long been considered a protective factor against heart disease in women, but recent epidemiological studies show only a relatively small effect.[26] Heart disease as an underlying risk in diving is hard to determine due to confounding effects of drowning and thus it may be underreported.[27] On the other hand, in the absence of evidence, the diagnosis may be biased toward cardiovascular disease-related causes in older victims. The diagnosis of disabling injury in our data was based on health history, accident scenario and autopsy findings.[18]

Excluding cardiac causes, which were suspected in one third of deaths in divers over 49 years of age, the fatality rate in older divers would still be greater than in younger divers (Table 1), as the relative risks for arterial gas embolism and drowning suggest. This may be related to a decline in physical ability – aerobic capacity, muscular strength, flexibility, coordination and dexterity – all of which may make older people prone to errors and more vulnerable.[17,28] Association of age with a relative increase of risk of death in diving requires additional research to establish its true meaning and possible causes.

The preventability of cardiac deaths in diving depends on divers' knowledge of their health risks, an appreciation of specific dive risks and a willingness to adjust behaviour which may be affected by readiness to trade risk for freedom. Some decedents in our database knew about their health status, but we do not know if they had sought specific evaluation regarding medical fitness to dive. Mandating strict annual medical evaluation outside of standard care would not necessarily contribute much to their self-knowledge. However, a fraction of decedents who were not aware of pre-existing conditions could have benefited from regular medical examinations. According to the American Heart Association (AHA), the screening for coronary artery disease risk factors should start at age 20. At this age it includes family and personal history. Most pre-participation questionnaires administered by training agencies fulfil this

purpose.[29] Divers over 35 might benefit from discussing their heart risk factors with their primary care physicians. In addition, the AHA suggests medically-supervised exercise stress tests in men over 40 years of age and women over 55 years of age who wish to engage in vigorous training or competitive sports and who have two or more coronary risk factors (other than age and sex) or one markedly abnormal risk factor. A selective approach to medical evaluation of fitness for diving based on individual risks seems most suited for recreational diving.[30]

The DAN membership indicates that a large number of older people chose diving for recreation. The benefits of this active lifestyle should not be underestimated, but stress and exertion (neither unique to diving nor the only factors that may precipitate sudden cardiac death) are hazards that must be considered when evaluating health and fitness to dive.[28,31] While there seems to be a broad consensus regarding criteria for medical fitness to dive, understanding the risks by both divers and their physicians is essential. The final decision to participate is up to the individual. Personal choices may go against medical advice but being properly informed makes this less likely.

Conclusions

We calculated fatality rates for DAN insured members and reported them with reference to the risk of mortality associated with other activities. The occurrence of dive-related deaths is rare and, even with a large group of divers, multi-year data are needed to investigate specific risk factors. We found that risk is greater for males and increases with age for both males and females. The most significant injury contributing to increased risk appears to be heart disease, but this alone does not account for all excess mortality. The risk of death associated with scuba diving reflects similar trends in injuries in the general population. Age itself is not a causative factor and more research is necessary to understand how age affects diving risk. Healthy lifestyle and regular physical activity affects quality of life, morbidity and mortality. All divers should take care of their health and maintain fitness with regular exercise. Older divers should regularly monitor their health and physical fitness and adjust participation accordingly.

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During the 1980s, the front page of the *SPUMS Journal* carried a series of cartoons by one of its members, Dr P Horne sadly now deceased, depicting a scene pertinent to that issue. This one, from a quarter century ago, highlights the growing concern at that time about cardiovascular health and the ageing recreational diving population.

Heat shock increases survival in rats exposed to hyperbaric pressure

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Key words
Decompression, bubbles, trauma & stress, Doppler, hyperbaric research

Abstract

(Medby C, Bye A, Wisløff U, Brubakk AO. Heat shock increases survival in rats exposed to hyperbaric pressure. *Diving and Hyperbaric Medicine*. 2008; 38: 189-193.)
It has been shown that a single bout of exercise performed 20 hours prior to hyperbaric exposure reduces bubble formation and increases survival in rats. Heat shock proteins (HSPs) are stress proteins expressed in cells that are exposed to different stressors. HSPs are known to protect cells, by binding to proteins and stabilizing them. As it is known that a single bout of exercise induces HSPs, and that HSPs exert their protective effects 20–24 hours after the stimulus for induction, we hypothesized that HSPs might be one mechanism behind the observed exercise-induced protection. We hypothesized that rats that expressed HSPs would develop fewer bubbles and have a lower mortality than their non-stressed control group. Twenty-four female Sprague-Dawley rats (300–330 g) were divided into a heat-shock group and a control group and anaesthetized. The rats in the heat-shock group were heated to 42 ± 0.5^{O}C for 15 min. The following day, all rats were compressed to 700 kPa for 45 min in a hyperbaric chamber. The right ventricles were insonated and bubbles were identified and graded. Six of 12 rats in the heat-shock group survived, while 1 of 12 control rats survived ($\chi^2 = 5.042$, P = 0.034). There was no difference in bubble grade between the groups. The study suggests that the effect of heat shock on survival is not the same as observed after exercise, as the heat-shocked rats developed bubbles. However, heat shock appears to protect rats against the effects of bubbles by an independent mechanism.

Introduction

We have shown previously that a single bout of exercise 20 hours prior to a simulated dive reduces bubble formation and increases survival in rats exposed to hyperbaric pressure.[1] The precise mechanisms are not known, but nitric oxide (NO) seems to be involved, as NO synthetase (NOS) blockade promotes bubble formation in sedentary rats and NO donors protect against bubble formation.[2,3] It is known that NO plays an important role in adaptive defence of the cardiovascular system, in particular as a result of induction of heat shock protein-70 (HSP70) synthesis.[4] NO production is also increased with increased expression of HSP90.[5] Thus, a link between NO and HSPs has been established in the literature.

HSPs are stress proteins that are induced when cells are exposed to different stressors, such as hyperthermia, hypoxia, hyperbaric stress or exercise.[6] Increased expression of these proteins is associated with cell protection, probably by acting as molecular chaperones and rescuing denatured proteins. The protection is not only against the original stressor, but also against other stressors (cross-tolerance).[7]

A non-significant 23% reduction has been observed in the incidence of decompression sickness in rats after preconditioning with heat shock, whereas a significant protection occurred against the effects of venous air infusion.[8] In either case, symptoms or death are caused by gas bubbles in the circulation, obstructing and injuring the vessels. This leads to reduced blood flow and consequent ischaemia.

Preconditioning with whole body hyperthermia ('heat shock') has been shown not only to protect rats against subsequent hyperthermia that is otherwise fatal, but also against ischaemic injury to the heart and central nervous system. The protection is thought to be mediated through increased expression of HSPs, and the optimal interval between the preconditioning and the insult seems to be about 24 hours.[9] This is similar to our training-induced protection against bubble formation and death. Acute severe exercise also increases HSP expression in rats, and this increased expression protects rats against ischaemia.[7,10,11] It is tempting to speculate whether HSPs could be responsible for one of the mechanisms behind the exercise-induced protection against decompression, as the time frame for the exercise-induced protection and the expression of HSPs is similar.[12]

Thus the aim of the present study was to determine whether rats exposed to heat shock are protected against hyperbaric exposure similarly to exercised rats. The rats were exposed to the same hyperbaric stress as in the exercise studies, and would be expected to behave similarly if the underlying mechanisms of protection were the same. We hypothesized that rats exposed to heat shock would develop fewer bubbles and have a lower mortality than the control group.

For protein analysis, HSP70 was chosen on the basis of

being the uppermost inducible protein of all the HSPs. HSP90 was analyzed due to its capability of inducing NO-production, in addition to its chaperon effect. Both proteins are considered markers of HSP induction. We did not aim to show any causation.

Methods

The study protocol was reviewed and approved by the Norwegian Animal Research Authority and all procedures were conducted in accordance with the *European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes*.

MATERIAL

A total of 26 adult female Sprague-Dawley rats (300–330 g) (Kirkeby, Sweden) were acclimatized for a minimum of five days. Prior to the experiments, the rats were kept in cages with a volume of 46 L in groups of four to six. The room was artificially lit from 1800 to 0600 h. Temperature was maintained at 25.2 ± 1.7^{O}C, and humidity at 49 ± 4%. The rats were fed a pelleted rodent diet (rat and mouse standard diet, B&K Universal, UK) and water *ad libitum*; the rats were not fasted at any time.

METHODS

Twenty-four rats were divided into a heat-shock or a control group. Rats from both groups were anaesthetized using a mixture of midazolam (1 mg.kg^{-1}), fentanyl (0.07 mg.kg^{-1}) and fluanisone (2 mg.kg^{-1}) as subcutaneous injections. The rats in the heat-shock group were placed in a custom-built heating chamber and heated for 31.2 ± 5.8 min until rectal temperature reached 42.0 ± 0.5^{O}C, maintained for 15 min. The cylindrical heating chamber was open at both ends, allowing room air to circulate freely. The temperature was regulated using electrical heating elements built into the wall. The rats were placed on a grate, in no direct contact with the heating elements. After the heat shock procedure, the rats were returned to the cage with free access to food and water and allowed to recover from anaesthesia.

Twenty-four hours later the rats did a simulated dry air dive in a hyperbaric chamber. Rats were compressed in pairs at 200 kPa.min^{-1} to 700 kPa. After 45 min, the rats were returned to surface pressure at 50 kPa.min^{-1} and immediately anaesthetized. The right ventricle was insonated using a GE Vingmed Vivid 5™ scanner, with a 10 MHz transducer. Bubbles were identified as bright spots in the ventricle or in the pulmonary artery, and graded on a scale from zero to five as described previously.[13] Doppler and slow-motion playback were used to aid grading. The rats were observed for 60 min, before surviving rats were sacrificed by decapitation. The hearts and the great vessels were examined for gas bubbles. The left ventricle of the heart and the aorta were dissected free, frozen in liquid nitrogen and stored at –80^{O}C for subsequent analysis of HSP70, HSP90 and endothelial NOS (eNOS) expression.

To investigate the relative contribution of the hyperbaric exposure to the expression of HSPs, two rats served as non-diving controls. The rats were treated like the control group, but were sacrificed without undergoing the compression–decompression procedure.

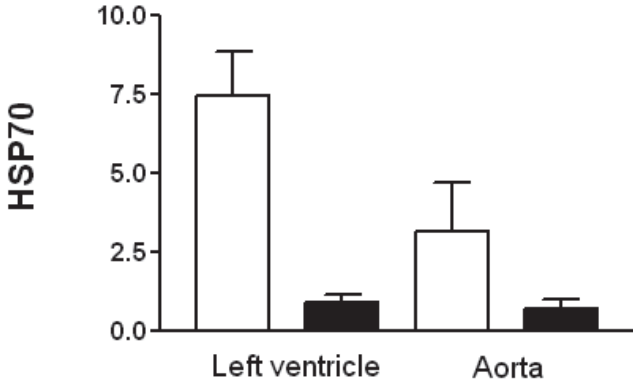
WESTERN BLOT ANALYSIS

The expression of HSP70 (inducible), HSP90 and eNOS was determined by Western immunoblotting. Tissue samples (20 mg) from both the aortas and the left ventricles of the hearts were homogenized in cold lysis buffer (100 µL) using a Mixer Mill MM301 at 20–25 Hz. The lysate was centrifuged at 12,000 *g* for 5 min at 4^{O}C. The protein concentration in supernatant was quantified by using a Coomassie Plus Protein Assay Reagent Kit (Pierce, Rockford, IL, USA) and was diluted with BupH Tris-HEPES-SDS Running Buffer (Pierce, Rockford, IL, USA) to a concentration of 1µg.µl^{-1}. Equal amounts of sample and sample buffer (12.5% Tris buffer (0.5 M), 2% SDS, 10% glycerol, 5% 2-β-mercaptoethanol and 0.05% bromophenol blue) were mixed together, and then denatured in boiling water for 5 min and stored on ice. 10 µg of total protein per sample was loaded on a 10% polyacrylamide gel (Pierce, Rockford, IL, US). Two standards were used on all gels: Magic Mark Western Standard (Pierce, Rockford, IL, US) and the pre-stained SeeBlue standard (Invitrogen, Carlsbad, CA, USA). The electrophoresis was performed under constant voltage (150 V) for 39 min. The proteins were then blotted onto a PVDF-membrane (BioRad, Hercules, CA, USA) under constant voltage (30 V) for 1 hour, using NU-PAGE Transfer Buffer (Invitrogen, Carlsbad, CA, USA). Nonspecific binding to the membrane was blocked by 5% bovine serum albumin in Tris-buffered saline (TBS) overnight at 4^{O}C. After two washes with TBS-T (containing Tween20) the blots were cut into appropriate pieces depending on the migration of the proteins on the gel. In this way, one might separate the bands of eNOS, HSP90, HSP70 and actin (housekeeping protein). Each piece of the membrane was incubated in its appropriate antibody for one hour at room temperature. The membrane pieces were subjected to four washes with TBS-T and incubated with their appropriate secondary antibody for one hour at room temperature. The membrane pieces were once again subjected to four washes with TBS-T and developed with a chemiluminescence detection system (Supersignal, WestFemto, Pierce, Rockford, IL, USA) for five min and exposed to film (Amersham ECL, Sweden). The results were quantified using VersaDoc Imaging system and QuantityOne software (BioRad, Hercules, CA, USA).

STATISTICS

The Chi-square test was used to evaluate differences in survival between groups. Fisher's Exact test (1-sided)

Figure 1
Semi-quantitative levels of heat shock protein-70 (HSP70) in left ventricular and aortic tissue; mean density ratios ± SD (arbitrary units)
□ Heat shock group ■ Control group



was used to calculate *P* values. Western immunoblotting is a semi-quantitative method in which it is possible to detect relative differences in concentration. Differences in protein expression were evaluated using the Mann-Whitney test (two-tailed). A value of $P < 0.05$ was considered significant.

Results

In the heat-shock group, six of 12 rats survived the observation period, while one of 12 control rats survived ($\chi^2 = 5.04$, df = 1, $P = 0.034$).

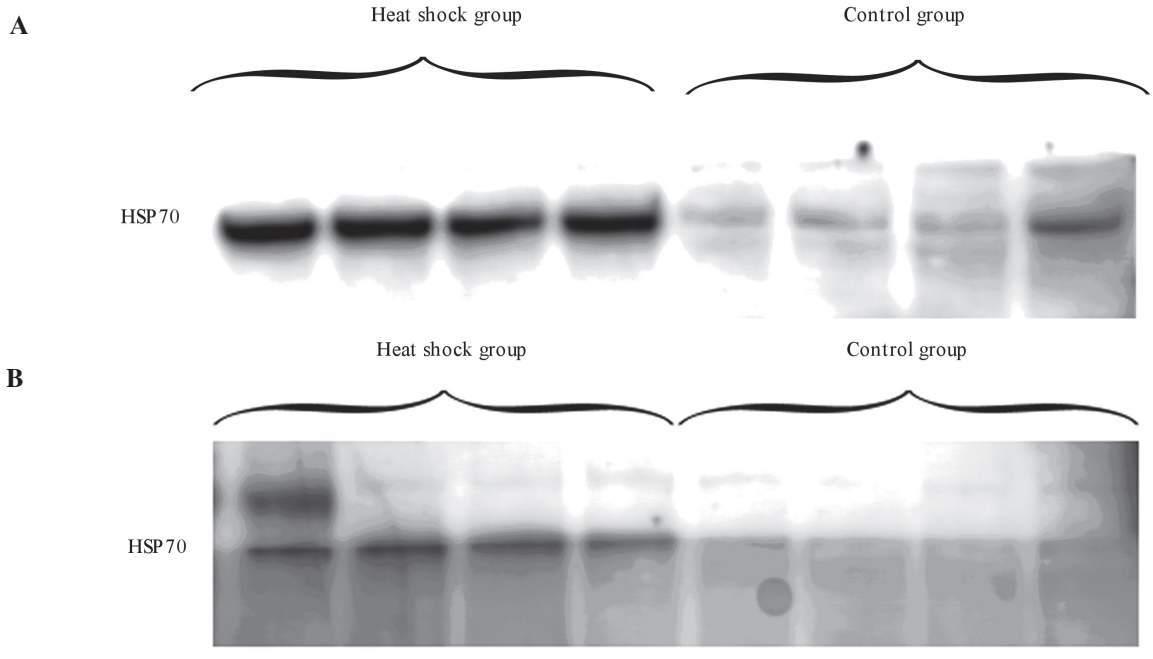
Eleven rats in each group developed severe bubbling, either grade 4 or fatal before they could be graded. In the control group all rats with bubbles died, while five rats in the heat shock group with bubbles survived ($\chi^2 = 6.47$, df = 1, P = 0.018). One rat in each group had bubble grade 0 during the entire observation period, and survived. At autopsy all rats that died before the end of the observation period had their hearts filled with blood foam. There was less gas in the hearts and vasculature of surviving rats, but bubbles could be identified in all rats, except the rats with a grade 0 bubble score.

Pretreatment with heat shock and exposure to hyperbaric pressure caused an eight-fold increase in HSP70 expression in left ventricular tissue ($P = 0.021$) and over a four-fold increase in aortic tissue ($P = 0.021$; Figure 1). Representative blots are shown in Figure 2. The eNOS and HSP90 levels were not affected by the pretreatment with heat shock (blots not shown). The non-diving controls expressed HSPs and eNOS at the same levels as the diving controls (blots not shown).

Discussion

This study demonstrates that preconditioning with heat shock protects rats from the effects of bubbles following decompression from a dive. However, the pretreatment does not affect the number of bubbles formed. Earlier, we reported that acute severe exercise performed 20 hours prior to a dive increased survival in rats,[1] but this protective mechanism appears different from the one revealed in the present study, since the exercise-induced protection also involves reduced bubble formation. In the present study, half the rats in the

Figure 2
Western immunoblotting for expression of heat shock protein 70 (HSP70); representative blots are shown
A – left ventricular tissue; B – aortic tissue



heat-shock group survived in spite of the bubble formation. The protective effect of exercise has been shown to be related to NO production.[2,3,14] Exercised rats also express HSPs and would be expected not only to be protected against forming bubbles, but also against the effects of the bubbles.

The dive profile in this study was chosen to make comparison with the exercise studies possible, and the results clearly show that HSPs alone cannot explain the protective effect of exercise. The animal model used is, however, too crude to draw any clear conclusions about the mechanisms behind the observed heat-shock-induced protection.

Western blot analysis showed a significant increase in HSP70 levels in both left ventricular and aortic tissues after thermal preconditioning, but the causality between HSP70 and increased survival after decompression remains speculative. Since increased expression of HSPs also appears to protect rats against myocardial ischaemia in infarct models, the increased survival in our study could be due to a better tolerance for ischaemia caused by hypotension and gas embolism.[15,16] It is plausible that the elevated amounts of HSP70 are involved in the protection against effects of myocardial ischaemia, such as arrhythmias. Increased tolerance for ischaemia means that cells would be able to withstand otherwise damaging effects of bubbles. HSP70 is known to stabilise different proteins responsible for maintaining homeostatic conditions.[17] Since preconditioning with heat shock causes elevated levels of HSP70, cells can initiate the protective HSP70 mechanisms immediately when subsequent cellular trauma, such as ischaemia, occurs. At the same time, we should consider other circulatory effects of heat shock, for instance that vasodilatation could theoretically lead to a wash-out of nuclei and hence reduced bubble load. This should, however, lead to fewer bubbles, which is not the case in this study.

A similar study, in which rats were exposed to the same heat treatment as in our study, but without a following dive, showed an increase in HSP70, HSP90 and eNOS in heat-shocked animals compared to controls.[5] These results are inconsistent with our results, which imply no change in eNOS and HSP90 after a dive compared to non-diving controls, with or without the pretreatment with heat. A possible explanation is that the differences between preheated rats and controls were too small to be observed.

Massive bubbling, as observed in this study, is known to cause extensive endothelial injury.[18] If HSPs and eNOS are mainly expressed in the endothelial cells of the vessels, the explanation of our findings of no changes in HSP90 and eNOS could be that the tissue samples are stripped of endothelium.[19] The bubbles could cause disruption of the endothelial cell membranes, allowing proteins of the cytoplasm to be washed out into the blood stream, leaving only parenchymal cells to be analyzed in the tissue samples. It is conceivable that the dive itself is capable of inducing a stress response causing increased expression of HSPs and eNOS, but in this study the rats in the diving control group showed no increase in either compared to the two non-diving control rats. Again, this could be due to endothelial injury, masking a possible stress response in the diving controls.

Conclusion

We conclude that heat shock induced a stress response as evidenced by the expression of HSP70. Prior heat shock reduced mortality but through a different mechanism to that of exercise, as exercise has been shown to also decrease bubble formation. Although we cannot conclude that the protective effect is caused by HSP70 we believe that HSP70 is part of the protective mechanism, either as a signal transduction factor, or as a more central component.

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Differential effect of high pressure on NMDA receptor currents in X*enopus laevis* oocytes

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Key words
High pressure neurological syndrome (HPNS), neurophysiology, hyperbaric research

Abstract

(Mor A, Levy S, Hollmann M, Grossman Y. Differential effect of high pressure on NMDA receptor currents in X*enopus laevis* oocytes. *Diving and Hyperbaric Medicine*. 2008; 38: 194-196.)
Hyperbaric environments over 1.1 MPa induce in mammals and humans the high pressure neurological syndrome (HPNS). HPNS is characterized by cognitive and motor decrements associated with sleep disorders, EEG changes, tremor, and convulsions. Previously, it was proposed that augmented responses of the glutamatergic *N*-methyl-D-aspartate receptor (NMDAR) may be involved. Recently, we have reported that, in rat hippocampus brain slices, isolated NMDAR synaptic responses were augmented at high pressure. We now test how high pressure affects NMDAR ionic currents. Mammalian (rat) cRNA of the most common hippocampal NMDAR compositions NR1-1a / NR2A and NR1-1b / NR2A were injected into *Xenopus laevis* oocytes, and NMDAR ionic currents were recorded, applying the two-electrode voltage clamp technique, at normobaric and at hyperbaric pressure (10.1 MPa). Statistical analysis revealed that high pressure increased NR1-1a / NR2A current amplitudes. By contrast, high pressure decreased NR1-1b / NR2A current amplitudes. These preliminary results demonstrate a differential high pressure effect on two types of NMDAR subunits. Moreover, augmentation of the NR1-1a / NR2A composition further supports the recently reported increase of NMDAR synaptic response in rat hippocampus brain slices. These results support the notion that increased NMDAR response, in addition to other mechanisms, plays an important role in HPNS.

Introduction

Humans and mammals exposed to 1.1 MPa and above may develop the high pressure neurological syndrome (HPNS).[1] HPNS signs and symptoms include dizziness, nausea, stomach cramps, vomiting, muscle twitching and tremors, EEG changes, and a reduction in cognitive function.[2,3] At greater depths, myoclonia, convulsions, and seizures occur.[4,5] Neuropharmacological studies at high pressure suggested an increase in excitatory N-methyl-D-aspartate receptor (NMDAR) responses in CA1 pyramidal cells.[6,7] In the same brain region, our recent electrophysiological studies showed a significant increase in the synaptic NMDAR response followed by postsynaptic excitability changes.[8,9] These studies of field potentials suggest an increase in NMDAR ionic currents. However, no work has been done on direct measurement of identified NMDAR currents at high pressure. NMDAR is a hetero-tetrameric receptor-ion channel constituted of different combinations of 'NR1' with at least one 'NR2' subunit. NR1 subunit has eight alternative splicing isoforms: NR1-1a, b; NR1-2a, b; NR1-3a, b; NR1-4a, b (according to Hollmann et al terminology).[9] The NR2 subunit has four genes: NR2A, B, C, D.[10] NMDAR is activated by the co-agonists glutamate and glycine simultaneously with the removal of Mg^{2+} blockade by membrane depolarization. These, in turn, gate the cationic channel that is permeable to Na^{+}, K^{+} and Ca^{2+}. A considerable proportion (approximately 11 %) of the current is carried by Ca^{2+}.[11]

To date, there are incomplete data on the NMDAR subunits' spatial distribution and function(s) in the adult mammalian brain. For example:

- the NR1-1a isoform occurs extensively and approximately homogeneously throughout rat brain grey matter.
- the NR1-1b variant is found primarily in the sensorimotor cortex, thalamus, hippocampal CA3 field, and cerebellar granule cells.[12]
- the NR2A subunit is expressed widely throughout the whole adult rat brain.[13]
- the composition of the NMDAR subunits may change during development.[10]
- the deactivation rate is roughly four times faster for NR1-1b / NR2B receptors than for the NR1-1a / NR2B receptors and, therefore, may be involved in long-term synaptic modulation and learning.[14]

These examples reveal a large diversity of NMDAR roles in the mammalian brain.

The goal of the present study is to examine the NMDAR component alone without the contribution of the central nervous system (CNS) network. We aim at examining directly all available NMDAR combinations. We report here our preliminary results of high-pressure effects on the currents of the two abundant and important NMDAR combinations NR1-1a / NR2A and NR1-1b / NR2A.

Methods

OOCYTE PREPARATION

Animal experiments were carried out in accordance with the guidelines laid down by the Ben-Gurion University of the Negev ethics committee for the care and use of animals for

experimental work. *Xenopus laevis* oocytes were prepared and maintained in NDE96 solution (at 18^{O}C) containing (in mM): 96 NaCl, 2 KCl, 1 $MgCl_2$, 1 $CaCl_2$, 2.5 sodium pyruvate, 5 HEPES (pH 7.5) and 50 $\mu g \cdot ml^{-1}$ gentamycin. The oocytes were injected with cRNA for co-expressions of rat NR2A (5 ng) with either NR1-1a or NR1-1b, (5 ng) subunits (produced by Prof. M Hollmann's laboratory).[15] After 3–5 incubation days individual oocytes were placed in a recording chamber specifically modified for oocyte experiments and perfused (7–8 $ml \cdot min^{-1}$) with a frog physiological solution without Mg^{2+}. Solutions were introduced to the pressure chamber by means of a high-pressure pump ('mini-pump', LDC Analitical Inc, Fl, USA).

NMDAR CURRENT RECORDINGS

Oocytes were voltage-clamped at -70 mV employing the two-electrode voltage clamp technique using an Axoclamp-2B amplifier, (Molecular Devices, Axon Instruments Inc, CA, USA). The co-agonists glutamate (100 µM, Sigma, Israel) and glycine (10 µM, Sigma, Israel) were applied to the physiological solution; exposure duration was 20 s. NMDAR currents were acquired under control (0.1–0.3 MPa) and hyperbaric (10.1 MPa, compressed helium) conditions, and analyzed off-line. Recovery at 0.1 MPa was always attempted. Temperature was kept constant at 25 ± 1^{O}C. The pressure chamber, perfusion system, helium compression, and the experimental setup have been described in detail elsewhere.[8]

DATA AND STATISTICAL ANALYSIS

Under the experimental conditions noted above, NMDAR current (in nA) was composed of two peaks. The first, relatively fast peak probably reflects current flowing through the oocyte's native Ca^{2+} dependent Cl^- channels.[16,17] The second peak represents NMDAR cationic maximal inward current amplitude. Therefore, only the second peak was measured and analyzed. The results of maximal current amplitude are expressed as mean amplitude ± 1 standard error of mean (SEM); n denotes the number of successful experiments (number of oocytes) for each experimental protocol. In each experiment, control and hyperbaric conditions were tested on the same oocytes. We used paired samples *t*-tests for analysis, assuming electrophysiological recordings meet the conditions of a normal distribution. The degree of significance was denoted by the values of *P*; results were considered statistically different when $P < 0.05$. All statistical data were analyzed using SPSS 13.0 (SPSS Inc, Chicago, IL, USA). Graphical representations were made by using Microsoft Office Excel 2003 (Microsoft Inc, Redmond, WA, USA) and SPSS 13.0.

Results

NMDAR currents were blocked at normal pressure by 2 mM Mg^{2+} and by 20 µM DL-2-amino-5-phosphonopentanoic acid (AP-5, Tocris, Bristol, UK), confirming NMDAR responses (data not shown). High pressure increased NR1-1a / NR2A current amplitude by 42.7 ± 17.7 % (n = 11, P = 0.04). By contrast, high pressure decreased NR1-1b / NR2A current amplitudes by 11.6 ± 8.4% (n = 10, P = 0.003) (Table 1, Figure 1). The NMDAR currents exhibited only partial recovery (Figure 1). In the case of NR1-1a / NR2A, out of 11 attempts, eight showed at least partial recovery (decreased responses), and three did not recover. In the case of NR1-1b / NR2A, out of 10 attempts, six recovered (increased responses), one failed to recover, and data were not available for three.

Discussion

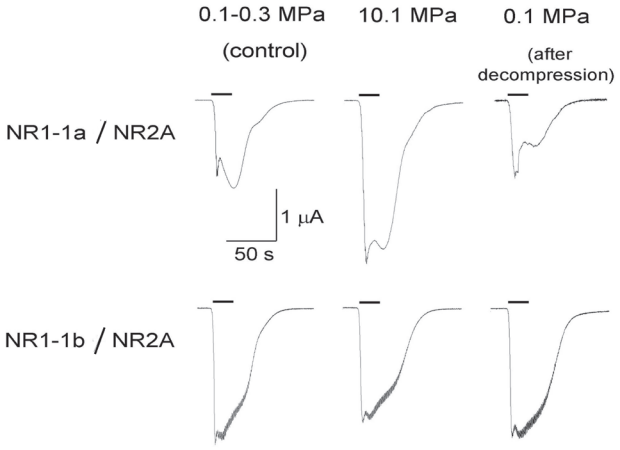
Our results reveal that high pressure selectively increases the NR1-1a / NR2A but reduces the NR1-1b / NR2A current amplitude. This pressure selectivity may point out important structural differences between the two NR1 subunits. However, it remains to be examined whether this difference is consistent for all other NR2 subunits and other NR1 1a/1b splice variant pairs. Another known difference is that NR1-1b / NR2B combinations produce currents with faster kinetics in comparison to the NR1-1a / NR2B combinations.[14] We assume that this is the reason for the greater proximity of the first peak (Ca^{2+} dependent Cl^- current) and the maximal NMDAR current (cationic current) peak recorded from the NR1-1b / NR2A combination in our experiment. The presence of the Cl^- current may distort to some extent the shape of the overall NMDAR current. At this stage we did not attempt to systematically separate the two components (e.g., by replacing Ca^{2+} with Ba^{2+}), in order to keep as close as possible to physiological conditions.

The NR1-1a is the most common NR1 subunit in the mammalian CNS.[18] Therefore, pressure effects on this subunit may determine the NMDAR pressure response in the CNS. These preliminary results are in accordance with our previous studies of pressure augmentation of NMDAR

Table 1
Statistical analysis of the NMDAR currents; n – number of experiments (oocytes); *P* – degree of statistical significance; SEM – one standard error of mean

Subunits composition	Amplitude (nA) Mean ± SEM 0.1 – 0.3 MPa	Amplitude (nA) Mean ± SEM 10.1 MPa	Amplitude (% change) Mean ± SEM 10.1 / 0.1 – 0.3 MPa	n	*P*
NR1-1a / NR2A	648 ± 141	925 ± 231	+42.7 ± 17.7	11	0.04
NR1-1b / NR2A	1858 ± 189	1561 ± 201	-11.6 ± 8.4	10	0.003

Figure 1
High pressure differentially modulates NMDAR currents in *Xenopus laevis* oocytes. NMDAR subtype NR1-1a / NR2A is increased and NR1-1b / NR2A decreased. The application of agonists (see text) is indicated by horizontal bars (20 s). The high-pressure effect is reversed after decompression.



synaptic responses in rat hippocampal (CA1) brain slices.[8,9] It is worth mentioning that an earlier study on NMDAR expressed in oocytes (isolated mRNA from rat cerebellum) exhibited pressure-increased responses.[19]

Conclusions

Our data support the postulated NMDAR involvement in HPNS hyperexcitability; however, they indicate a selective role for specific combination(s) of the receptor subunits. It is important to note that the NMDAR hyperactivity is only one factor in a multifactorial model for HPNS that may include reduced inhibition, synaptic frequency modulation, and dendritic boosting mechanisms.

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Short communication

Post-training dive inactivity in Western Australia

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Key words
Recreational divers, diver numbers, survey, questionnaire

Abstract
(Buzzacott P, Pikora T, Rosenberg M. Post-training dive inactivity in Western Australia. *Diving and Hyperbaric Medicin*e. 2008; 38: 197-199.)
Introduction: A lack of recent diving experience is often cited as relevant in analyses of diving fatalities. The purpose of this study was to measure diving inactivity amongst trained scuba divers within Western Australia and to identify reasons for extended diving inactivity.
Methods: In March 2005, self-administered surveys were mailed through 22 diving training centres to Western Australians who had completed a scuba diving course within the previous five years (n = 2,077).
Results: Three-hundred-and-five of 505 returned questionnaires were suitable for analysis. Within two years of completing scuba training, one in five divers had not dived for at least one year, with one half of these making only one post-course dive. Compared with active divers, inactive divers were younger, more likely female, owned less dive gear, were less likely to own a boat and completed fewer additional dive courses. Reasons cited for not diving were a lack of time, diving equipment, someone to dive with and/or money.
Conclusion: Divers likely to spend extended periods between dives, without intending to permanently give up diving, may benefit from additional support during their first post-course year.

Introduction

One widely adopted schema for safe diving practice states: "*Keep proficient in diving skills,… reviewing them in controlled conditions after a period of diving inactivity,..*".[1] There is evidence that a proportion of divers do not follow this advice. A Texas study of divers aboard a dive boat classified divers as 'active' if they had made one or more dives during the previous year, but of 528 divers surveyed just 461 (87%) reported making dives during the previous year, suggesting 13% of the certified divers on their way to a popular dive site were returning after an extended break.[2]

Why divers discontinue diving for extended periods is of particular importance to the recreational diving industry. Of 159 Australian recreational diving fatalities occurring amongst certified divers in open water between 1978 and 2002, 21% (n = 34) were noted as having died whilst returning to diving after an extended break.[3–9] Of these divers, 59% (n = 20) were also noted to have been inexperienced, suggesting many inactive divers commence their extended break soon after they complete their training.

If the proportion of divers in Texas found returning to diving after an extended break were to be representative of the Australian experience, we might consider returning divers are over-represented in diving-related fatality reports. For this reason, it is important to investigate the reasons why Australian divers may have absences from diving. Previous studies in both Australia and the USA suggest these reasons include a lack of time, money, diving equipment, dive buddies and/or local dive sites.[10–12]

Methods

A cross-sectional survey of recreational divers certified within the previous five years was conducted between March and September 2005. The research was approved by the University of Western Australia Human Research Ethics Committee. The self-administered questionnaire collected information on frequency of diving since completing a dive certificate, demographics and reasons for not diving if no dives were reported during the previous 12 months (defined as 'inactive'). Divers were also asked to report ownership of eight types of dive gear: mask/snorkel/fins, wetsuit/weight-belt, dive watch, dive computer, safety sausage, regulators/gauges, buoyancy control device and scuba cylinder. Validity was assessed during questionnaire development and pilot tested with 20 Western Australia (WA) dive club members.

Thirty-nine dive businesses listed in telephone directories current in WA at the commencement of the study indicated they trained divers and, of these, twenty-two (63%) mailed surveys to their customers on behalf of the research team, ensuring client confidentiality. To determine if respondents differed significantly from the population of interest, two dive centres supplied the age and gender for each diver mailed a survey.

Table 1
Gender and age distributions for two dive centres and respondents

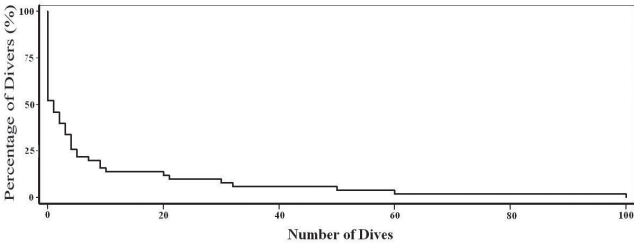
	Two dive centres	Respondents	Two dive centres age at certification	Respondents age at certification
	n (%)	n (%)	Years (SD)	Years (SD)
Males	248 (71%)	350 (70%)	29.8 (+/- 11.7)	31.2 (+/- 11.2)
Females	101 (29%)	126 (25%)	29.5 (+/- 10.4)	29.2 (+/- 10.4)
Total	349 (100%)	476 (95%)*	29.7 (+/- 11.3)	30.6 (+/- 11.0)

* Twenty-three values (5%) missing

DATA ANALYSIS

The data were analysed using SAS ver. 8.02 (SAS Inc, North Carolina). Reported univariate P values are the result of Wilcoxon rank sum test for difference in mean age and Fisher's exact tests for categorical variables.[13] Odds ratios (OR) are reported with 95% confidence intervals (CI). Variables were fitted to a logistically transformed, general linear model, and backwards elimination used to remove least significant effects. Significance was accepted at $P < 0.05$.

Figure 1
Number of post-course dives before an extended break from recreational diving



Results

Of 2,077 surveys mailed to certified divers within WA, 108 were returned unopened, marked "not at this address", and 505 were returned completed. Of those 505, six were completed by divers living outside of WA and 194 by divers trained for less than a full year, resulting in 305 useable surveys and a response rate of 17% (305/1,769). Of the 305 divers living in WA who had been trained for more than one year at the time of survey, 85% (n = 259) were active divers and 15% (n = 46) had not dived for at least 12 months. One half of inactive dive course graduates reported commencing an extended period of diving inactivity immediately following their course and 75% before making six post-course dives (Figure 1).

CHARACTERISTICS OF DIVERS

Divers who were inactive during the year prior to the survey were significantly younger when certified than those classed as active (32.8 years, range 12.7–64.8, versus 27.6 years, range 14.2–52.1, P = 0.003). These divers were also significantly more likely to be female (OR 3.54, 95% CI 1.85, 6.75), although female divers made, on average, the same number of dives as males before commencing an extended break. There was no significant difference between the 349 training records supplied by two dive centres and the 499 WA respondents in either mean age at certification (P = 0.24) or gender distribution (P = 0.31) (Table 1).

Compared with active divers, inactive divers were more likely to report owning fewer than two types of gear (median 1 versus 6, $P < 0.0001$) and less likely to have taken any additional dive courses (6.5% versus 35.9%, $P < 0.0001$). No significant differences were found between active and inactive divers on the basis of height, weight or body mass index (BMI), the number of standard alcoholic drinks reportedly consumed per week by either males (P = 0.40) or females (P = 0.33), or smoking status (P = 0.09). Active divers were, however, more likely to report owning a boat (OR 2.4, 95% CI 1.2, 4.9).

Fitting the variables to a logistic model and eliminating least significant effects (smoking status (P = 0.76), BMI (P = 0.48), alcohol consumption (P = 0.40) and age (P = 0.28)) the predictors of diving inactivity were owning fewer than two items of dive gear (OR 10.1, 95% CI 4.6, 22.5), the lack of additional training (OR 8.6, 95% CI 2.4, 30.5), being female (OR 3.0, 95% CI 1.4, 6.3), and not owning a boat (OR 2.3, 95% CI 1.0, 5.2). For the 46 inactive divers, the reasons why they had not dived during the previous year are listed in Table 2. Multiple reasons were given by many of them. Reasons grouped as 'other' included low confidence

Table 2
Reasons given for diving inactivity

Reason	n (%)
No time	32 (70)
Lack of equipment	24 (52)
No dive buddy	20 (43)
Cost	19 (41)
Medical/health	12 (26)
Other	9 (20)

diving without a refresher course, geographical location and poor motivation.

Discussion

This survey suggests most divers taking extended leave from the sport do so within one or two post-certification dives and report having too little time, no equipment and/or no dive buddies. These reasons are similar to those provided by other inactive diving populations though we did not explore causality in this study.

The significant limitation of this study was a response rate of only 17%. For mail surveys, response rates of between 10% and 40% are common, but rates higher than 50% are desired before non-response bias becomes less of a concern.[14,15] Contacting non-responders multiple times, and/or using multiple modes of contact such as mail, telephone and e-mail may have helped but, as the survey was anonymous, participating dive centres were unable to determine which divers had responded and which had not, so divers were only contacted once. Although no significant differences were found between the 349 training records supplied by two dive centres and the 305 respondents with useable data in either mean age at certification or gender distributions, respondents may differ from the rest of the WA recreational diving population in other, unidentified ways. Accordingly the results of this study should be interpreted with caution.

Recent diving inactivity is often cited as a relevant factor in analyses of diving fatalities. It is likely, however, that many divers do not return to diving after a year or more passes since their last dive. Research identifying whether divers who permanently leave the sport differ from temporarily inactive divers would be valuable if methods to encourage engagement in the sport beyond the first two dives could reduce the likelihood of divers (who do not intend giving up diving altogether) taking early, extended, yet temporary periods away from diving. This might include supporting multiple dives within the first year following training, incorporating organised dive trips with equipment hire and additional training.

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Review article

Genotoxicity of hyperbaric oxygen and its prevention: what hyperbaric physicians should know

Michael Gröger, Peter Radermacher, Günter Speit and Claus-Martin Muth

Key words
Toxicity, hyperbaric oxygen, oxygen, stress, diving, review article

Abstract

(Gröger M, Radermacher P, Speit G, Muth C-M. Genotoxicity of hyperbaric oxygen and its prevention: what hyperbaric physicians should know. *Diving and Hyperbaric Medicine*. 2008; 38: 200-205.)
Hyperbaric oxygen (HBO) therapy is used for the treatment of a variety of diseases, but also leads to oxidative stress as a result of increased formation of reactive oxygen species. The consequences may be damage to the lung, the central nervous system and the genome. The oxidative attack on DNA causes, among other damage, single and double strand breaks. Using the comet assay, a well-established genotoxicity test, it was possible to show that a single HBO exposure leads to increased levels of DNA strand breaks in a close dose-effect relationship. On the other hand, it was possible to demonstrate that these strand breaks are repaired rapidly and that, in repeated HBO exposures, DNA strand breaks occur only after the first treatment, not subsequent ones, indicating an induction of protective mechanisms. In healthy organisms, DNA repair and antioxidant mechanisms maintain a steady-state level of damage with minimal risk to the cell or the whole organism, but it cannot be excluded that HBO might lead to a significant mutational burden in situations where antioxidant defence is deficient or overwhelmed. The administration of antioxidants draws an ambivalent picture; Vitamin C, E or even N-acetylcysteine seems to be ineffective to prevent HBO-induced genotoxicity, whereas the orally effective vegetal superoxide dismutase (SOD, Glisodin®) is effective, and, thus, may play a role in the prevention of oxidative DNA damage.

Introduction

Hyperbaric oxygen (HBO) therapy comprises inhalation of 100% oxygen at supra-atmospheric ambient pressure. HBO has been successfully used for the treatment of a variety of diseases such as decompression illness, acute carbon monoxide intoxication, gas embolism, soft tissue infections, radiation necrosis and impaired wound healing (e.g., in the context of 'diabetic feet'). However, besides its beneficial effects, HBO may also have deleterious effects, not only on the central nervous system (Paul Bert effect) and on the lung (Lorrain-Smith effect)[1], but also on the genome. It is well known that prolonged exposure to normobaric hyperoxia induces DNA damage.[2] Hence, the induction of DNA damage during HBO is a matter of interest, in particular to the consequently raised question whether HBO has a cancer-promoting effect.[3,4]

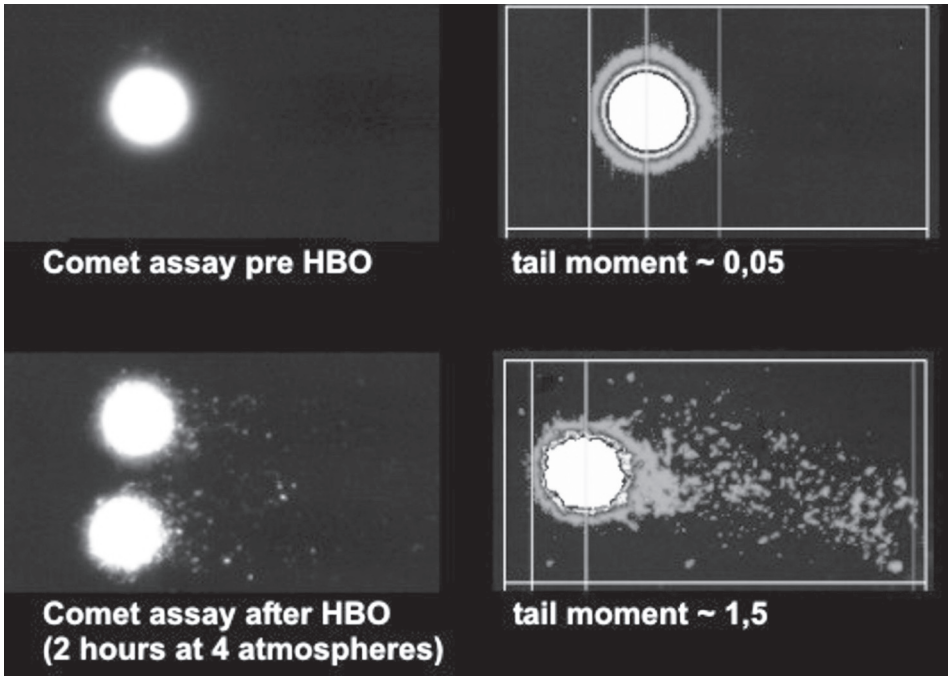
The harmful effects of high oxygen concentrations are due to the abundance of oxygen free radicals, which possess one ore more unpaired electrons.[1,5,6] The collective term 'reactive oxygen species' (ROS) comprises free radicals like O_2° and HO° as well as non-radical oxygen derivates like hydrogen peroxide (H_2O_2) and peroxynitrite ($ONOO^-$).[5,6] ROS are unstable and react with all kinds of cellular compounds, which may result in lipid, protein and DNA damage.[1,5–8] This effect is particularly pronounced in situations of reduced antioxidant defences. Conditions in which ROS production is higher than elimination are called 'oxidative stress', no matter whether they originate from increased ROS formation or decreased elimination.[1,9,10]

Among other cellular structures, the genome is particularly vulnerable, and the possible results of the oxidative attack on DNA are single- and double-strand breaks, abasic sites, 'alkali-labile' sites and oxidized bases.[8,10,11] This, in turn, can lead to mutations, if the lesions are not adequately repaired. The ultimate consequence can be the initiation or the progression of cancer, if specific genes like tumour suppressor genes or oncogenes are affected.[3,12,13] Therefore, there has been worry about the cancer-causing effect of HBO, albeit the available literature does not show any clear evidence for this.[4]

Detection of DNA damage

DNA-damage comprises modifications of DNA bases or damage to the backbone, such as strand breaks. Various methods for the detection of DNA damage are in use, such as [^{32}P]-post-labelling, alkaline unwinding, alkaline elution or the so-called 'comet assay', also known as single-cell gel electrophoresis. The comet assay detects strand breaks on the single cell level and is a simple, fast, sensitive and well-established genotoxicity test.[14,15] The comet assay is a

Figure 1
Comet assay with isolated lymphocytes before and after hyperbaric oxygen (HBO) therapy



microgel electrophoresis technique, using a small sample of cells suspended in a thin agarose gel. The sample is lysed, electrophoresed and stained with a fluorescent DNA-binding dye whilst on a microscope slide. DNA damage can be analysed by image analysis. Nuclei with increased strand breaks show increased DNA migration in the electric field, and this resembles the shape of a comet (Figure 1).

Several parameters can be quantified for determining the length and amount of DNA migration. The most frequently used parameters are 'tail intensity' (% tail DNA) and 'tail moment' (a product of both length and intensity). DNA migration is mainly induced by DNA single-strand breaks, DNA double-strand breaks and alkaline-labile sites (e.g., abasic sites). Though sensitive, the comet assay is not specific with regard to the genetic relevance of the observed effects. Different kinds of genotoxic effects can cause increased DNA migration, and further information is needed to evaluate biological significance. Such additional information can be derived by using lesion-specific endonucleases, e.g., formamidopyrimidine-DNA-glycosylase (FPG), which detects and cuts out specific oxidized bases, producing additional strand breaks.[16]

The comet assay can be performed with virtually any eukaryote cell population *in vitro* and *in vivo*. Various tissues can be comparatively investigated such as whole blood, isolated lymphocytes, liver, lung, heart and kidney. In addition, the assay is highly sensitive for even low levels of DNA damage and requires only small samples. The comet assay has already been used in many studies to assess DNA damage induced by various agents in a variety of cells *in vitro* and *in vivo*. The test has widespread application in genotoxicity testing, environmental biomonitoring and human population monitoring.

DNA repair and mutagenesis

The comet assay not only detects DNA damage but also enables an investigation of DNA repair. For this purpose, the time-dependent removal of induced lesions, i.e., the decrease in DNA migration, is monitored. Using the comet assay, HBO-induced DNA migration in healthy young male volunteers, who had been exposed to a therapeutic HBO treatment protocol (253 kPa for a total of three 20-minute periods of pure O_2 breathing, interspersed with five-minute periods of air breathing), was shown to be reduced by more than 50% within the first hour after exposure. However, it also showed that blood taken six or 24 hours after HBO no longer showed increased migration, indicating fast and complete repair of the HBO-related DNA damage.[17]

It must be noted that the comet assay measures only the kinetics of strand break repair but not its accuracy. Thus incorrectly repaired lesions do not contribute to migration but may still have a mutagenic potential. Therefore, in order to investigate the biological significance of the effects of HBO shown with the comet assay in humans, the micronucleus test (MNT) was performed. The same blood samples that showed a significant rise in DNA migration in the comet assay did not exhibit increased micronucleus frequencies.[17] Although the effects in the MNT are limited to proliferating lymphocytes, this observation demonstrates that the genotoxic effects occur in the whole population

of white blood cells. Therefore, it can be presumed that, under therapeutic exposure conditions, the primary DNA damage is repaired before the cells enter the mitotic S-phase and chromosome aberrations can be produced. In fact, no evidence of the induction of chromosome damage was found after this single *in vivo* HBO exposure in lymphocytes from healthy human volunteers.[17]

Earlier investigations suggested increased frequencies of chromosome aberrations after HBO exposure. However, these results were found in patients with diverse diseases and drug treatments after repeated HBO exposures.[18] As the comet assay results indicate that repetitive HBO exposures do not induce further DNA damage, but rather induce adaptive protection, it is likely that the observed increased chromosome aberration rates are not directly associated with the HBO treatment.

In healthy organisms, efficient antioxidant mechanisms together with DNA repair maintain a steady-state level of damage with a minimal risk to the cell or the whole organism.[29] On the other hand, we cannot exclude HBO, on comet assay results alone, as an important cause of mutational burden in situations where antioxidant defence is deficient or overwhelmed.[18]

The use of cultured mammalian and human cells *in vitro* makes it feasible to increase HBO exposure in order to investigate the question whether HBO induces DNA damage under conditions where antioxidant capacities are overwhelmed.[19] The main advantages of these *in vitro* studies in comparison to *in vivo* exposure of human subjects are the possibility of a permanent O_2 exposure (i.e., without interspersed air breathing) and increased pressures. Using the comet assay, a genotoxic effect of HBO could be demonstrated in diverse cell types.[19] More intense HBO exposures using both higher pressure and longer duration than for therapeutic use of HBO clearly caused mutagenic effects in cultured mammalian cells *in vitro*.[20,21] A correlation between O_2 partial pressure, exposure time and the frequency of chromosome aberrations was found in V79 cells (a permanent Chinese hamster cell line) using the MNT.[20,21] The clastogenic (chromosome-breaking) effect of the treatment in this cell line was directly related to the rise in DNA damage assessed with the comet assay. Increased HBO exposure also elevated mutant frequencies in a mammalian cell gene mutation assay at the tk-locus of mouse lymphoma cells.

In contrast to this, HBO failed to provoke mutations in the *in vitro* hypoxanthin-P-ribosyl-transferase test (HPRT test) with V79 cells (which mainly detects point mutations).[20,21] This negative finding suggests that, even under intensive exposure conditions, HBO does not significantly produce point mutations but mainly acts via a clastogenic mechanism. Consequently it is likely that after HBO exposure, reactive oxygen species develop their mutagenic potential through DNA lesions like single- and double-strand breaks, with gross deletions and chromosomal effects following, as a result of incomplete and incorrect repair. This clastogenic mechanism has also been proposed for normobaric hyperoxia, which induced comparable mutagenic effects *in vitro*. Taken together, the *in vitro* studies clearly prove that HBO with long exposure times or high pressure has the potential to induce mutations via a clastogenic mechanism.

One of the crucial mediators of HBO-induced DNA damage seems to be nitric oxide (NO). The release of NO is tightly regulated by the protein heme oxygenase-1 (HO-1) and an increased formation of NO *per se* caused DNA strand breaks no matter whether NO release was a result of administration of NO donors like molsidomine or due to cytokine stimulation.[22–25] The genotoxic properties of NO are presumed to be caused by the generation of peroxynitrite from NO and superoxide under conditions associated with increased release of these two molecules.[26] On the other hand, elevated DNA damage observed in other studies was not related to the blood nitrate concentrations.[27,28] It has also been noted that NO has both anti- and pro-oxidant properties depending on the local milieu, and both increased and decreased NO production has been reported during HBO exposure.[29,30]

Protection against HBO-induced DNA damage

As mentioned above, HO-1 plays an important role in protection against oxidative DNA damage. Lymphocytes from healthy volunteers showed significantly increased HO-1 concentration after HBO exposure both *in vivo* and *in vitro*.[31,33] Moreover, HO-1 over-expression significantly reduced the HBO-induced DNA damage in V79 cells *in vitro,*[33] whereas the inhibition of HO-1 with sn-mesoporphyrine aggravated the HBO-related genotoxicity and completely reversed the adaptive protection against HBO-induced DNA damage, again both *in vitro* and *in* vivo.[22,32,34]

The typical therapeutic HBO regimen comprises repeated HBO treatment over several days. Because it was found that a single HBO treatment induced DNA damage in healthy volunteers,[35] and there is the already mentioned close dose-effect relationship concerning both duration and pressure,[20] it was supposed that repetitive therapeutic HBO treatments may lead to a significant accumulation of DNA damage which might cause a significant mutagenic risk. However, it has been shown that human volunteers undergoing repeated HBO exposures exhibited DNA damage only after the first treatment, but not after any subsequent exposure.[17,36] In fact, the number of DNA strand breaks after repeated HBO exposures was even lower than in the initial blood sample taken before the first HBO.[17,36]

Another interesting finding is the fact that a lower initial dose of HBO (20 min at 153 kPa (1.5 bar)) did not induce any

DNA strand breaks but was associated with the induction of adaptive mechanisms that protected against further HBO-induced DNA damage.[17,36] Subsequent studies have shown that the adaptive effect is due to a cellular response that cannot be explained by enhanced repair activity and seems to be a consequence of either increased scavenging of oxygen species distant from nuclear DNA or enhanced sequestration of transition metals.[17,36]

The role of antioxidants for DNA protection

In the course of evolution, oxygen-consuming organisms have developed a variety of defence mechanisms against oxidative stress. Several enzymes show strong antioxidative properties, e.g., superoxide dismutase (SOD), which catalyses the dysmutation reaction of the superoxide radical.[5,37] The product of this reaction is H_2O_2, which in turn is either catalysed into water and molecular oxygen by the enzyme catalase, or removed by glutathione peroxidase (GPx).[5,38] GPx catalyses the reaction of two molecules of reduced glutathion (GSH) and H_2O_2 to the oxidized form GSSG and two molecules of water.[5,9,39]

Besides enzymes, vitamins (e.g., vitamin E and C) play an important antioxidative role.[38–40] Vitamin C has two functions. Firstly, it is needed to restore Vitamin E located in lipoproteins and membranes, where it interrupts the radical-induced chain reaction of the lipid peroxidation, and, secondly, Vitamin C has radical scavenging properties of its own. Whether exogenous antioxidant supplementation prevents HBO-induced genotoxicity is still a matter of debate. Vitamin E and the synthetic antioxidant N-acetylcysteine did not affect the HBO-induced DNA damage in healthy volunteers,[40] but no data are available in patients with decreased antioxidant capacity. However, N-acetylcysteine attenuated the rise of blood lipid peroxidation markers in patients undergoing repetitive HBO treatment sessions.[41] Glisodin®, an orally effective nutritional formula containing a plant (*Cucumis melo L.C*) SOD extract, effectively protected white-blood-cell DNA against formation of strand breaks in healthy volunteers.[42] These results indicate that long-term prophylactic antioxidant supplementation may indeed attenuate HBO-induced DNA damage.

Diving and DNA damage

Given the well-established phenomenon of HBO-induced DNA damage one might assume that frequent diving might also influence DNA damage, either due to a possible induction of protective adaptive mechanisms or as a consequence of an increased sensitivity against increased oxygen partial pressure (ppO_2). Interestingly in a yet unpublished study with healthy male recreational scuba divers of at least four years' experience, including at least 50 dives per year at depths of more than 10m, our group found that isolated lymphocytes exposed to HBO (two hours, 405 kPa) did not show any difference in the induced tail moments compared to lymphocytes from non-diving volunteers of the same age. Subsequently we studied combat swimmers and underwater demolition team (UDT) divers.[43] These subjects perform dives over several years breathing pure O_2 and/or O_2-enriched inspiratory gas mixtures using closed and semi-closed breathing apparatus respectively. Thus, these divers represent a population with a particularly long-term repetitive exposure to increased ppO_2. Isolated lymphocytes from these groups were compared to those from both non-diving naval pentathlon athletes (chosen because they have a comparable degree of endurance training to the diver groups) and untrained controls of the same age following the same HBO regimen mentioned above. DNA repair was maintained over 2 hours after HBO exposure. As shown in Table 1 all groups showed a marked rise in the tail moment, which was, however, nearly twice as high in the combat swimmers as in the three other study groups. Nevertheless, in all groups, the increased tail moment returned to normal values within one hour after the HBO exposure, without any inter-group difference. Hence, combat swimmers who undergo particularly high and prolonged HBO exposures not only show the most pronounced HBO-induced DNA damage but also the most rapid and effective repair.[43]

Conclusion

The DNA damaging and mutagenic potential of HBO is not in dispute, as shown by *in vitro* studies with mammalian cells. DNA damage has been observed with therapeutic exposures, but mutations and chromosome aberrations were not detectable in blood cells under the same conditions.

Table 1
DNA-damage in isolated and HBO-exposed lymphocytes after long-term, repetitive exposure to increased ppO_2; data are mean (standard deviation); * depicts P < 0.05 versus control; § depicts P < 0.05 versus pre-HBO (from reference 43, with permission)

	pre HBO	post HBO	1 h incubation	2 h incubation
Combat swimmers (N = 7)	0.12 (0.03)	0.38 (0.09)*§	0.13 (0.04)	0.10 (0.01)
UDT divers (N =7)	0.10 (0.02)	0.24 (0.08)§	0.12 (0.03)	0.10 (0.02)
Navy pentathlon athletes (N = 6)	0.10 (0.02)	0.22 (0.05)§	0.10 (0.01)	0.11 (0.02)
Control (N = 24)	0.12 (0.04)	0.28 (0.14)§	0.13 (0.04)	0.14 (0.05)

Even if blood cells do not seem to be subject to an increased risk of (chromosome) mutations, mutagenic effects in other target cells cannot be completely excluded and, hence, the potential genotoxicity of hyperbaric oxygen should be taken seriously.

On the other hand, there is a fast repair of oxidative DNA damage as well as an adaptation to subsequent oxidative stress. Furthermore, a simple and efficient way to prevent organisms from HBO-induced DNA damage is to start with a shortened treatment before the standard protocol is applied, and therefore an adaptation of the commonly used treatment protocols should be considered.

The use of antioxidants such as vitamin C, E or even N-acetylcysteine seems to be ineffective in preventing HBO-induced genotoxicity. In contrast, the orally effective vegetal SOD (Glisodin®) protected against HBO-induced DNA damage and thus may play a role in the prevention of such damage. Finally it has to be pointed out that, regarding the effect of antioxidants, the available data refer to collectives of healthy and young volunteers, and no firm conclusions can be drawn for patients with a reduced antioxidative capacity (e.g., radionecrosis, chronic wound healing defects, chronic infection).

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Case report

Influence of scuba diving on asymptomatic isolated pulmonary bullae

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Key words
Pulmonary barotrauma, arterial gas embolism, fitness to dive, risk assessment, medical conditions and problems, case reports

Abstract

(Germonpré P, Balestra C, Pieters T. Influence of scuba diving on asymptomatic isolated pulmonary bullae. *Diving and Hyperbaric Medicine*. 2008; 38: 206-211.)
Pulmonary blebs and bullae are generally considered an absolute contra-indication for scuba diving, because of a high estimated risk of pulmonary overpressure syndrome due to air-trapping inside the bulla. This is primarily based on a number of retrospective studies and case reports; formal prospective evidence of a higher risk is lacking. We present three cases where a pulmonary bulla was radiographically shown to increase in diameter, seemingly related to scuba diving activity, and causing ultimately a barotraumatic diving accident. These cases provide pathophysiological clues as to how even an isolated, non-ventilated bulla can be the cause of pulmonary barotrauma. The most likely mechanism for this phenomenon is a 'stretching' of the bulla upon ascent from the dive: after a period of compression (Boyle's Law), there is a gradual diffusion of air through the bulla wall, with restoration to its initial size by the end of the dive. Upon ascent, the air diffuses only slowly out of the bulla, causing a temporary increase in diameter and stretching of the bulla wall. This repeated stretching causes the bulla to grow gradually. At one point, the cyst wall may become critically thin and rupture during the ascent.

Introduction

Scuba diving has gained enormous popularity over the past two decades. With the development of reliable, comfortable diving breathing apparatus, diving has become accessible for people of all ages and physical fitness. The usefulness of a medical examination prior to engaging in recreational scuba diving is little disputed; however, the exact extent of such an examination is subject to much discussion. With regard to pulmonary function tests, there is evidence that abnormal flow-volume loops and compliance represent an increased risk for pulmonary barotrauma (PBT).[1] With regard to pulmonary blebs or bullae, the necessity to perform high-resolution imaging tests for detection has to be weighed against the estimated risk that such a bulla presents.

Asymptomatic pulmonary blebs can be found in a large number of persons, but no large series have been published to establish prevalence in a normal population.[2] Likewise, the prevalence of large bullae in a normal population is unknown. This makes a risk estimation practically impossible, since the denominator in the risk equation is missing. As PBT in recreational diving is fortunately a rare event (estimated between 1:19,800 and 1:34,000 dives),[3] the suggestion has been made that it is not justified to use CT scan screening for blebs in recreational or even professional divers.[2]

Retrospectively in divers with PBT, functional or anatomical abnormalities of the lung (scarring, emphysematous bullae or blebs) can be demonstrated in a number of cases using high-resolution (spiral) CT scanning.[4] These lesions are often undetectable on plain chest radiographs. Therefore, even if no large series exist that compare divers with PBT with controls, the presence of such structural anomalies is generally considered a contra-indication to diving, because of the risk of air trapping on ascent.[1]

In (large or small) ventilated bullae, air trapping can happen either on the basis of a one-way valve mechanism ('real' air trapping) or by a volume increase upon ascent because of a narrow inlet-outlet opening ('virtual' air-trapping). The risk of isolated, non-ventilated bullae is in most cases considered similar, although no plausible explanation is given. Some authors express their doubts on the presumed risk of isolated bullae, since pressure-volume mechanistic theory (Boyle's Law) would predict that these will never expand to greater than their initial volume.[5]

We describe three patients in whom isolated, non-ventilated pulmonary bullae were observed to increase in size during a period of three years of intense scuba diving. In two cases, this led to an episode of pulmonary overpressure syndrome with cerebral arterial gas embolism (CAGE). In one diver, the condition was followed up and during the next seven years of not diving, the bulla remained unchanged in size.

Case 1

Case 1 was a male born in 1942 with a 20-pack-year history of smoking (he stopped smoking in 1973). He

started recreational scuba diving in 1981, and performed approximately 1,200 uneventful dives over 16 years (about 75 dives per year in mostly cold water, to depths of 40–50 metres' sea water (msw)). In 1991, during a routine chest X-ray, a 35 mm diameter, thin-walled, asymptomatic bulla was discovered in the lower lobe of the left lung (Figure 1). On spiral CT scanning, there was no apparent ventilation orifice of this lung cyst. Ventilation-perfusion scanning was not performed. Routine pulmonary function testing, including flow-volume curve, was normal, and alpha-1-antitrypsin serum level was normal. He continued diving.

In April 1997, after an uneventful dive, he suddenly experienced severe general fatigue, headache, paraesthesia and mild paresis of both lower limbs. These symptoms recovered with normobaric oxygen administration; no hyperbaric treatment was given. Emergency chest X-ray upon arrival in hospital revealed a large bulla – approximately 100 mm in diameter. A repeat chest X-ray and CT scan six months later showed a reduction in the diameter of the cyst: 50 mm, thin-walled (Figure 1). He discontinued diving.

In 2002, because he wished to resume diving, a new chest X-ray and CT-scan were done, revealing a bulla diameter of 51 mm (Figure 1). A ventilation-perfusion scan showed an isolated, non-ventilated bulla. After discussion with a diving medical officer (DMO), he decided against resuming his diving hobby.

Summary of case 1: Asymptomatic bulla in left lower lung; increased diameter after six years of diving; hyperinflation of bulla immediately after diving accident with symptoms of CAGE; stable diameter after seven years of not diving.

Case 2

Case 2 was a female born in 1950, non-smoker, who had annual chest X-rays as part of occupational medicine checkups (she worked as a nurse in a respiratory ward). These were always classified as "normal" by the reviewing radiologist. She had received prophylactic anti-tuberculosis treatment after a positive Mantoux test in 1982.

She took up diving in 1988 and performed more than 100 dives per year for the next three years. In December 1991, after an uneventful dive, she experienced the following symptoms upon surfacing: mild chest discomfort, moderate dyspnoea, general fatigue, rigidity of neck and jaw, dysarthria, and marked coordination disturbances of the upper limbs with uncontrolled jerking when attempting to perform fine movements. These symptoms were initially attributed to cold, and no specific treatment other than

Figure 1
Case 1; serial chest X-rays and comparable CT slices taken over 11 years on the dates shown (see text for details)

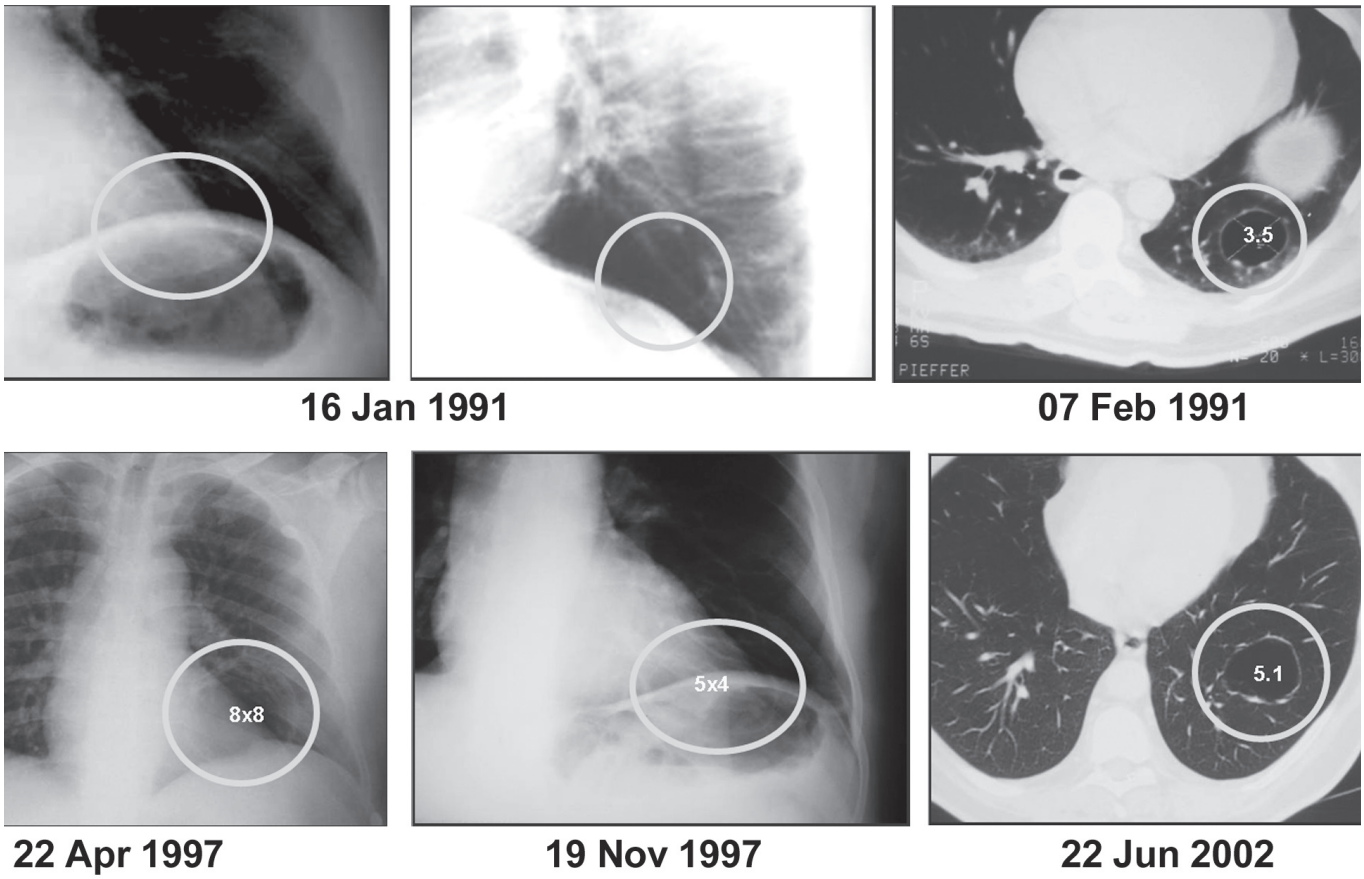
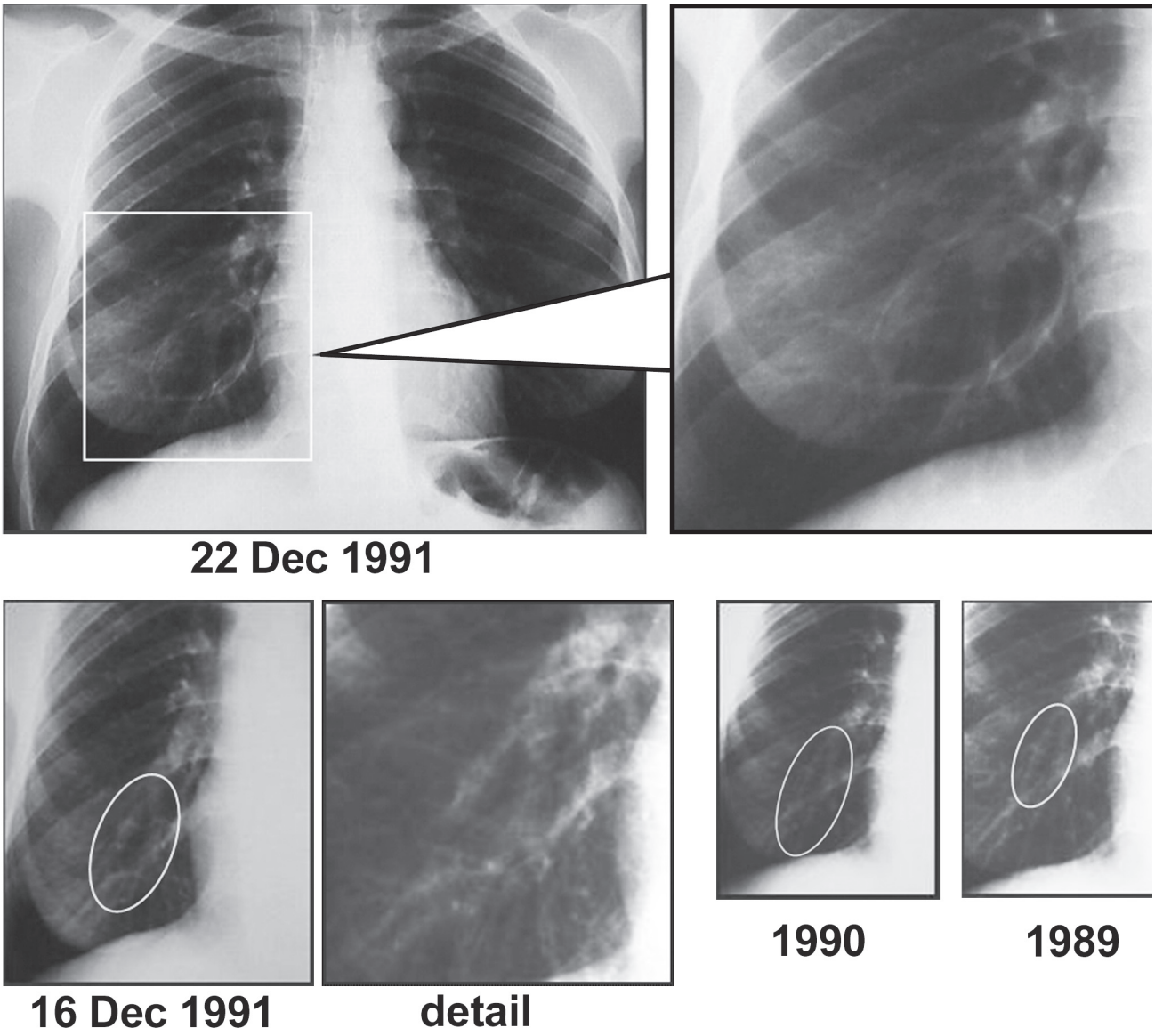


Figure 2
Case 2; serial chest X-rays taken over two years on the dates shown (see text for details)



rewarming was performed. She presented at the hospital 18 hours after surfacing, because these symptoms had taken longer than expected to resolve. She did not have major residual symptoms upon admission.

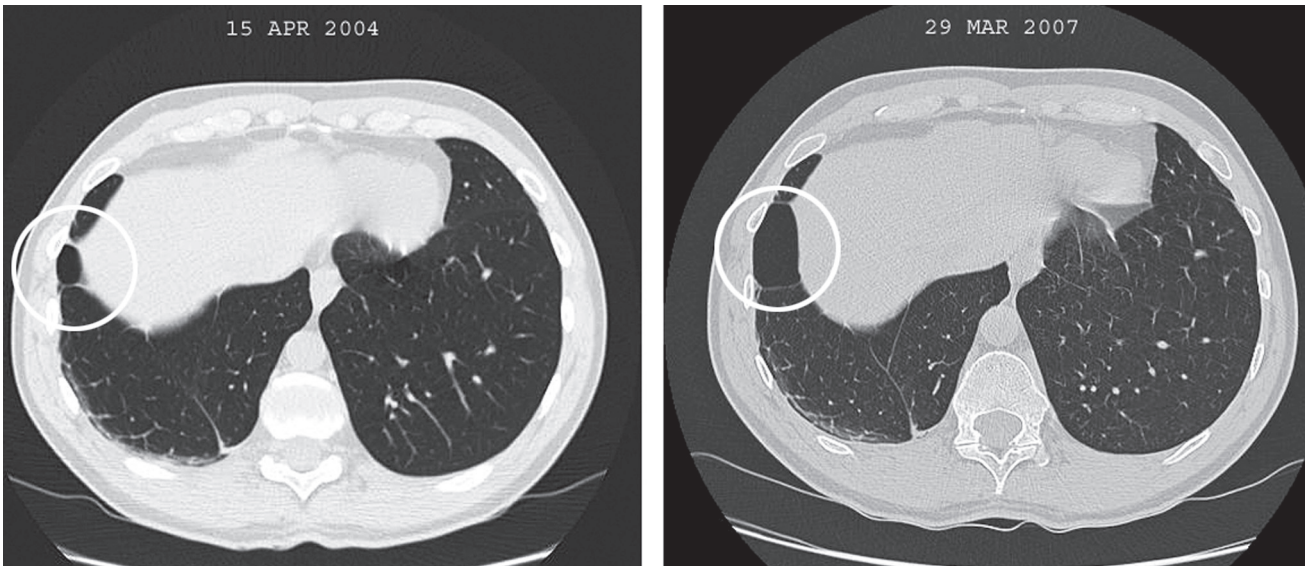
A chest X-ray revealed a large, spherical, thin-walled bulla in the right lower lung with a diameter of 70 mm (Figure 2). CT scan confirmed this to be a thin-walled cyst with no apparent ventilation orifice. A ventilation-perfusion scan showed no ventilation. She was not treated with hyperbaric oxygen, because of the absence of residual symptoms. A control chest X-ray one week after the incident showed a decreased diameter of the bulla, which was now partly collapsed and ellipsoid, measuring 6 by 3 cm. Serum alpha-1-antitrypsin level was normal. Routine pulmonary function testing, including flow-volume curve, was normal. A review of previous chest X-rays was undertaken (Figure 2):

- **16 December 1991** (one week prior to the accident): described as "normal". Upon careful review, the lung cyst can be seen as a faintly visible, flattened, ellipsoid measuring 60 by 30 mm.
- **1990**: classified "normal". Upon review the lung cyst is visible, but is notably smaller: 40 by 20 mm.
- **1989**: as above, 30 by 15 mm
- **1988**: as above, 20 by 10 mm

Following the DMO's advice, she stopped diving. A control chest X-ray three months later showed the bulla to be unchanged in size. She was then lost to follow up.

Summary of case 2: Asymptomatic bulla in right lower lobe; progressive increase documented with yearly chest

Figure 3
Case 3; comparable CT slices taken three years apart (see text for details)



X-rays over three years of diving; hyperinflation of bulla immediately after diving accident with symptoms of CAGE; all previous chest X-rays classified "normal".

Case 3

Case 3 was a non-smoking male born in 1959, who at the age of 20 had two episodes of spontaneous right-sided pneumothorax, the second episode of which was treated with unilateral chemical pleurodesis. He started diving in 1998, and made 160 uneventful dives. In April 2004, after a dive to 41 msw, he suffered from inner ear decompression sickness, and was treated with repeated hyperbaric oxygen sessions. As he was found to have a large patent foramen ovale (PFO), the decompression sickness was attributed to paradoxical embolisation of nitrogen bubbles through this PFO.

During the diagnostic work up, a high resolution CT scan of the lungs showed an ellipsoid pleural air space in the lower right lobe, with diameters 18 by 14 by 20 mm (Figure 3). Extensive pulmonary function tests, including flow-volume loops and methacholine provocation testing, were normal. He was advised by the DMO to stop diving; however, he chose to ignore this advice, as two independent respiratory physicians stated that, because the air space was isolated, there should be no problem for diving.

Over the next three years, he made a further 100 dives, during two of which he suffered from "unexplained vertigo after the dive". He was extensively reviewed in March 2007, with contrast echocardiography and high-resolution CT scan of the lungs (Figure 3). With regard to the PFO, this was found to be widely patent, as before. The lower right pleural bulla, however, was found to have substantially increased in size, measuring now 4.2 by 2.7 cm and extending well into the costo-diaphragmatic angle, to a total vertical length of 3.7 cm. Volume calculation (assuming the bulla has a roughly ellipsoid shape) shows an increase from 2.6 ml to 22 ml over these three years, or an 800% increase. He was again advised not to dive.

Summary of case 3: Asymptomatic isolated pleural bulla after pleurodesis 25 years previously; marked increase in size over the course of three years of regular diving.

Discussion

Pulmonary barotrauma is a major concern in compressed-air diving because of the high possibility of life-threatening complications such as tension pneumothorax and CAGE.[6] Although the occurrence of PBT is often associated with inappropriate ascent procedures (failing to exhale during ascent will induce a rapid increase in the pulmonary volume), many cases are reported where no such risk behaviour was observed or apparent.[4,7,8]

Alveolar rupture has been shown to occur with pulmonary overpressures of only 80 mmHg (equivalent to a water depth of only 65 cm). Proposed mechanisms by which such a slight increase of transpulmonary pressure might cause PBT include:

- *Decreased pulmonary elasticity* ('stiff' lungs). In a study of 14 young men who had suffered pulmonary barotrauma, it was shown that these individuals had less distensible lungs and airways than healthy divers and non-smoking non-divers.[9] The authors suggested that the relative stiffness of the airways increased the elastic stresses in the peribronchial alveolar tissue leading to an increased risk of alveolar rupture. It has been suggested that the increase in thoracic blood volume

during immersion may compound this problem by further reducing lung distensibility.[10]

- *Local or regional air trapping*. Classically, asthma (defined as a history of wheezing and abnormal pulmonary function tests) would disqualify a person from scuba diving, on the presumption that allergic or exercise-induced bronchospasm of the small airways may likely cause local zones of inefficient air exhalation, resulting in local overexpansion of pulmonary tissue.[11] This is supported by a number of case reports, where asthma was considered to be the only risk factor present.[4,12] In recent years, and considering that many asthmatic subjects apparently do dive without a high rate of pulmonary barotrauma, these guidelines have been somewhat relaxed, excluding now only those individuals with active, exercise- or emotion-induced asthma.[1] The remaining individuals are allowed to dive but warned of the potential risks. The value of pulmonary function tests as the sole criterion for detection of divers at risk for pulmonary barotrauma has been challenged because of low specificity.[13]
- *The presence of pulmonary blebs or bullae*. Numerous case reports are available in which divers with PBT were subsequently shown to have one or more (smaller) blebs or (larger) bullae, sometimes not visible on standard chest X-rays.[14] The same observation has been made in patients with spontaneous (non-traumatic) pneumothorax or recurrent spontaneous pneumothorax.[15,16] It is generally hypothesized that bullae predispose to pressure-reduction barotrauma either by a one-way valve mechanism at their 'entrance' (allowing entry of air upon descent but blocking outflow of air during ascent from a dive), or by an insufficient outflow capacity through a small orifice during rapid ascents.[14]

A direct causal relationship between the presence of bullous structures and lung tissue overpressure has not been established. Indeed, as imaging technology improves, asymptomatic pulmonary blebs can be found in a large number of persons. Although the exact prevalence is not known, it is reported that radiologists in a major hospital do not even mention the presence of small blebs as "*they are so common as to be considered normal findings in the patient population seen by a major hospital radiology department*".[2] Similarly, the prevalence of large bullae in a normal population is essentially unknown. Moreover, the causal relationship between the presence of bullae or blebs on chest CT and the occurrence of a first pneumothorax on the contralateral lung or recurrent spontaneous pneumothorax on the ipsilateral lung is still heavily disputed.[16,17]

When a diver is found to have pulmonary function test abnormalities after a pulmonary barotraumatic incident, few people would question the statement that these were already present before the dive. This, of course, lends credibility to a possible causal relationship. Only recently it has been suggested that diving by itself may induce changes in pulmonary function.[18] Experienced sports divers were shown to have a significantly reduced maximal expiratory flow at 25% (MEF25) and at 50% (MEF50) of vital capacity ($P < 0.01$ and 0.05 respectively) compared to non-divers.[19] There was a higher prevalence of cold-air hyper-reactivity in divers and there appeared to be a relationship with diving experience. The same observations have been made in professional saturation divers.[20]

When, after a pulmonary barotrauma, a diver is found to have pulmonary bullae on high-resolution CT scanning, the question may arise whether these bullae were pre-existent or whether they are the consequence of the barotrauma. As pre-barotrauma high-resolution pulmonary CT scans are inevitably lacking, it is impossible to ascertain the pre-existent nature of any bulla observed, making it impossible to state with certainty a significant causal relationship. Factors suggesting these bullae to be pre-existent and not caused by the diving accident could be: thick bulla wall, absence of liquid level and the presence of multiple non-ventilated spaces in the pulmonary parenchyma in combination with only localised barotrauma. Also, pulmonary over-distension bullae that have been acutely caused by PBT tend to resolve spontaneously within a few months.[21] A control CT scan some four months after the accident should therefore be performed in all cases.

Although divers are usually excluded from further pressure exposure after a PBT and/or detection of lung cysts or bullae, there is controversy as to whether a diver with an isolated, asymptomatic bulla should be excluded from diving. The discussion focuses on the possible mechanisms for such a bulla to rupture in the course of a dive. A common position suggests that according to Boyle's Law, such an air-filled structure will be compressed proportionally as the environmental pressure rises; then, when the diver ascends, the bulla will return to its initial, but not a greater volume. How then, would this promote rupture?

We hypothesize that during the dive, even if there is no direct ventilation orifice, a gradual diffusion of nitrogen through the bulla wall can take place, driven by the pressure gradient. As the elasticity of the lung tissue counteracts the volume reduction by Boyle's Law, air will diffuse from the nearby lung tissue into the bulla cavity, causing it to gradually re-expand, while still remaining in pressure equilibrium with the surrounding lung tissue. Then, as the pressure is reduced, and the air is 'trapped' inside the bulla, the bulla will grow beyond its initial volume. The elasticity of the surrounding lung tissue will now exert a concentric pressure on the bulla wall, and the resulting slight overpressure will make air (or nitrogen) diffuse out of the cavity into the surrounding tissue; the bulla will gradually return to its initial volume again. When this cycle happens in a repetitive manner, the bulla wall will get progressively stretched and the 'resting volume' of the bulla will increase. It is probable that the wall

gets thinner as this happens. At one point, the acute stretching during an ascent from a dive will cause an air leak from the bulla wall and may cause symptoms of PBT.

Conclusions

To our knowledge this is the first documented case series of non-ventilated pulmonary bullae where an increase in size can be attributed to scuba diving activity. Although a causal relationship cannot be demonstrated by the existing literature, these case reports suggest strongly that at least some pre-existing bullae can and will increase in size over the course of a few years' intensive diving. Because of the low sensitivity of plain chest X-rays, it may be advisable to obtain high-resolution CT scans of the chest in candidate divers where personal medical history leads one to suspect possible pulmonary parenchymal damage. If a diver with an isolated bulla were to be allowed to dive, it would then be advisable to perform serial follow-up CT scans after a number of years or exposures (dives), in order to observe a possible size increase in a timely manner.

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The world as it is

Diving Accident Guidelines of the German Society for Diving and Hyperbaric Medicine: summary version

Peter HJ Müller (Chairperson), Wilfried Beuster, Wolfgang Hühn, Peter Knessl, Hans Joachim Roggenbach, Volker Warninghoff, Wilhelm Welslau and Jürg Wendling

Editorial statement:

These guidelines are published here with the approval of the Executive Committees of EUBS and SPUMS, but do not necessarily reflect the policies of either Society. It was felt that their promulgation would be of use to members, and we thank the German Society for making their work more widely available. Minor editing has been necessary to fit.

Introduction

The following is a synopsis of the German Diving Accident Guidelines, which may be found on <www.gtuem.org> in German and English. It is the second revision of a guideline initially published in 2002. The first version was reviewed in 2005 and then revised and updated in 2008. It is valid until October 2011, when the next revision is scheduled. Each version was developed by a group of experts and then presented to a consensus conference organised by the GTÜM. An independent international steering committee (jury) at the conference created the final document. The current guideline, therefore, reaches level two evidence for guidelines. This is the first version published in both English and German. The extensive reference list used in preparing this document is available on the GTÜM website.

Definitions

Diving accidents, in the sense of these guidelines, are also referred to as 'decompression accident', 'decompression illness', 'decompression incident' or 'decompression injury' (DCI). Such accidents, caused by a rapid reduction of ambient pressure, are characterized by the formation of gas bubbles in blood and tissues. Depending on the physiological mechanisms involved, a distinction is drawn between 'decompression sickness' (DCS) and 'arterial gas embolism' (AGE). However, in many cases the clinical picture does not allow a distinction between DCS and AGE. Differential diagnoses may be barotrauma of the inner ear (round window membrane rupture), cerebral embolism or bleeding, vertebral disc herniation, myocardial infarction, hypoglycaemia or epilepsy (Figure 1 and Tables 1 and 2).

First aid in suspected diving accidents (Figure 2)

FIRST AID BY LAYPERSONS

In most cases, first aid is provided by the dive partners. Effectiveness of first aid and further treatment depend on an appropriate training of all divers, an emergency kit fitted to the needs of the planned dive, and failsafe communication devices (e.g., mobile phone and relevant phone numbers).

For mild symptoms (unusual tiredness, skin itching):

- give 100% oxygen (irrespective of the breathing gas used during the dive)
- give fluids orally (0.5–1.0 litres, no alcoholic or caffeinated beverages)
- protect against hypothermia as well as hyperthermia
- perform basic neurological examination
- never try in-water recompression
- if symptoms disappear within 30 min, continue 100% oxygen, call diving physician, and observe for 24 h
- if symptoms persist longer than 30 minutes: treat like severe symptoms.

If symptoms appear while still underwater or if other symptoms are present, such as:

- skin rash or discolouration
- pain
- tingling and/or numbness
- physical weakness/paralysis
- breathing difficulties
- vision, hearing or speech problems
- nausea and/or vertigo
- impaired consciousness,

follow the instructions below.

Specific first aid

- Cardiopulmonary resuscitation according to ERC guidelines, as indicated
- If diver is unconscious, put in recovery position; otherwise put in a supine position

Figure 1
System of dive accidents

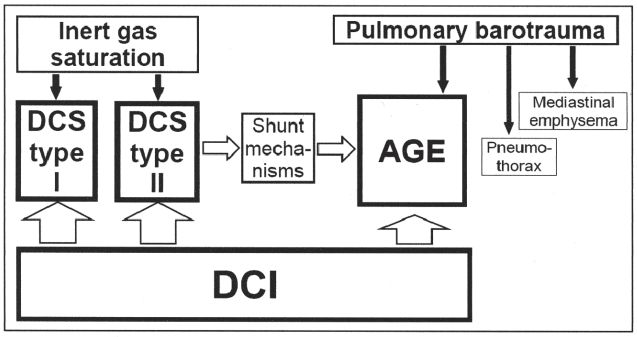


Table 1
Pathogenesis and symptoms of diving accidents
*** These symptoms can also be indicative of DCS type II or AGE**

	Decompression Sickness (DCS)	Arterial Gas Embolism (AGE)
Pathogenesis	• greater diving depth / high ambient pressure • long exposure time • saturation of body tissue with inert gas (depending on breathing gas used, usually nitrogen (N_2) • too fast ascent after long and/or deep dives with high level of tissue saturation	shunting of venous gas bubbles into arterial circulation due to: • pulmonary barotrauma with over-distension of the lungs • paradoxical embolism due to a) shunting of venous gas bubbles via lung blood vessels b) shunting of venous gas bubbles via a patent *foramen ovale* (PFO)
Time until symptoms appear	minutes to hours max. 24 hrs after completion of dive (in rare cases max. 48 hrs)	minutes after completion of dive sometimes already during ascent
Symptoms	**DCS type I** **Skin symptoms** • itching • punctate reddening • swelling • marbled skin * **Muscle and joint pain ('bends'):** • large and middle-sized joints (also depending on exertion level) • skeletal muscles. • rare: foot and hand joints **Lymphatic system:** • swollen, tender lymph nodes, (rare) **Other:** • unusual tiredness * **DCS Type II** • apathy / unconsciousness • vertigo / vomiting • sensory disturbances, paresis, paraplegia • urinary and faecal incontinence or retention • disturbed motor coordination • hearing / vision / speech troubles • acute dyspnoea ('chokes') accompanied by chest pain, coughing, feeling of suffocation • additionally sometimes muscle and joint pain developing during ascent (distribution same as with type I) • other neurological symptoms	**AGE** • apathy / unconsciousness • vertigo / vomiting • confusion / disorientation • speech and/or vision difficulties • various neurological deficits: mild sensory disturbances to complete paralysis • if respiratory centre is affected: rapid reduction in blood pressure, breathing difficulties, cardiac arrest • unilaterally dilated pupil • other neurological symptoms

- Give 100% oxygen (start as soon as possible, irrespective of the breathing gas used during the dive):
  a) if breathing sufficiently: via face mask (with demand valve or closed circuit system with CO_2 absorber); if not available: with constant flow (15–25 L.min^{-1}, non-rebreather mask with O2 reservoir)
  b) If not breathing sufficiently perform artificial respiration with 100% O_2 (Ambu/Laerdal bag with reservoir, constant flow (15–25 L.min^{-1}) or demand valve or closed circuit system with CO_2 absorber)
- Give O_2 without breaks until reaching chamber; give highest possible O_2 concentration even if O_2supply is limited (no air mix, no constant flow below 15 L.min^{-1}).
- Fluids:
  a) Diver fully conscious: give fluids orally (0.5–1.0 L.h^{-1}); no alcoholic or caffeinated beverages

Table 2
Differential diagnosis of diving accidents. It is often difficult to distinguish between DCS and AGE at the site of the accident; mixed types are common; note: treatment is the same for both diagnoses

	Differential Diagnosis	Clinical Symptoms
DCS type II with inner ear symptoms	• barotrauma of the inner ear (rupture of the round window membrane)	• hearing loss • tinnitus • vertigo Caution: many patients do not exhibit the complete classical triad of symptoms
DCS type II with neurological symptoms	• cerebral insult caused by embolism or bleeding • vertebral disc herniation	• motor, sensory or cerebral neurological deficits • symptoms of paraplegia
DCS type I	• cardiac infarction	• e.g., pain in the left shoulder
DCI (AGE)	• hypoglycaemia	• e.g., unconsciousness
DCI (AGE)	• epilepsy	• e.g., seizures
DCI suspected uncertain symptoms	• breathing gas contamination (CO etc.) • toxic effect of breathing gas in mixed gas diving • hypercapnia due to skip breathing • hyperventilation due to psychological stress • hypoglycaemia • psychotropic drugs	• headache • impaired consciousness • vertigo

b) Diver with impaired consciousness: do not give fluids orally

- Call emergency control centre and notify them of "*suspected diving accident*".

Additional actions

- Perform basic neurological examination
- Protect against hypothermia and hyperthermia; if hypothermic: no active re-warming
- Never try in-water recompression
- Organize transport
- Call emergency control centre.
  a) Mode of transport: no preference for specific mode of transport, transport fast and gentle, no restrictions for helicopter transport (lowest safe flying altitude)
  b) Transport destination: nearest emergency unit, preferably close to hyperbaric treatment chamber
- Document diving data, development of symptoms and treatment
- Observe dive partner as well
- Impound diving gear (e.g., decompression computer)
- Consult adiving medical phone hotline, if necessary; key words "diving accident".

1 National DAN hotline in Germany and Austria: 00800 326 668 783 (00800 DAN NOTRUF)
2 National DAN hotline in Switzerland: +41 333 333 333 (or 1414 within Switzerland)
3 VDST hotline: +49-1805-660560
4 Office of the Naval Medical Institute, German Navy: +49-431-54091441
5 Diver hotline aqua med: +49-700-34835463
6 International DAN hotline: +39-0396057858

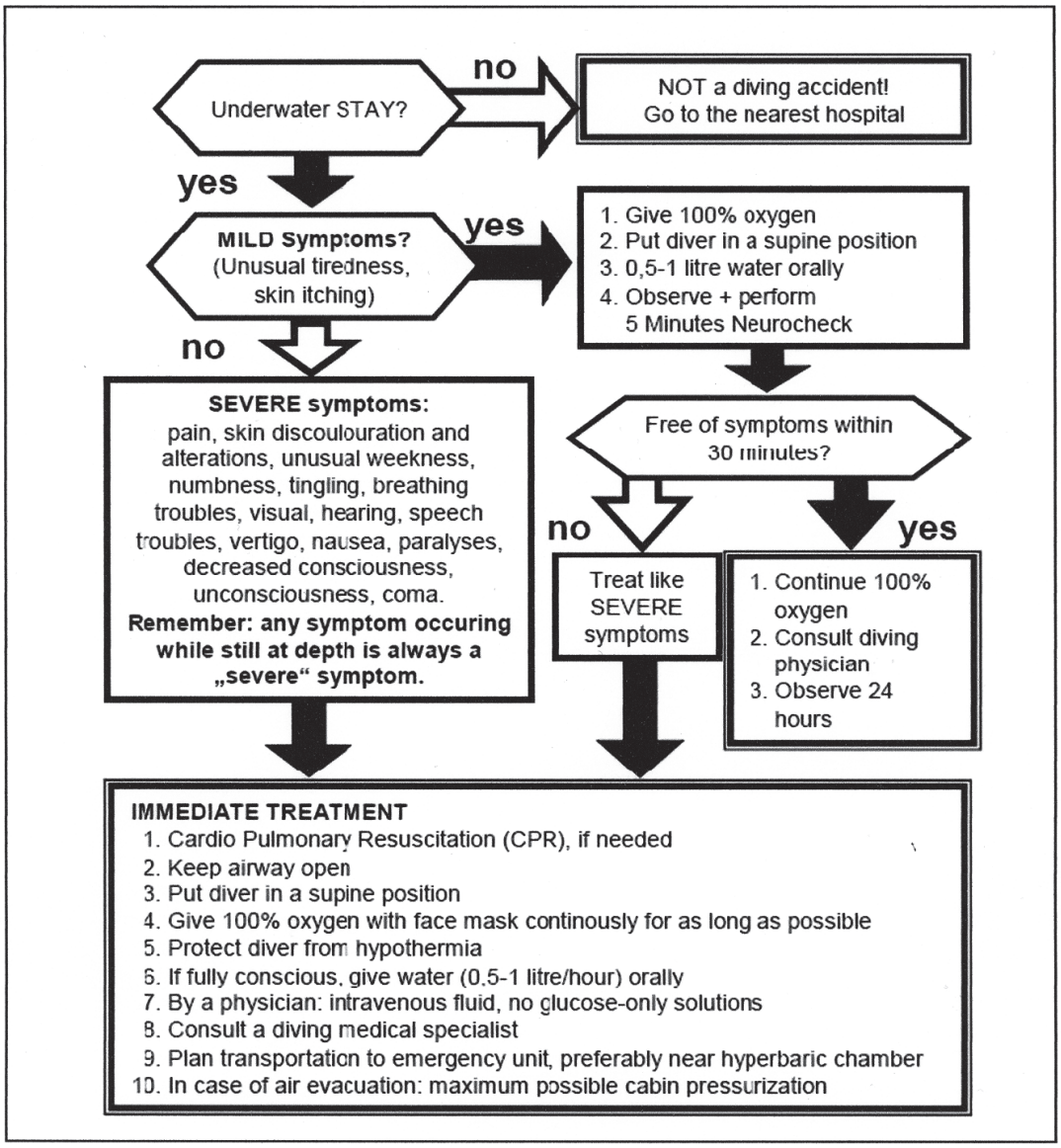
More hotlines can be found at <www.gtuem.org>

FIRST AID BY MEDICAL PERSONNEL

Specific first aid

- Cardiopulmonary resuscitation according to ERC guidelines, as indicated
- If diver is unconscious, put in recovery position, otherwise put in a supine position
- Give 100% oxygen (irrespective of the breathing gas used during the dive):
  a) if breathing sufficiently: see above
  b) if not breathing sufficiently: perform artificial respiration with 100% O_2 (aim at FiO_2 = 1.0), if necessary via endotracheal tube, without breaks until reaching hyperbaric treatment chamber
  c) Give highest possible oxygen concentration even if oxygen supply is limited (no air mix, no constant flow below 15 $L.min^{-1}$)
- Intravenous fluids: give 0.5–1.0 $L.h^{-1}$ i.v.; do not use glucose-containing solutions
- Medication: generally follow standard procedures in emergency medicine; up to now, no drug has been

Figure 2
Flow chart: diving accident management (modified after Divers Alert Network Europe)



definitively proven to be specifically effective in the treatment of diving accidents.

Additional actions

- Perform basic neurological follow-up examinations
- Urinary catheter, as indicated
- Pleural drainage, as indicated
- Protect against hypothermia as well as hyperthermia. If hypothermic, active re-warming only with ICU-like capabilities on site
- Hyperbaric treatment as soon as possible, after consultation with a diving medical hotline; even delayed hyperbaric treatment is often effective
- Consult diving medical hotline (see above)
- Monitoring and documentation: emergency treatment protocol, documentation of diving data by laypersons, development of symptoms and treatment, impounded instruments (e.g., decompression computer).

Transport

Transport by helicopter (lowest safe flying altitude), land-based vehicle, boat, or plane (cabin pressure close to 1.0 bar). Transport with as little vibration as possible and without reduction of ambient pressure; Continue O_2 treatment without breaks until reaching hyperbaric chamber; continue commenced treatment.

First hyperbaric treatment (Figure 3)

Hyperbaric treatment chamber, working pressure min. 280 kPa (2.8 bar; 18 msw), in Europe, construction and equipment according to EN 14931, emergency medicine equipment according to DIN 13232 in Germany.

Actions before treatment

- Perform neurological examination (documentation!)

- If pulmonary barotrauma is suspected: perform chest X-ray (p.a./lateral) / thoracic CT scan if feasible within reasonable time frame
- Pleural drainage, as indicated
- Urinary catheter, as indicated
- Myringotomies, as indicated
- If patient is intubated: fill cuff with fluid or check cuff pressure continuously
- Consult diving medical hotline as needed (see above).

Treatment tables
- Standard treatment table is US Navy Treatment Table 6 (or modifications of this) for all diving accidents irrespective of breathing gas used by the casualty
- For omitted decompression without symptoms, shorter tables may be used, e.g., US Navy Treatment Table 5.

Actions during treatment
- Repeated neurological examinations (documentation!)
- Repeated auscultation of lungs; perform auscultation prior to each decompression
- Periodic check of all gas-filled confinements (e.g., endotracheal tube cuff, infusion, drip chamber, blood pressure cuff)including before each decompression.

Adjuvant treatment
- Generally follow standard procedures in emergency and intensive care medicine
- If the patient is awake, special psychological support for reassurance and relief of anxiety
- Check bladder; ensure adequate hydration and fluid balance
- Up to now, no drug has been proven definitively to be effective in the treatment of diving accidents
- Documentation for physicians continuing treatment.

Transport to hyperbaric treatment centre

If symptoms persist after first hyperbaric treatment, one or more follow-up treatments are necessary starting within 24 hours. If inpatient care cannot be provided between treatments at the initial hyperbaric chamber, organize transport to a hyperbaric centre which has this capability. In principle, flight transport with regular cabin pressure (e.g., 81 kPa; 0.8 bar abs.) is possible after one hyperbaric treatment. As a general rule, in-flight oxygen breathing should be available. Decide about transport together with experienced diving physicians, taking into account previous and persisting symptoms in the specific case.

ACTIONS DURING TRANSPORT
- Generally follow standard procedures in emergency medicine and intensive care medicine
- Continue commenced treatment
- Give 100% oxygen depending on symptoms
- Ensure adequate hydration and fluid balance, especially during flights (i.v./orally)
- Perform basic neurological follow-up examinations
- Documentation, e.g. emergency medicine protocol
- Medication: follow standard procedures in emergency medicine and intensive care medicine.

Subsequent hyperbaric treatments

- If needed, perform second treatment according to standard DCI treatment table or hyperbaric oxygen treatments, e.g., 'problem wound treatment protocol'; maximum two treatments within 24 hours, maximum interval between treatments 24 hours.
- Diagnostics: depending on clinical symptoms, MRI, CT and periodic examination by neurologist, reassessment of pulmonary function, additional examinations by specialists depending on clinical symptoms
- Physiotherapy: between hyperbaric treatments depending on clinical symptoms, starting max. three days after diving accident. Physiotherapy during hyperbaric treatment is possible, but there is no evidence that this will have any additional advantage.
- Medication and additional treatment: depending on clinical symptoms and following recommendations of consulted medical specialists
- Decision on stopping hyperbaric treatments: after complete and lasting disappearance of symptoms, hyperbaric treatments may be terminated. If symptoms do not continue to improve over three to five days after an initial reduction, hyperbaric treatment should be stopped and rehabilitation treatment recommended for neurological symptoms should be continued.
- Documentation
- Rehabilitation: if neurological deficits persist after hyperbaric treatment has ended, rehabilitation according to the clinical symptoms will follow immediately.

Diving fitness after diving accident

With recreational divers, the assessment of diving fitness after diving accidents should generally follow the recommendations of national and international diving medical associations. Special statutory provisions apply to occupational divers. The reassessment of fitness to dive requires that the treatment has been terminated and a stable treatment result has been achieved. Assessment of fitness to dive should be reserved to experienced diving physicians with a minimum qualification comparable to 'Diving Medicine Physician, EDTC' and with practical experience in treating diving accidents.

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Key words
Diving accidents, decompression illness, decompression sickness, cerebral arterial gas embolism (CAGE), first aid, hyperbaric oxygen therapy, transport, medical society

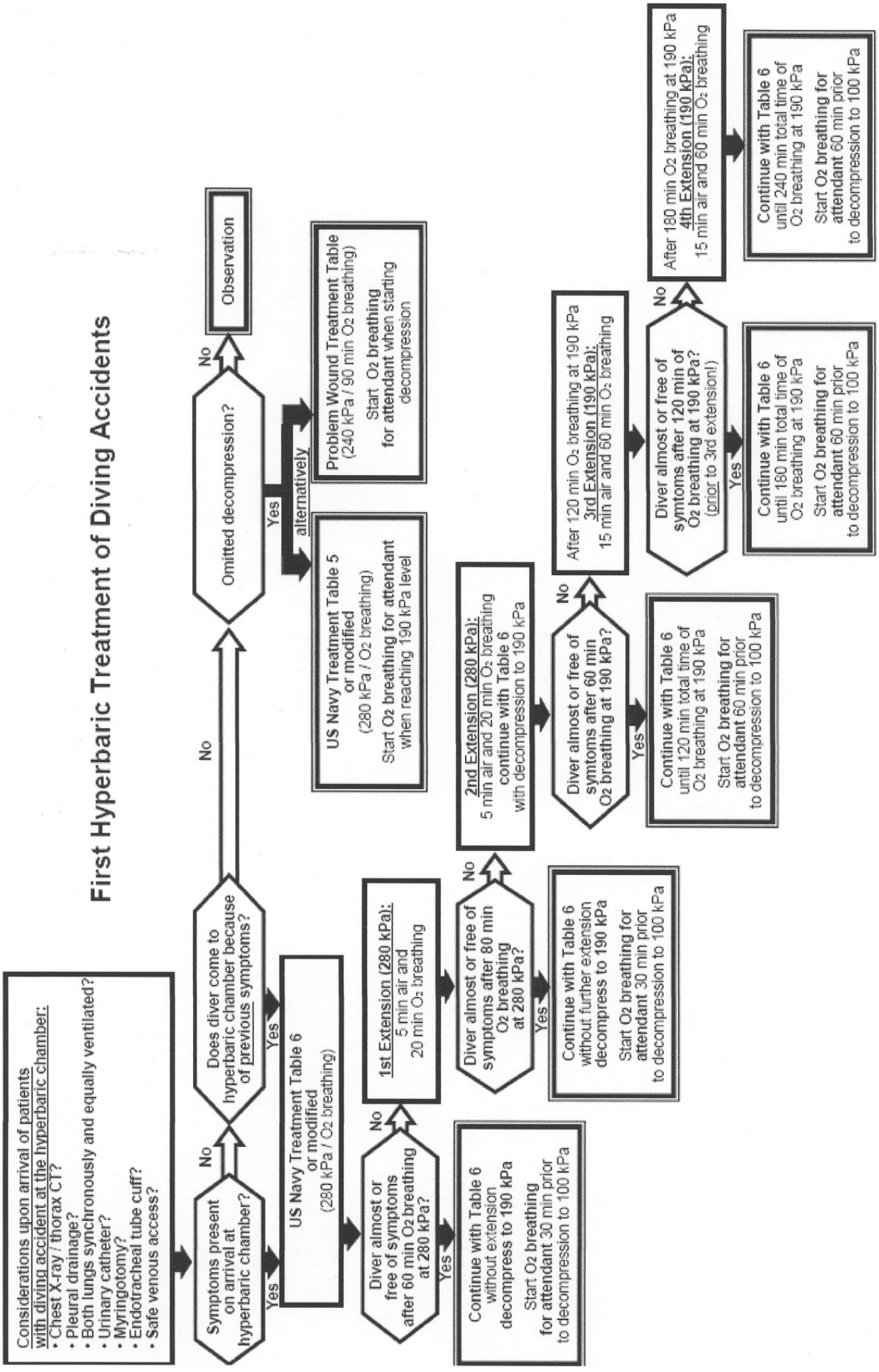

Figure 3. First hyperbaric treatment of diving accidents

Critical appraisal

Exogenous nitric oxide may reduce bubble formation in both wet and dry divers

Bottom line
1. Dry diving produces fewer bubbles than wet diving.
2. Nitroglycerine probably reduces bubble formation in both wet and dry dives.

Citation
Dujic Z, Palada I, Valic Z, Duplancic D, Obad A, Wisløff U, Brubakk A. Exogenous nitric oxide and bubble formation in divers. *Med Sci Sports Exerc*. 2006; 38(8): 1432-5.

Lead author's name and e-mail: Zeljko Dujic, <zdujic@bsb.mefst.hr>

Three-part question
Among divers, would a short-acting NO donor, nitroglycerine, reduce bubble formation after standard dives and decompression in man?

Search terms
Decompression illness, nitric oxide, diving

The study
Non-blinded, randomised trial with intention-to-treat.

The study divers
16 experienced divers; all fit and well.

CONTROL GROUPS
(N =16; 16 analysed) 10 wet divers, 30 metres' sea water (msw), swimming for 500 m; bottom time 30 min with 3 min safety stop at 3 msw; 6 dry divers, 18 msw, bottom time 75 min with stop at 3 msw for 7 minutes.

EXPERIMENTAL GROUPS
(N = 16; 16 analysed) As above, preceded by 0.4 mg of nitroglycerine oral spray 30 min before diving.

The evidence
See Table 1

Comments
1. The allocation to wet versus dry diving was randomised, but all divers then did the control dive followed by the nitroglycerine dive. Allocation to each dive in a random order may have been a more methodologically rigorous approach.
2. Because we did not have the original data, our table indicates the mean difference and 95% CI for an independent t-test for the NO/no-NO comparisons. Hence our 95% CIs indicate no statistically significant differences, while the authors appropriately used a paired t-test and reported a P-value of 0.04 (wet dives) and 0.05 (dry dives).
3. Results may not extrapolate to all divers; only divers with "*considerable experience of air and oxygen diving*" were eligible.
4. No side-effects measured.

Appraised by:
Ing Han Gho and Mike Bennett, Prince of Wales Hospital, Sydney; Friday 1 August 2008
E-mail: *<m.bennett@unsw.edu.au>*

Key words
Bubbles, Doppler, diving, decompression, nitric oxide, critical appraisal

Source
<www.hboevidence.com>

Table 1
Independent as opposed to paired t-test for the significance of the difference in bubble count for the NO/no-NO comparisons for two groups of divers during 'wet' and 'dry' dives

Measure	Control group		Nitroglycerine group		Difference	95% CI
	Mean	SD	Mean	SD		
Wet dive (bubbles.cm^{-2}) (N = 10)	0.87	1.3	0.32	0.7	0.55	-0.43 to 1.53
Dry dive (bubbles.cm^{-2}) (N = 6)	0.12	0.23	0.03	0.03	0.09	-0.12 to 0.30
	Wet divers (N = 10)		Dry divers (N = 6)			
Wet vs. dry dive (bubbles.cm^{-2})	0.87	1.3	0.12	0.23	0.75	-0.42 to 1.92

From the recent literature

Hyperbaric oxygenation in the management of cerebral arterial gas embolism during cavopulmonary connection surgery

Andrew Newcomb, Geoff Frawley, Andrew Fock, Martin Bennett and Yves d'Udekem

Summarised from *J Cardiothorac Vasc Anesth*. 2008; 22: 576-80.

This paper discusses two cases of cerebral gas embolism that were treated at the Alfred Hyperbaric Unit, Melbourne. Both cases were unusual in that they were infants and had been undergoing corrective open-heart surgery for uni-ventricular congenital heart disease. Paediatric congenital heart surgery is not performed at our institution, so in addition to the issues of treating critically ill infants in a remote environment, these cases also involved significant logistical hurdles in the transportation and management of infants in a hospital with predominantly adult treatment experience.

The authors describe the details of the surgery and the mechanics of the entry of gas in each case. In both cases, a number of therapies were instigated immediately that gas embolisation was detected. These included cooling, Trendelenburg positioning, retrograde cerebral perfusion, 100% oxygen and lignocaine infusion. At the completion of surgery, the patients were transferred to the Alfred for hyperbaric oxygen therapy (HBOT) with the aim of eliminating any remaining intravascular gas. The paediatric ICU team accompanied the patient during transfer and both a hyperbaric nurse and a paediatric intensive-care nurse attended the patient during the HBOT. Both patients were successfully treated with a Royal Navy Treatment Table 62 without complication despite being inotrope-dependent and immediately post major cardiac surgery.

The patients were transferred back to the parent hospital at the completion of the HBOT and it was decided not to perform further treatments due to the risks associated with transfers and other logistical issues. Both patients had excellent post-operative outcomes with no observed neurological sequelae.

The paper discusses the various treatment options for gas embolisation and the fact that there has been only one other case report of HBOT use following infant open-heart surgery. Patient management during HBOT was a collaborative process between the paediatric ICU and hyperbaric teams.

Andrew Fock, Hyperbaric Medicine Unit, Alfred Hospital, Melbourne
E-mail: *<A.Fock@alfred.org.au>*

Key words
Cerebral arterial gas embolism (CAGE), children, cardiovascular, case reports, gleanings (from medical journals)

The diving doctor's diary

Haemophilia and diving

"I recently assessed an 18-year-old who wished to become a recreational diving instructor. He had a history of haemophilia, requiring Factor VIII a few times in his life after injury. Other than coming with his mother and being somewhat 'stroppy', he was completely well and no abnormalities were identified in the medical examination. He came with a letter of support for his fitness to dive from his haematologist".

- Advice was sought from a world authority on diving medicine. What do you think his letter said?
- Why are people with haemophilia encouraged to dive recreationally by their support groups and haematologists, often without any discussion of the potential risks?

Readers are invited to put their views to the journal at <spumsj@cdhb.govt.nz>. Comment by a haematologist experienced in haemophilia care would also be appreciated. Responses will be collated in the next issue.

Book reviews

Hyperbaric oxygen for neurological disorders

John Zhang, editor

Hard cover, 480 pages
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Flagstaff, AZ: Best Publishing Company; 2008
Price: US$79.00 plus postage
Copies can be ordered from Best Publishing Company, 2355 North Steves Boulevard, POBox 30100, Flagstaff, AZ, USA 86003-0100
Website: <www.bestpub.com>
Phone: +1-928-527-1055
Fax: +1-928-526-0370

This book is a collection of papers/chapters contributed by a variety of authors from all over the globe, compiled by John Zhang, MD, Professor of Neurosurgery, Loma Linda, California. In an extensive preface to the book, Dr Zhang commends the contributors for their motivation and commitment in the controversial field of HBO for neurological disease. There can be no doubt that all authors are compassionate medical scientists, caring for the wellbeing of their patients, and surely all are committed to this topic, as this area of application of HBO therapy is mostly looked upon in a negative way.

The idea of assembling the 'state of the art' on this topic in a single-volume book is a good one. However, after having read this book from cover to cover (not an easy task, I might add!), I am left with mixed feelings. Most of these can be attributed to the editing work or, rather, the absence of it. It seems that Dr Zhang has not done any editing at all, but, perhaps out of respect for his contributors, simply placed the entire contributions one after the other without review in the form of a book.

The applications covered in this book may all be neurological in nature, but encompass a huge range of conditions, including stroke, traumatic brain injury, carbon monoxide poisoning, radiation damage, autism, multiple sclerosis, cerebral palsy, amyotrophic lateral sclerosis, and a contribution on "HBO in China". There is no or very little logic to the order in which these chapters have been placed, and the nature of the contributions varies from theoretical discussion to case report to historical overview to critical appraisal.

It is not apparent that there has been any agreement or guidance with regards to content or form. This results in the first three (even four) chapters essentially repeating the same information, with a slightly different focus ("Brain physiology and HBO"; "Cerebral physiology and HBO"; "Brain tissue oxygenation"; "Mechanisms for HBO in neurological injury"). No attention has been paid to a uniform formatting of the references, some texts use reference numbers (Vancouver style); others use Harvard style (name, publication year).

This results in a very uneven scientific rigour to the various chapters. In the very first chapter, the difficulties in extrapolating results from animal research to human clinical application are highlighted. The complexity of the human brain, its vascular and energy supply, its energy household, is so great, that it would be surprising if hyperbaric oxygen therapy would not have an influence on some of these processes. Therefore, to conclude that HBO may be a useful intervention in brain pathology, is perhaps too hasty a conclusion. Unfortunately, many of the authors commit exactly this error in their text. In many chapters, basic animal studies are extrapolated to the human clinical HBO setting with little critical appraisal. It is remarkable that these chapters are followed by hundreds of references.

Other chapters have no scientific value at all, but are simply 'opinion' articles and are, in my view, out of place here. However much I may disapprove the kind of emotional dispute that has gone on for years regarding the use of HBO for multiple sclerosis or cerebral palsy, there is no place for an equally emotional plea in favour of this indication in a book like this. Other chapters use data from non-indexed publications, and one wonders why these have not been published in peer-reviewed journals.

There are two chapters on evidence-based medicine by Mike Bennett, based on his contributions to the Cochrane Library of Systematic Reviews. These, not surprisingly, conclude that, both for acute traumatic brain injury and for multiple sclerosis, there is insufficient evidence to recommend HBO at the present time as a treatment modality.

This book may provide some insights into brain pathophysiology and where/how HBO may influence these processes. It discusses clinical and experimental studies, but not in a systematic way, and thus, except for the Cochrane chapters, does not add any evidence to evaluate the role of HBO in neurological disorders. The book may be a useful starting point of information for those who take a specific interest in HBO and neurology, but is, in general, too superficial, unstructured and insufficiently objective to serve as a good reference text.

Peter Germonpré
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Key words
Hyperbaric oxygen, neurophysiology, brain injury, multiple sclerosis, medical conditions and problems, textbook, book reviews

The ECHM collection, volume 3

Marroni A, Mathieu D, Wattel F, editors

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Website: <www.bestpub.com>
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Fax: +1-928-526-0370

It is nice to see an extract from a book review given by oneself sent back as a promotional quote for the next volume of a series of books. So it was with some interest that I was asked to review the third instalment of the European Committee for Hyperbaric Medicine (ECHM) collection, their consensus meetings from 2004 to 2006.

This volume has a similar format to the previous two, reporting on the findings of clinical consensus meetings. The clinical components start with clinical questions on a topic, move on to outline the evidence and end with a consensus statement. This is certainly true for the first part, which looks at the evidence for various indications for hyperbaric oxygen therapy (HBOT). Unfortunately this is where a book coming out four years after the event can be caught out. Hyperbaric medicine has changed rapidly in that time, and many of the Cochrane Reviews, other seminal research and newer textbooks, all published since 2006, are inevitably missing. Textbooks by their nature are out of date by the time they are published and this section of the third ECHM collection suffers as a result. All this being said, it is interesting to see how evidence-based medicine was used in developing the consensus statements; I found their classifications somewhat non-standard but they seemed to fit the work produced.

The interesting second and third parts of the first meeting looked at the development of the hyperbaric facility as a clinical entity. Luminaries like Jordi Desola look at aspects such as the organization of the hyperbaric unit, the roles of people within it and the role of the unit in diving and occupational medicine. There are also good sections from people experienced in the field on the two ends of the spectrum with "HBO in emergency and intensive care" and "HBO in outpatient care". Advice is limited by space but these are good summaries of the issues that a new facility will be getting into in these modalities of patient care.

The rest of the first section looks at equipment, risk-management standards, education and research in hyperbaric medicine, topics often not well covered in general textbooks. These are from a European perspective but incorporate international standards such as NFPA 99. Europe is trying to meld various national standards into a single European standard for hyperbaric medicine, and this process is well worth reading for both medical and technical staff.

The second section is from a workshop in 2005 looking at quality assurance (QA) in the hyperbaric facility. Experiences from Great Britain (except Scotland), Poland, other European countries and the USA make for some enlightening reading on QA approaches. This section also has an interesting chapter from an independent certification entity (Germanischer Lloyd), which provides an insight into how these organisations work, and will aid facilities that intend to obtain independent certification in the future. This section does not seek to develop a method for establishing a common European Union QA system, but illustrates well the differences between countries.

The final section is the report from the "Conference on oxygen and tissue repair", which was a joint meeting between ECHM and the European Tissue Repair Society held in Ravenna in 2006. It is divided into three parts, with part one being in the standard format of developing questions to be answered by the Committee with an indication of the level of evidence to support their findings. Included with this is a biblographical overview of the problems in wound healing, looking at specific issues such as the incidence and prevalence of chronic wounds, the cost of chronic wounds (and their treatment), the assessment of wounds and the rationale behind treatment of these types of wounds.

The second part to this conference looks at our understanding at that time of the basic science of chronic wounds and the role of oxygen. Unfortunately some authors submitted only abstracts, which is frustrating as it diminishes the quality of the book.

A final chapter attempts to rationalise the cost of HBOT as a treatment modality. It comes with some remarkable financial data from Italy: that using HBOT for necrotizing infections, diabetic wounds and radiation-induced wounds would save Italy €164,735,050 per annum. I was unable to verify the economic model they used but it would certainly be a good example to send to health care funds providers!

Overall, *The ECHM Collection: Volume 3* is a little disappointing compared to the two previous excellent volumes, which had much better cohesion between the sections and chapters. This volume does have some gems but, as a whole, it is somewhat disorganised and seems to have been put together in a rushed manner. This may be due to the need to get proceedings out in a timely fashion but this should not be done at the expense of the quality of the overall publication, which is what seems to have happened here.

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Key words
Hyperbaric oxygen therapy, hyperbaric facilities, safety, wounds, 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 is provided 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

Education Officer's Report

This is my first report as SPUMS Education Officer. It is an honour to have assumed the role at the SPUMS AGM this May. This report has been delayed as a result of my recent ill-health, the complex backlog of work that I have inherited, and the need to come to grips with the broader aspects of the role. I offer my apologies to members who may have experienced delays with regard to Diplomas. I am happy to receive correspondence and will do my best to resolve the many outstanding issues.

It is my intention to raise the profile of the SPUMS Diploma, reflecting its unique position as a qualification in diving and hyperbaric medicine. The SPUMS Diploma complements the Society's other academic activities, the Journal, *Diving and Hyperbaric Medicine*, and the Annual General Meetings. The Diploma is referenced by Medicare in Australia as a higher qualification to permit billing for treatment in recompression facilities. There are very few qualifications (other than those of the specialty colleges) that have been afforded this status at Government level.

In raising the profile of the Diploma it will also be necessary to adhere to formal process. SPUMS has stipulated that a number of criteria must be satisfied before diplomas can be awarded, and these are documented on the facing page. The basic criterion is SPUMS membership for two years; we do not award SPUMS Diplomas to non-SPUMS members. The remaining criteria do not need to be met in any particular order, except that a research proposal *must* be submitted to the Education Officer prior to commencing a diploma project.

Diploma projects

One of the criteria for successful completion of the SPUMS Diploma is completion of a research project. This requires *prospective registration*. A written proposal for research in a relevant area of underwater or hyperbaric medicine is submitted to the Education Officer for consideration. Where relevant, ethics approval must be obtained. Once approved by the Education Officer (assisted by the Academic Board), the project is then embarked upon. The approval process may include some suggestions regarding research design and methods from the Academic Board. All members of the Academic Board have higher research degrees, and our aim is to assist SPUMS members in producing quality research. Applications with completed research papers that have not previously been registered and assessed as proposed diploma projects will not be accepted.

There are several reasons for prospective registration:

- To ensure fair and appropriate process is followed (including ethical committee approval), and that projects are consistent with SPUMS guidelines.
- To avoid duplication of projects between members.
- To assist the candidate with the project, with the opportunity for some early expert advice from Academic Board members. Hopefully this will also assist with improving the quality of the papers when they are finally published.
- Early goal setting to ensure that the project is completed in a timely manner.

The process is intended to assist the candidate, not serve as a barrier; however, it is expected that the candidate will provide the energy and intellectual stewardship and ownership of their project.

There may be a delay of up to six weeks after a project proposal is received before final approval is given, while the proposal is fine tuned by the Academic Board in consultation with the candidate. I am a pragmatist, having worked as a clinician all my career and I fully recognise the difficulties of undertaking research whilst in full-time clinical work. We are particularly interested in general practice and community-based projects, and there are numerous opportunities for epidemiological research in diving.

Whilst attending to the backlog of SPUMS Diplomas that have been registered, I have adopted two fundamental principles to assess and review the many projects in their various stages of evolution:

i Natural justice – if individuals have previously submitted in good faith and have evidence of acceptance of their project, then it will be considered as a registered SPUMS project.

ii No disadvantage – if candidates have evidence of registering their project, and the completed report has been confirmed as meeting Diploma standards, then, provided all other criteria are satisfied, their SPUMS Diploma will be awarded.

We have a long list of candidates, but for whom we may have inaccurate information and the status of their diploma process may not be clear. There may also be names missing from the list. I will be attempting to contact all candidates to assess their situation and to see if any assistance is required. However, do not hesitate to contact me prospectively if you think you should be on that list.

Approved diving and hyperbaric medicine courses

Inherent in the SPUMS Diploma process is a need for SPUMS to evaluate and accredit courses in diving and hyperbaric medicine. This has not been done for many years and we intend to review both the process of evaluation and the courses available and their contents. This is a considerable undertaking that will take several years and will need the assistance of colleagues from SPUMS, EUBS, and possibly a professional educationalist.

This requires a zero-based content assessment to answer such questions as:

- What knowledge and skills are required to work at a hyperbaric facility?

- What knowledge and skills are needed to assess medical fitness to dive?
- What extra knowledge and skills are needed for assessment of the occupational diver?
- How does the course evaluate its performance?

This will enable us to review and compare content from courses internationally and to develop a system of mutual recognition with our overseas colleagues. The process of evaluating course content is obviously very relevant to the SPUMS diving medical and the Diving Doctors List. There is much to do and I look forward to the challenges.

Associate Professor David Smart
Education Officer SPUMS

Minutes of the Annual General Meeting of SPUMS held at Liamo Resort, Kimbe, PNG on Wednesday 28 May 2008

Opened: 0833 hr

Present: The President, Dr C Acott, and 30 voting members

Apologies: Dr S Sharkey

1 Minutes of the 2007 AGM
Minutes of the previous meeting were published in *Diving and Hyperbaric Medicine*. 2007; 37(2): 101-5 and were posted on the noticeboard.
Motion that the minutes be accepted as an accurate record.
Proposed, Dr C Acott, seconded, Dr G Williams, carried

2 Matters arising from previous minutes
Nil

3 President's report: Dr C Acott

4 Secretary's report: Dr S Lockley (on behalf of Dr S Sharkey)

5 Education Officer's report: Dr F Sharp
No report presented

6 Treasurer's report: Dr G Williams
Comments on report: Dr M Davis commented in regard to additional income received through advertising payments ($168 x 4 per year).

Motion that the subscription fees be increased. Full members AUD$150 (internet transaction); AUD$170 (manual/paper-based transaction). Associate/retired/medical student members AUD$80 (internet transaction); AUD$100 (manual/paper-based transaction). (This will require an update of the website to allow reminders/membership renewals.)
Proposed, Dr G Williams, seconded, Dr V Haller, carried

7 Election of office bearers:
The following were elected/appointed unopposed:

President:	Mike Bennett (elected)
Secretary:	Sarah Lockley (elected)
Committee Members:	Sarah Sharkey (elected)
	Scott Squires (elected)
Education Officer:	David Smart (appointed)

8 Appointment of the Auditor 2008:
The Treasurer has recommended the appointment of Barrett, Baxter and Bye (Medical Accountants) as Auditor. This will allow electronic transfer of all information and use of 'Quickbooks' software.
Motion that Barrett, Baxter and Bye be appointed as Auditor.
Proposed, Dr G Williams, seconded, Dr C Acott, carried

9 Business of which notice has been given:
9.1 Motion that the Journal Editor's report be added as a standard AGM agenda item.
Proposed, Dr M Davis, seconded, Dr C Acott, carried
Journal Editor's report was then presented.
9.2 Motion that Neal Pollock be elected to Full Membership.
Proposed, Dr M Davis, seconded, Dr S Mitchell, carried
(Note this item appears in Agenda 2008 AGM published in *Diving and Hyperbaric Medicine*. 2008; 38: 50.)

Closed: 0911 hr

President's report

This is my last address as President.

The first combined issue of the Journal for SPUMS and EUBS was published in March. Mike Davis and Peter Müller are to be congratulated on their excellent work. The Journal has always been the SPUMS' flagship, so now it will be for both societies. We are, however, in a trial period of 24 months and if all goes well – and there is no reason to suspect that it will not – then a formal arrangement will be signed between the two societies.

The SPUMS Diving Medical is about to change. A draft has been submitted to the Committee by Mike Bennett et al and will be debated at length at a later date.

The future of the Society depends on membership and the establishment of a recognised indexed journal. Membership

depends on the recruitment of the 'younger' generation and I'm pleased to announce that the new committee has two new faces – both from the RAN School of Underwater Medicine.

The future of the journal's indexing rests with the publication of scientific articles of merit so please encourage your colleagues to submit to DHM instead of other journals. I guess it is 'publish or perish'.

Finally I would like to thank Sarah Sharkey and Guy Williams who have done excellent jobs as Secretary and Treasurer respectively – the two hardest jobs of the Society. I would also like to thank Robyn Walker, David Vote, Fiona Sharp, David Smart, Christine Lee, Mike Davis and Vanessa Haller for their efforts on the Committee.

SPUMS is in good hands with the new incoming committee. I wish Mike Bennett, the new President, and the new committee success.

Thank you.
Chris Acott

Secretary's report

Since the 2007 AGM, the SPUMS Committee has held two meetings – one face-to-face and one by telephone. Despite the infrequent meetings, geographic spread and competing priorities for most of us, some important progress has been made by the Committee with respect to improvements in financial control, implementation of a review of the SPUMS Diving Medical including policy on Diabetes, review of ASM administrative guidance, website improvements and most significantly the merging of EUBS and SPUMS journals.

SPUMS Membership. At the 2007 AGM SPUMS membership was 837, with 162 outstanding renewal payments. Current membership is 849 with 124 outstanding renewals; 18 are new members.

It has been a privilege to serve as Secretary of SPUMS for the last three years. It was a challenging and rewarding role and I wish my successor and the new committee all the best in the term ahead.

Sarah Sharkey

Editor's report

This is the first formal editorial report to a SPUMS annual general meeting. The year since the AGM in Tutukaka in April 2007 has been a very eventful one for the Society's journal, *Diving and Hyperbaric Medicine* (DHM).

For over two years, discussions have been held with the European Underwater and Baromedical Society about incorporating their journal with ours into a combined Australasian and European journal. Dr Robyn Walker and I represented SPUMS at the EUBS meeting in Sharm El Sheikh in September, where Dr Peter Müller, the Editor of the *European Journal of Diving and Hyperbaric Medicine*, and I gave a joint presentation to a plenary session. Dr Walker and I met with the EUBS Executive and had several informal meetings, along with other SPUMS members, with senior members of EUBS. At their AGM, the proposal to proceed with the amalgamation was greeted with acclamation. Thus, the March 2008 issue was the first joint publication of the two societies. I hope to continue as Editor, now with the assistance of an Editorial Board consisting of three members from each Society. I believe this is a major step in ensuring a future for the Journal in the highly competitive modern world of medical publishing.

The second important event has been our application and assessment during 2007 for an ISI rating. Just two weeks before this AGM we heard that our application had been successful and that DHM will be indexed on Science Citation Index Expanded (SCIE) from the beginning of 2007.

Thirdly, the establishment of the Rubicon Foundation (<http://archive.rubicon-foundation.org/>) will be of considerable importance in generating a huge searchable electronic database of the world's diving medical literature that will be available in the public domain. SPUMS has supported this process by providing the complete 35-volume collection of the *SPUMS Journal* which should be in place by the end of 2008. Further volumes of DHM will be entered after a three-year delay from the date of publication. Members are urged to support Rubicon by a donation to the Foundation.

On the negative side has been the continued difficulty in obtaining in a timely manner publishable manuscripts from speakers at our ASMs. This and the limited numbers of original papers that are submitted means that DHM continues to live a hand-to-mouth existence and opportunities to plan ahead and to develop themed issues are few and far between. This cannot continue, especially as SCIE citation is an ongoing process, requiring the maintenance of a high standard of publication in the Journal, which, of course, is entirely dependent on the members of the two societies to ensure.

It is with considerable regret that I advise of Sarah Webb's resignation as Editorial Assistant after the June 2008 issue. Sarah has been an essential part of the journal's development in the past six years and it will be very difficult to replace her, especially at this critical time of expansion for the journal. I would like to extend to her the Society's thanks and best wishes for the future.

Mike Davis

Treasurer's report

SPUMS remains in a sound financial situation, but our membership base is not growing (and in fact declining); so revenue is declining. At the end of 2007, we had approximately AUD$120,000 with St George Bank. We have three accounts: a general operating account, a journal account and an account for ASM expenses. All transactions are now via EFT, with dual authorisation required for every process.

Our new venture with EUBS is in its very early phase, with our first combined journal having been recently distributed to members.

The SPUMS website is overdue for a major revamp, which will be an extra expense for 2008, but we need to streamline SPUMS administrative processes, particularly membership renewal and ASM registrations – internet-based transactions reduce our workload and costs.

SPUMS membership fees have not increased for some time, and our costs slowly rise with inflation, hence the need for an increase in fees. We may lose EUBS members who joined SPUMS to receive the journal. SPUMS runs very leanly at present; I suspect many of the Committee absorb SPUMS-related costs into their own practices.

Copies of the Audit Statement were distributed at the AGM and are available for viewing in the members-only section of the Society website, <www.spums.org.au>.

Guy Williams

The South Pacific Underwater Medicine Society Ltd
Balance Sheet as at 31 December 2007

Members' funds	**2007**	**2006**
Balance at 1 January 2007	112,499	130,161
Surplus/(Deficiency) for year	7,068	(17,662)
Balance 31 December 2007	$119,567	$112,499
represented by:		
Non-current assets		
SPUMS Website, at cost	32,285	32,285
Less, Provision for amortization	32,285	(27,715)
		4,570
Current assets		
St George General	39,177	
St George Annual Scientific Meeting	23,504	
ANZ Access Cheque Account	12,434	8,316
ANZ VZ Plus	44,464	89,698
ANZ SPUMS Annual Scientific Meeting	259	5,938
BNZ Achiever Savings		1,678
2008 ASM income less expenses in advance	196	
GST recoverable		2,299
	20,034	107,929
Total assets	120,034	112,499
Less, current liability		
GST owing	467	
Net assets	$119,567	$112,499

These are the accounts referred to in the report of D S Porter, Chartered Accountant.
Dated: 12 May 2008

Statement of Income and Expenditure for the year ended 31 December 2007

	2007	2006
Income		
Subscriptions & registrations	110,828	88,118
Interest	5,055	3,540
ASM 2006 refund	8,500	–
Sundry income	168	3,472
	124,551	95,094
Expenses		
Accounting fees	1,509	720
Administration, secretarial, etc	17,988	21,667
Amortization of website	4,570	10,741
ASM costs	7,895	8,911
Office expense	1,338	–
Donation	–	250
Stationery, printing, postage	–	1,173
Journal & editorial expenses	47,704	47,771
Committee expenses	11,264	965
Computer equipment	736	4,558
Maintenance website	8,043	–
Miscellaneous/subscriptions	2,006	396
Bank charges and card charges	5,548	3,320
Audit	2,920	2,100
Insurance	5,245	5,665
Telephone	382	608
Treasurer	335	3,911
	117,483	112,756
Surplus/(Deficiency) for year	$7,068	$(17,662)

Movements on bank balances for the year ended 31 December 2007

	2007	2006
Opening Balances		
ANZ bank		
- ASM account	8,316	7,162
- Access Cheque	5,938	53
- SPUMS ASM	1,678	2,812
- BNZ Achiever	89,698	95,165
- VZ Plus	105,630	108,992
add, Receipts	124,551	95,094
	230,181	204,886
less, PAYMENTS	110,343	98,456
Closing Balances		
St George – General	39,177	
− ASM	23,504	
ANZ Bank		
− Access Cheque	12,434	8,316
− VZ Plus	44,464	89,698
− SPUMS ASM		5,938
BNZ Achiever Savings	259	1,678
	$119,838	$105,630

NOTE: Receipts and payments may include Balance Sheet items which are not included in the Income and Expenditure statement.

Annual General Meeting of the Australian and New Zealand Hyperbaric Medicine Group, Saturday, 16 August, 2008, Sea World Resort, Gold Coast, Queensland

Opened: 1615 hr

1. Attendance
D Smart, D Wilkinson, M Bennett, B Spain, G Hawkins, B Webb, S Mitchell, C Meehan, S Squires, B Trytko, J Lehm.
The Chair welcomed Dr Tony Lee from Malaysia.

2. Apologies
B Long, M Walker, D Cooper, M Hodgson, M Davis, I Millar, B Wong.

3. Office bearers
Drs Smart and Wilkinson offered to remain in their respective positions of Chair and Secretary for another two-year term. With no other nominations, this was accepted unopposed. Dr Smart indicated this would be his last term as Honorary Chair and encouraged consideration from others for the role. There was discussion as to a potential benefit by staggering the terms of Chair and Secretary to provide some continuity. It would seem Dr Wilkinson is destined to keep writing these minutes.

4. Minutes of 2007 Annual General Meeting
No alterations notified, minutes accepted unanimously

5. Business arising
No issues.

6. Address by Chair of ANZHMG (D Smart)
6.1. Hyperbaric Medicine Funding in Australia:
The ANZHMG currently is funding for soft tissue radiation injury and non-diabetic hypoxic problem wounds under Item 3C with the Federal Health Minister's approval. This funding continues through 1 November 2007 to 31 October 2010. A new application for these two conditions is required by May 2009 to continue the funding. Members from ANZHMG will be required to contribute to this application so that it can be assessed. At the ANZHMG meeting, the process for this application and our strategy will need to be discussed. It was

resolved to submit two separate submissions for the 2009 MSAC funding.

6.2. SPUMS issues:
The incorporation of the EJUHM into the *Diving and Hyperbaric Medicine* Journal is taking place at present and it is almost complete. Congratulations to Mike Davis and others for the wonderful work they have done in this regard. There is clearly a lot of work still to be done with the amalgamation of the two societies and the one academic journal. It can only strengthen diving and hyperbaric medicine in Australia and New Zealand and certainly will lead to improved scientific content in the journal. I would like to also add to the SPUMS news that I have been appointed as Education Officer and Mike Bennett has been appointed as the President of SPUMS.

6.3. Research:
Congratulations to Ian Millar for the work done to date with the HOLLT trial. This has multiple centres involved around the world and ANZHMG encourages Australian facilities to participate in the trial.
The HORTIS trial was published earlier this year for radiation proctitis, demonstrating HBO as an effective treatment for this condition. Recruitment is continuing for other arms of the HORTIS trial and again ANZHMG encourages facilities in Australia to participate.

6.4. Wound Study:
This has now completed its second year; it is largely due to the energies of Dr Glen Hawkins and Dr Mike Bennett that this is continuing. We now have most facilities in Australia participating in the study and contributing data and this will be helpful to the cause in 2009 when we are submitting for funding for hypoxic non-diabetic problem wounds.

6.5. ANZHMG list of indications for hyperbaric oxygen treatment:
This will be due for review in 2009, having been published in *Diving and Hyperbaric Medicine* in 2007.

6.6. Support for the HTNA Conference:
Again I encourage all ANZHMG members to provide contributions to the conference. It is a unique event that needs to be supported and we congratulate Wesley Hyperbaric Facility in running this year's conference.

6.7. Courses in Diving and Hyperbaric Medicine:
Unfortunately over the last 12 months there have been some difficulties with the University of Auckland Postgraduate Diploma in Medical Science - Diving and Hyperbaric Medicine, leading to its disbandment. There have also been some difficulties with the Royal Adelaide hyperbaric medicine course. The remaining courses in Australia are the Prince of Wales introductory course in diving and hyperbaric medicine and also the Royal Australian Navy course at HMAS Penguin. It would be useful to discuss at the meeting the content and future of these courses in Australia given the difficulties that appear to be occurring.

6.8. Royal Hobart Hospital Department of Diving and Hyperbaric Medicine review:
The RHH hyperbaric facility is subject to an external review before it is guaranteed a place in the new RHH being constructed in the next five years. Dr Smart will speak to this issue at the meeting. Assistance may be required from other members of ANZHMG and also the UHMS.

6.9 Australian Standards:
Australian Standard AS2299.1 2007 was finally published last year. More recently AS 2299.2 has been reviewed and also the Training Standards Series 2815. A Dive Supervisor Training Unit 2815.5 has been included and is currently available for public comment.

7. MSAC report and Federal Government funding issues

Members were reminded that we have a temporary Medicare item number for the indications of non-diabetic hypoxic wounds and soft-tissue radiation injury and that this situation is due for review by MSAC. A tight time frame exists as, while Medicare approval is given until 2010, MSAC require our submission by May 2009 to allow for the review to take place. A strategy is required to re-approach this MSAC review; discussion points are included:

a) The review will be by the same people and so we can expect the same jaundiced reception.

b) In re-applying for funding for non-diabetic hypoxic wounds and soft-tissue radiation injury, the members present agreed that two applications should be made. (and so analysed separately).

c) Value in researching other group applications for guidance.

d) The wound indication is largely reliant on the National Wound Database.

e) Soft-tissue radiation injury is reliant on HORTIS.

f) The "Annane" paper was discussed, some concerns are expected to be addressed in an upcoming Cochrane review.

8. Hyperbaric problem wound study

An update of the data was presented at this meeting. The results are very impressive with about 82% remaining healed at 12 months. With some 250 patients enrolled, this is the largest collection of data of its kind and it forms the backbone of our application to MSAC. Just imagine how useful this would be if all hyperbaric facilities contributed. Once again, all units are asked to provide data; we already have numbers, we need to demonstrate cooperation and cohesion.

9. HORTIS

Recent article on radiation proctitis is awaiting publication but is available on the website of the *International Journal of Radiation, Oncology, Biology and Physics* (as an article in press).

10.ANZHMG/SIG list of approved indications for HBO_2

No new applications – current list endorsed unchanged.

11.Introductory course in hyperbaric medicine

Dr Bennett reported on the successful course run earlier this year with thanks to all who helped, particularly Dr Smart. The next course will be run 9th to 20th March 2009. A recent course candidate who found the course so useful that he wanted to do it again has been co-opted into helping rather than just attending! This course has the educational imprimatur of UHMS and ANZCA.

12.Hyperbaric facility accreditation

Facilities are encouraged to undertake accreditation under the structure promoted by the ANZCA SIG, Tasmania is about to undergo its re-accreditation review. While the ANZCA SIG will continue to be important for training issues, the ACHS was suggested as a possible route for facility accreditation. Some method of accreditation that could be undertaken between units was also discussed.

13.Australian Standards report

See Chair's report.

14.Diving and Hyperbaric Medicine

The Journal has now incorporated that of EUBS and the joint journal is now in circulation. There is a formal Editorial Advisory Board with equal representation, and an open and transparent editorial process. There is apparently lots of good material available. Despite a few administrative issues to resolve, we look forward to the high-quality publication and support its improved recognition amongst the scientific community. Well done.

15.Minimum dataset / registry developments

Some concern was expressed with using a Monash University-centred database or registry. This was related to issues such as control over, and use of, such information, particularly over the longer term. Dr Webb is currently working to consolidate database fields from different facilities into a common database using easily-accessed software (i.e., Microsoft).

16.Clinical trials for discussion

Discussed last night at a separate meeting – minutes will be forwarded separately.

17.HTNA issues

No issues.

18.Other business

18.1. SPUMS

Dr Bennett, as current President of SPUMS, raised the situation of the ANZHMG being a subcommittee of SPUMS. Does this relationship need to be reviewed and revised? The ANZHMG is asked to forward the President of SPUMS any suggestions for consideration.

Dr Bennett advised of the intent of the SPUMS Education Officer to review courses approved by SPUMS in the field of diving and hyperbaric medicine. The form of this review has not been defined.

A review of the medical examination to determine 'fitness-to-dive' for recreational divers has been ongoing by Drs Bennett, Mitchell and others. It is now at a stage where it can be sent to the SPUMS Committee for further discussion. It is anticipated that the Committee and invited guests/members will arrange a meeting to seek consensus on the form and content of this document. It appears a date in November is contemplated. If anyone feels they need to be there also, they are asked to discuss this with the President, Dr Bennett.

18.2. UHMS

Dr Hawkins wished to notify that he was now Chair of the International Affairs Committee. Together with Dr Bennett as Vice-President of UHMS and Dr Wilkinson on the Membership Committee, we do have good representation at the UHMS. If you are currently an international member of UHMS and feel your views are not adequately considered, or are not currently a member of UHMS but might consider such if certain performance criteria were achieved, then you are encouraged to talk to any of these people. Whilst we have plenty of work to do on our own turf, the value of an international body representing hyperbaric practice cannot be overstated.

18.3. Townsville.

Dr Webb advised that the Hyperbaric Unit at Townsville had received approval for a 12-month training job from the College of Emergency Medicine (6 months Hyperbaric/ED). He was congratulated by the group on this achievement. Please contact Dr Webb if interested.

18.4. Clinical Indicators

Dr Lehm raised an interest in developing clinical indicators for hyperbaric practice similar to those developed in anaesthesia. These would have quality assurance and benchmarking roles. Suggestions and discussion have been referred to the ANZHMG chat line.

Closed: 1740 hr

David Wilkinson
Honorary Secretary, ANZHMG

ANZCA 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
Chair, ANZCA/ASA Special Interest Group in Diving and Hyperbaric Medicine

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The
SPUMS
website is at
www.spums.org.au
Members are encouraged to log in

SPUMS

37th Annual Scientific Meeting

24–30 May 2009

Themes

Diving, Flying and Space Exploration

Future synergies in Diving Accident Management

Ear Injuries and ENT Workshop

The ENT workshop will cover ENT diagnostic dilemmas in divers, practical case examples of ear injuries, principles and practical use of Tympanometry.

Keynote Speaker

Professor Bruce Spiess MD, FAHA

Bruce Spiess is Professor and Chief of Cardiothoracic Anesthesia and Director of Research in the Department of Anesthesiology at Virginia Commonwealth University. As Director of the Virginia Commonwealth University Reanimation Engineering Shock Center (VCURES), he is researching perfluorocarbons as blood substitutes and their potential in treating decompression illness and gas embolism. Professor Spiess also conducts research into decompression sickness and submarine escape with the United States Navy, and is working with NASA on decompression sickness in astronauts.

Abstracts

Abstracts for presentation should be submitted before March 31st 2009 as a Word file of up to 250 words (excluding references – four only) and with only one figure. Intending speakers are reminded that it is SPUMS policy that their presentation is published as a full paper in *Diving and Hyperbaric Medicine.* The Editor will contact speakers prior to the meeting.

Papers should reflect the theme of the conference: Diving, flying (including aeromedical retrieval), space exploration, future synergies in diving accident management, systems of care and treatment.

If you wish to present a paper please contact the Convenor.

SPUMS 2009 Convenor

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Mobile: +61-(0)419-508577

All travel, accommodation and diving enquiries to:
<allways@bigpond.com.au>

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:
A number of rooms have been reserved. Please book early.
The accommodation booking service is provided by Aberdeen Convention bureau.

Contact details:

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
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EUBS European Underwater and Baromedical Society

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

EUBS General Assembly, Graz, 5 September 2008 at 1230 hr

The Vice President Peter Germonpré welcomes the members and informs them that he will substitute as Chairperson for President Alf Brubakk who could not join the meeting for personal reasons.

Minutes of General Assembly 2007

The agenda and the minutes from the General Assembly 2007 are accepted.

Status of current Annual Scientific Meeting

Dr Beatrice Ratzenhofer, Secretary-General of the 33rd Annual Scientific Meeting informs about the meeting. 162 participants attended the meeting, which shows a growing interest in the field. The meeting is considered as a success.

Zetterström Award:

The Zetterström Award has been awarded by the Zetterström Committee (composed of J Schmutz, M Cimsit, E Jansen, and J Kot) to E Gempp, JE Blatteau, E Stephant, JM Pontier, P Constantin, C Peng for their poster entitled: *"Spinal cord decompression sickness and MRI in scuba divers"*.

Travel Grants:

There were six applications for travel grants. Only two of them have completely fulfilled the conditions for reimbursement: submission of a minipaper to be published in the Proceedings or submission of a full paper to *Diving and Hyperbaric Medicine* (DHM). The ExCom proposes to invite the other four applicants to submit a paper to DHM by the end of September. If their paper is accepted for review, they will be reimbursed by bank transfer for their travel costs and congress fees.

EUBS publications: journal and website

The most visible change of our Society in 2007–2008 has been the Journal: *Diving and Hyperbaric Medicine*, a merger of the *SPUMS Journal* and *EJUHM* into a single quarterly publication. Peter Müller informs the membership that DHM has started its career as planned during last year's conference. The Editorial Bconsists of SPUMS and EUBS members. All EUBS members have received the first two issues and there are sufficient articles in the editorial pipeline to foresee a smooth run. At this moment, DHM is still mainly SPUMS-orientated. More articles from EUBS members are necessary in order to increase EUBS representation.

DHM is now indexed both in Embase and in the Scientific Citation Index Expanded (SCIE), which makes it a valuable journal to publish in for those seeking academic recognition. To obtain indexing in Medline, even more high-quality publication input is needed, and all EUBS members are again encouraged to do so. Instructions for authors are downloadable from the EUBS website.

A joint meeting between SPUMS and EUBS has been suggested.

The new version of our website will be online in September 2008. Among the new changes, membership application will be possible online; there will be a 'membership only' section with individual password access where members can manage their personal data, including membership fees and also have access to the membership directory. Last but not least, free access to the GTÜHM bibliography data base. Access is of course related to payment of the membership fee and to a valid e-mail address. Members are therefore requested to send an e-mail to <secretary@eubs.org>. They will then receive their access password.

Votes and elections

103 ballots have been returned and all changes in the Bylaws proposed by the ExCom have been accepted. The Bylaws will be updated on the website.

Peter Knessl was elected Member at Large 2008–2010. The EUBS wishes to express their thanks to Armin Kemmer, who is leaving the ExCom after three years.

Next year a ballot is scheduled for the election of Vice-President and for a Member at Large. Proposals have to be sent according to the Bylaws to the Honorary Secratary <joerg.schmutz@hin.ch>

– 100 days before the 2009 Annual Meeting
– 15 signatures of sponsors accompanying a written consent to serve by the candidate.

Financial

The financial report was prepared by Tricia Wooding and audited by Sarah Munday from DDRC in Plymouth. The EUBS ExCom expresses their thanks to DDRC for graciously providing this audit. The financial report is accepted by the GA. It will be published in the September issue of DHM. EUBS has 315 members currently, 15 new members came in last year, reflecting the interest of young scientists in our Society. More than 70 members paid by PayPal. Like the previous years, only 200 members have paid their annual dues up to this meeting. One reason may be the unclear cut of the membership year, which the ExCom wishes to make explicit here.

Each membership year starts on 1 July of the current year, and ends on 30 June of the following year. This means that everyone who paid up until today is a member for the 'Year 2008', until 30 June 2009. Because many members still pay at the Annual Scientific Meeting, directly to our Treasurer, members will benefit from membership advantages (the Journal, access to the members area of the website) until September 15th of 2009. Their membership will then be suspended unless they pay membership fees before 15 September. The proposal is accepted.

Due to high banking costs on the Society's Sterling account, it is proposed to the general assembly to enquire about a Euro account with lower banking and exchange costs. This is accepted by the members.

The necessity of an increase in membership fee, mainly due to the changes in the Journal, the increased expenses in the form of student travel grants, the Zetterström Award, administrative costs etc., was accepted by last year's General Assembly. Based on our calculations and comparison with other societies, an amount of 100 Euros for regular membership, 75 Euros for undergraduate membership and 600 Euros for corporate membership, is proposed. Corporate members will receive in return one copy of the Journal, a logo on the website and in the Conference Proceedings and reduced registration fee at the Annual Meeting for two persons. This proposal is also accepted by the members.

Next meetings

The next meeting will take place in Aberdeen, 23–28 August. John Ross will be the Secretary General.

Following meetings are scheduled for:

2010 Turkey (Istanbul)
2011 Poland
2012 Serbia
2013 France (Bordeaux)
2014 Belgium

A guideline for meeting organizers will be available in the near future on our website. This guideline is based on the original document produced by Hans Ornhagen in the 1980s and improved over the years by the ExCom.

J Schmutz
Honorary Secretary, EUBS

Important EUBS website update

A 'Members' section has been added to the EUBS Website, providing you with the following extra benefits:

- Secure access to the full-text literature database of the German Society for Diving and Underwater Medicine (GTUEM)
- Access to your own membership data and membership status
- The ability to renew your membership online with a few mouse-clicks
- Browsing the EUBS Membership Directory (who is who, and how to contact them)
- A 'Discussion Forum' to make contacts, exchange information and more...

For your first login, you will need a password, sent to you by the EUBS Secretary. If you have already provided a valid e-mail address, you can use the option "*I have forgotten my password*" when you log in and a new password will be sent to you automatically. If you have not yet provided a valid e-mail address, please send a mail to the Secretary and a password will be mailed to you.

Peter Germonpré, EUBS Webmaster

Scott Haldane Foundation Diving Medicine Education

In collaboration with the Dutch Society for Diving Medicine
Diving Medicine Courses, first semester 2009

24 January Refresher Course: Pulmonary function testing for divers. Rotterdam, The Netherlands
24 January – 8 February Basic Course: Diving medicine for ENT specialists. Manado, Sulawesi
28 March & 3–4 April Basic Course in Diving Medicine Amsterdam, The Netherlands
19 June Advanced Course: Evidence-based diving medicine. Driebergen, The Netherlands

For further information and registration:
Website: <www.scotthaldane.nl>
E-mail: <info@scotthaldane.nl>

5th Karolinska Postgraduate Course in Clinical Hyperbaric Oxygen Therapy

Date: May 7, 2009
Venue: Stockholm, Sweden

The course will cover past, present and future clinical trials with a focus on 'evidence-based medicine'

Speakers include: Stephen Thom, Neil Hampson, Lin Weaver, Simon Mitchell, Jon Buras, Ian Millar, Dirk Bakker, Michael Bennett, Daniel Mathieu, Tom Hunt, Christer Hammarlund and Dick Clarke.

Registration & Information:
<www.oxygeninfection.se>
Contact person: <folke.lind@karolinska.se>

Oxygen and infection

ESCMID EUROPEAN SOCIETY OF CLINICAL MICROBIOLOGY AND INFECTIOUS DISEASES

European Committee for Hyperbaric Medicine

Dates: May 8-9, 2008
Venue: Stockholm, Sweden

A European Committee for Hyperbaric Medicine Conference, endorsed by the European Society of Clinical Microbiology and Infectious Diseases, to review the role of oxygen in infectious diseases from basic science to clinical practice. The effects of anoxia, hypoxia, normoxia and hyperoxia on microbes, antibiotics, leukocyte bacterial killing and inflammation will be examined.

Registration & Information:
<www.oxygeninfection.se>
Contact person: <folke.lind@karolinska.se>

The poetry doctor

A pearl of wisdom

An oyster is an amazing creature
For its special coping feature,
When grit irritates it under the skin
It does not rant or fester within.
Instead it accepts it and over a time
Adorns it with layers and layers of lime
Whose slow-laid deposits in the finest of swirls
Creates a perfection we know as a pearl.
So let us learn from this plain creature's strife
That any annoyance is a lesson in life.
Instead of reacting we should pause and know
This grit is a gift from which we can grow.

John Parker
<wwwthepoetrydoctor.com>

The Hyperbaric Research Prize

The Hyperbaric Research Prize encourages the scientific advancement of hyperbaric medicine and will be 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>

Situations vacant

The Alfred Hospital, Australia

Registrar / Fellow in Diving and Hyperbaric Medicine

Applications are sought for 2009/2010 appointments as full-time Registrar in Hyperbaric Medicine at The Alfred Hospital in Melbourne, Australia. Usual fellowship durations are 6–12 months (February to July and/or August to January inclusive). The Alfred Hyperbaric Service is an integrated department within a major academic teaching hospital and operates a large, modern, rectangular, multiplace hyperbaric chamber. It provides around 4,000–5,000 treatments per annum to a range of ambulatory through to critically ill patients each year, including around 30 divers, 30 necrotising soft-tissue infection cases and 150 acute trauma and ischaemia cases in addition to problem chronic wound and post radiotherapy cases. Opportunities can be provided to attend formal courses and to undertake research.

For further information contact:
Dr Ian Millar, Unit Director
Phone: *+61-(0)3-9076-2269*
E-mail: *<i.millar@alfred.org.au>*

The Australia and New Zealand Hyperbaric Medicine Group

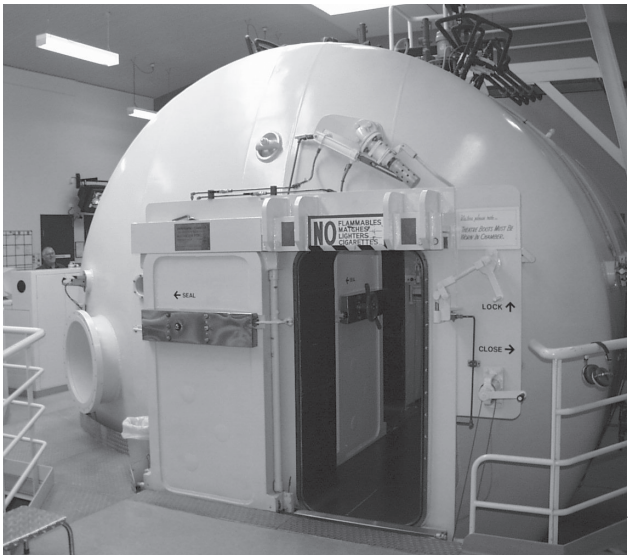
Introductory Course in Diving and Hyperbaric Medicine

Dates: 9 to 20 March 2009
Venue: Prince of Wales Hospital, Sydney, Australia

Course content includes:
- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen (including necrotising infections, acute CO poisoning, osteoradionecrosis and problem wound healing)
- Wound assessment including transcutaneous oximetry
- Visit to HMAS Penguin
- Marine envenomation
- Practical sessions including assessment of fitness to dive

Contact for information:
Ms Gabrielle Janik, Course Administrator
Phone: +61-(0)2-9382-3880
Fax: +61-(0)2-9382-3882
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

This course is approved as a CPD Learning Project by ANZCA – Cat 2, Level 2 – 2 credits per hour (Approval No. 1191)



The Prince of Wales chamber, Randwick, Sydney

This 'grand old lady' of Australian hyperbaric chambers will soon be decommissioned as PoW proceeds to install a new chamber on the same site. I hope that Associate Professor Mike Bennett and his colleagues will be able to provide us with a news item on their major new developments in due course for the Journal.

Undersea & Hyperbaric Medical Society

Annual Scientific Meeting 2009
Preliminary notice

Dates: 25 to 27 June 2009
Venue: Crowne Plaza Resort
Los Cabos-Grand Faro
Blvd San Jose s/n, Zona Hoteler
San Jose del Cabo, 23400 Mexico
For further information:
E-mail: <uhms@uhms.org>
Website: <www.uhms.org>

Asian Hyperbaric & Diving Medical Association

5th Annual Meeting – preliminary announcement
Dates: 25 to 27 September 2009
Venue: Goa, India
Further details (academic programme, registration fees and hotel tariffs) to follow soon
For further information:
E-mail: <ahdma.goa@gmail.com>

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

Hyperbaric Nursing Course

Department of Diving and Hyperbaric Medicine
Fremantle Hospital and Health Service

Dates: 30 Mar – 3 April 2009
For further information contact:
Sue Thurston, Clinical Nurse Manager
Phone: +61-(0)8-9431-2233
Fax: +61-(0)8-9431-2235
E-mail: susan.thurston@health.wa.gov.au

DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA

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

Instructions to authors

(revised August 2008)

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 should 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 columns are preferred as tab-separated text rather than using the columns/tables options or other software and each table submitted double-spaced as a separate file.
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 (> 8 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 July 2003. Website for details: <http://www.icmje.org/index.html>). In this system, references 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 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.

There should be a space after the semi-colon and after the colon, and a full stop after the journal and the page numbers. Titles of quoted books and journals should be in italics. Accuracy of the references is the responsibility of authors.

Any manuscript not complying with these requirements will be returned to the author before it will be considered for publication in *Diving and Hyperbaric Medicine*.

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 and in Vol 38, June 2008 issue.

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1-800-088-200 (in Australia, toll-free)
+61-8-8212-9242 (International)

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

SOUTH-EAST ASIA
+65-750-5546 (Singapore Navy)
+63-2-815-9911 (Philippines)
+605-681-9485 (Malaysia)
852-3611-7326 (China)
010-4500-9113 (Korea)
+81-3-3812-4999 (Japan)

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

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

SOUTHERN AFRICA
0800-020-111 (in South Africa, toll-free)
+27-11-254-1112 (International, may be called collect)

The DES numbers 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

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