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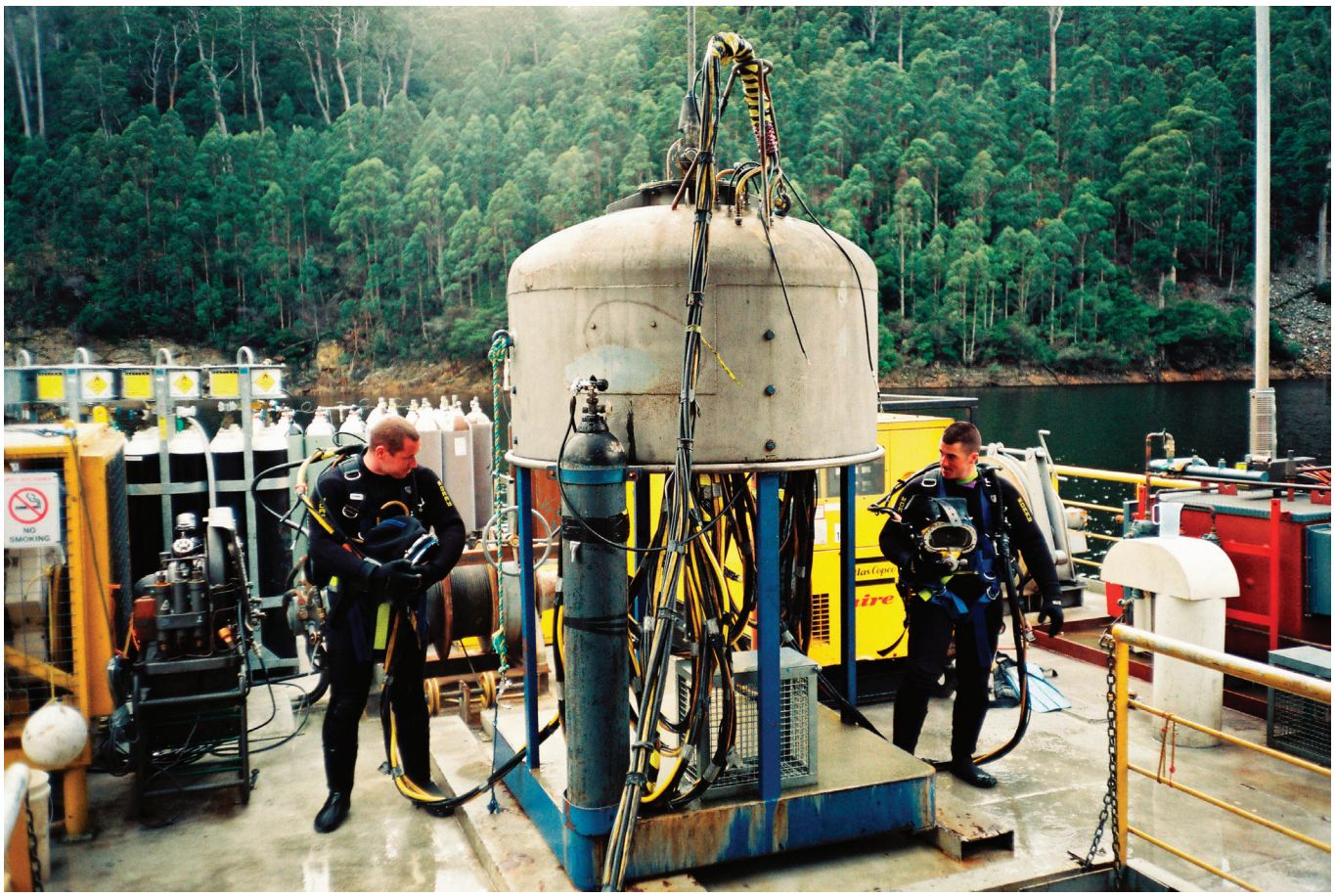

Aerobic fitness and scuba diving

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Improving diving medicine courses

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The Editor's offering

Diving medicine education

In Australia, diving medicals are performed by doctors who have undertaken a course in diving medicine. This principle is now enshrined in the SPUMS Diving Doctors List, in Australian recreational and occupational diving standards and in Queensland law. Similar requirements exist elsewhere, such as within the European Union, but in many countries, including the USA and New Zealand, the examining doctor is not required to have any knowledge or training in diving medicine. The value of so-called 'short' courses in diving medicine has been questioned over the years. The survey by Simpson and Roones, for all its limitations, has been studiously ignored in addressing whether a few pressure-cooker days with some erudite diving medical pundits really provides an adequate platform for physicians to examine divers.[1] Carl Edmonds raises this question again in his article on the second tier of diving medical training.

Many readers may be confused about the training and education (there is a clear distinction between these two terms) available in diving medicine. In Australia, this has a four-tier structure. In the first tier are the four- to five-day courses, available in several centres, designed to teach GPs how to examine divers for 'fitness to dive'. Those completing the course can then add their names to the SPUMS Diving Doctors List, to which the Australian standards and Queensland legislation defer. It is the value of these courses that Simpson and Roones questioned. Similar courses are run by DAN-USA and in many European countries.

The second tier are the two-week diving medicine courses provided by the Royal Australian Navy and the Royal Adelaide Hospital, which qualify doctors to undertake commercial-diving medicals. It is the content and philosophy of these courses that Dr Edmonds discusses in this issue, based on his wealth of experience in teaching on such programmes. Slightly off to one side, because of its greater hyperbaric medicine content, is the course provided by SPUMS itself, the annual two-week course in Diving and Hyperbaric Medicine run by the ANZ Hyperbaric Medicine Group with faculty drawn from throughout Australia and New Zealand. Graduates of this course are also listed on the Diving Doctors List as qualified to undertake commercial-diving medicals in Australia.

The third tier is the SPUMS Diploma (or the now-defunct University of Auckland Diploma) in Diving and Hyperbaric Medicine (DHM). As well as attending a two-week course, the Diploma candidate must write a dissertation related to DHM and have the equivalent of six months' full-time experience working in a hyperbaric medicine unit. The final requirement precludes many medically qualified members of SPUMS from ever progressing to this level as such medical jobs are few and far between and do not fit well with general practice or training in many hospital specialties. The Auckland distance-learning diploma programme was designed to overcome this barrier, but has been curtailed on financial grounds after only a few years, having failed to engender sufficient interest in the international diving and hyperbaric medical community.

The fourth tier is the Certificate in DHM of the ANZ College of Anaesthetists. The requirements for this qualification are set out in this issue on page 155. The Certificate is now regarded (at least by those who possess it, including the writer) as the specialist qualification in this field. Interestingly the College does not allow certificate holders to place this post-graduate qualification after their name, somewhat of a 'Clayton's' qualification. The ANZCA Certificate is open to graduates of any specialty.

The first three tiers are similar to the system being established in the European Union by the European Diving Technical Committee and the European College of Hyperbaric Medicine (ECHM). All ECHM courses gain approval from the European College of Baromedicine, University of Malta. Level I requires a one-week course, designed like those in Australasia, for diving medical examiners. A Level II physician will have further training and some experience in both diving and hyperbaric medicine, and is qualified to medically supervise diving operations and examine commercial divers, and this is the entry level for hyperbaric physicians. At Level III (specialist level) the training objectives have yet to be clearly defined, but will involve more extensive university-based education somewhat similar to components of the defunct University of Auckland programme. There is no reciprocity between the Australian and European standards for medical training in diving medicine, though interestingly the South African programmes have been approved by the Europeans.

Dr Edmonds' review of the second-tier courses is timely because of the rapid developments throughout the world. Courses for doctors appearing on the SPUMS Diving Doctors List are approved by the Society's Academic Board. However, such reviews have not occurred for many years, to my knowledge, and it is time this was done. Nor have any moves been made to achieve reciprocity of qualifications with the international diving medicine community. This is urgently needed at all levels, and I hope that Dr Edmonds' article will generate sufficient interest to stimulate new activity in this important area of our Society's activities.

Reference

1 Simpson G, Roones D. Scuba diving medical examinations in practice: a postal survey. *Med J Aust*. 1999; 171: 595-8.

Michael Davis

Front page photo of two divers under training preparing for a wet bell run at the deep-water Lake Cethana, Tasmania, was taken by Dr David Smart.

Review articles

Aerobic fitness and underwater diving

Neal W Pollock

Key words
Aerobic capacity, exercise, oxygen consumption, fitness to dive, scuba, review article

Abstract

(Pollock NW. Aerobic fitness and underwater diving. *Diving and Hyperbaric Medicine*. 2007; 37: 118-24.)
Physical fitness is necessary to ensure that the normal and emergent needs of diving can be met. Reserves of both strength and aerobic capacity are important. Aerobic capacity (aerobic fitness or $VO_{2\ max}$) is defined as the maximum amount of oxygen that can be consumed per unit time. Alternatively, it can be described as metabolic equivalents (MET), dimensionless multiples of the oxygen consumption of an assumed resting metabolic rate (3.5 $mL.kg^{-1}.min^{-1}$), yielding a range of 5–25 MET in the healthy population. A minimum capacity as high as 13 MET has been proposed for diving qualification. While limited, the available research data suggest that this is an unrealistically high threshold. A minimum capacity in the range of 7–10 MET may be more appropriate. In recognition of the importance of physical fitness and the decline associated with normal ageing, training programmes should promote awareness of the problems and risks that may be associated with low levels of fitness and the benefits of enhanced fitness. Training to understand and increase aerobic capacity should be encouraged.

Introduction

Poor physical fitness is a growing problem, particularly in more developed nations. The United States Surgeon General Report indicated that more than 60 per cent of adults perform less than the recommended amount of regular physical activity. Approximately 25 per cent of the adult population is classified as completely inactive.[1] Similarly, two-thirds of Canadians aged 22–55 are not physically active enough to meet recommended guidelines.[2]

Body mass index (BMI) is an index of height–weight proportionality commonly used to estimate adiposity. While a rise in BMI can indicate an increase in muscle mass, it more commonly reflects an increase in the body fat fraction, so it has some utility as a population scale estimate. BMI equals weight in kg divided by the square of height in metres. The following categorisation is commonly applied: underweight <18.5 $kg.m^{-2}$; normal weight 18.5–24.9 $kg.m^{-2}$; overweight 25.0–29.9 $kg.m^{-2}$; obese >30 $kg.m^{-2}$. Based on BMI data, obesity in the United States has more than doubled from 1960 to 2004, and now exceeds 33 per cent.[3]

While fitness data are extremely limited for the diving community, high BMI scores establish a basis for concern. Fatality records available to Divers Alert Network (DAN) America include annual BMI-estimated incidence of obesity ranging from 41–55 per cent for 2002–2004 (Figure 1).[4] Medical forms reviewed from Scottish Sub-Aqua Club divers indicated a surprisingly low BMI-estimated incidence of obesity of 2.5 per cent but a significant rise in mean BMI scores from 1991 to 1998.[5] Self-reports of height and weight for 346 Australian club divers responding to a health survey indicated a 12.7 per cent BMI-estimated incidence of obesity.[6]

Physical fitness can be described as a function of strength, flexibility/agility and aerobic capacity. Divers clearly benefit from robust levels of physical fitness. Strength and flexibility/agility are required to don, carry and manipulate equipment, most noticeably through the entry and exit phases of a dive. Aerobic capacity is required to meet the energetic demands of physiological work done pre-dive, at the surface and underwater. The absolute capacities required will vary with the rigour of the environment, the equipment and the nature of the dive. Questions concerning readiness become more important given the general declines of physical fitness observed in the population and the potentially long-term involvement of individuals in diving.

Reserves of both strength and aerobic capacity are important to manage expected and unexpected demands of diving. The adequacy of strength for diving is often evaluated through simple management of equipment on every dive. Aerobic capacity is typically not measured but simply discussed conceptually. This paper will review issues related to aerobic capacity and diving.

The need for aerobic fitness

Adequate physical reserves can be critical when quick, effective responses may keep small problems from becoming serious ones. The draw on cardiorespiratory systems begins with the donning of gear and continues with water immersion and the concomitant shift of blood volume to the central circulation. Little research is available that separates the

contribution of stressors such as physically constraining equipment, entry/exit demands, water immersion, breathing resistance, thermal stress, water condition, compression, decompression, buoyancy control, water resistance, psychology and any additional emergent conditions. While difficult to study, the individual and synergistic effects of the stressors must still be managed on every dive.

Robust levels of aerobic fitness reduce the strain produced by diving (or other) stressors. Instead of representing a large portion of the physiological reserve of an unfit diver, the strain represents a relatively smaller portion for the fitter diver. Aerobic fitness may offer additional benefits to divers. While the data are limited, there is some evidence that elevated aerobic fitness can reduce decompression-induced bubble formation in animals and humans and reduce the incidence of serious decompression sickness following provocative exposures in animals.[7–9] Further investigation is required to reconcile these findings with others concluding that aerobic fitness was not related to bubble formation.[10]

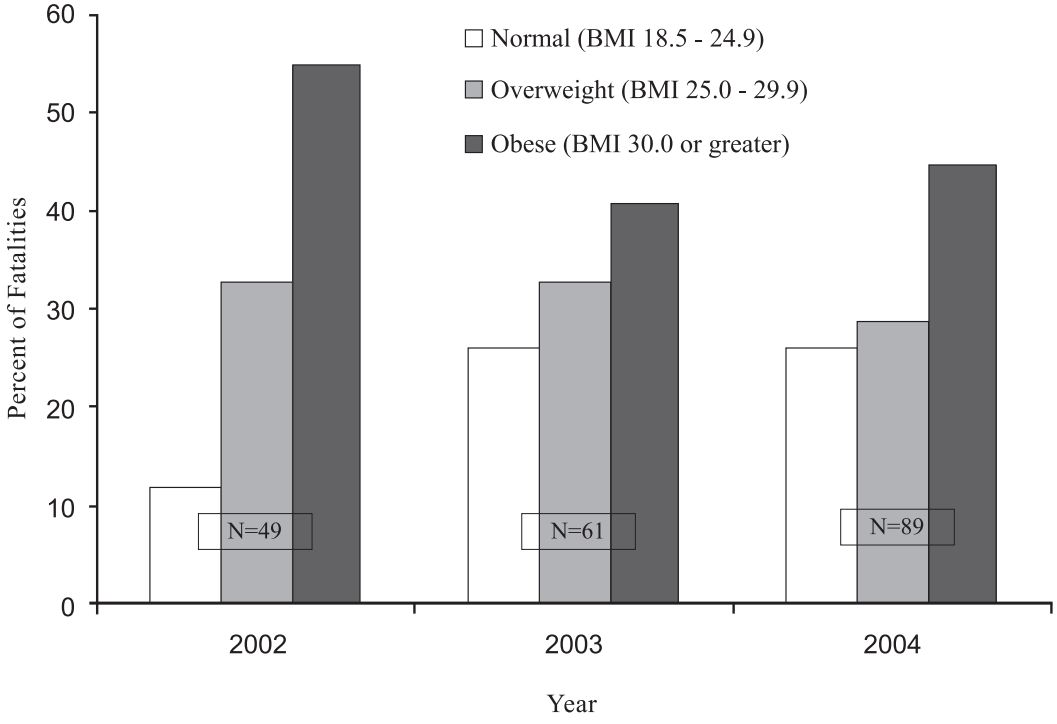
Measuring aerobic fitness

Aerobic fitness (aerobic capacity or $VO_{2\ max}$) can be defined as the maximum amount of oxygen an individual is capable of consuming per unit time. Definitive testing requires the measurement of expired gases throughout a progressive exercise test that ends when the subject can no longer continue. The progression is generally selected so that exhaustion is reached within 8–13 minutes, before issues of thermal stress become a confounder. Such tests are temporarily exhausting, but a relatively quick and non-invasive means of evaluating a key aspect of physical fitness.

Aerobic capacity testing is most commonly completed with treadmill running or stationary cycling. Treadmill tests typically produce the highest values because of greater whole-body involvement. For all but trained cyclists, cycling tests may produce maximal scores 5–10 per cent lower than treadmill tests.[11] Cycling tests may be more appropriate for divers given the similar focus on lower-body exercise. In-water maximal testing is also possible and sometimes available to special groups, but few facilities are set up to do so.

Strategies have been developed to estimate aerobic capacity by a variety of techniques, typically based on regression formulae developed from maximal testing of sample groups. The University of Houston non-exercise (Houston) test, for example, predicts $VO_{2\ max}$ based on height, weight, age and self-reported patterns of physical activity over the previous month on a 0–7 scale.[12] Similar predictive tests have been developed based on submaximal exercise testing. Two of the most well known are the one-mile walk and the up-and-down step tests.[13–16] Predictive tests are generally quite weak on an individual basis. This may be a case of the individual being dissimilar to the group used to develop the formulae. This is a substantial problem for the classic tests that are based on normative data collected 40 to 50 years ago or for

Figure 1
Classification of DAN recreational diver fatalities by BMI ($kg.m^{-2}$) for 2002, 2003 and 2004[4]



tests developed with somewhat idiosyncratic groups. Small deviations in procedure can also have dramatic effects on the prediction. The step test is a prime example, in which a slight variation in the step height will markedly influence the heart rate response and subsequently the estimate. Maximal testing is far superior for true quantification of aerobic capacity. The greatest strength of predictive tests is their field utility. Repeated testing with a standard protocol can be useful to identify patterns of change over time, even if the absolute scores are not accurate.

Classically, $VO_{2\ max}$ was reported as the whole-body rate of consumption of oxygen per minute ($L.min^{-1}$). Problematically, this measure is insensitive to body size, a major source of inter-individual variability. The aerobic fitness of a smaller person is greater than that of a larger person with the same absolute $VO_{2\ max}$. The shortcoming is partially resolved by reporting $VO_{2\ max}$ indexed to body mass, specifically millilitres of oxygen consumed per kg body mass per minute ($mL.kg^{-1}.min^{-1}$). Further resolution is possible by indexing oxygen consumption to lean body mass to discount metabolically inactive fat, although this method is not as widely used.

The numbers and units associated with $VO_{2\ max}$ results may seem unnecessarily complicated to the uninitiated reader. A simpler way to present the results is to use metabolic equivalents (MET). The MET is a dimensionless value describing a subject's aerobic capacity relative to the standard assumed metabolic rate of a resting person (3.5 $mL.kg^{-1}.min^{-1}$). Quite simply, $VO_{2\ max}$ in the same units is divided by 3.5. For example, an individual with a $VO_{2\ max}$ of 35 $mL.kg^{-1}.min^{-1}$ would have a 10 MET capacity (MET_{max}). Gender is not traditionally considered in computing MET values. However, given their normally higher percentage of body fat, women are inappropriately penalised with the standard computation. A correction can be made for females by dividing oxygen consumption by 3.2 instead of 3.5. The normal range of MET_{max} in the healthy population is approximately 5–25 MET. Table 1 lists fitness levels associated with various $VO_{2\ max}$ and MET_{max} capacities.[17]

What level of aerobic fitness has been prescribed for divers?

Minimum swim-test performance is commonly required for entry into dive training programmes. Such tests, however, are a much better indicator of swimming ability than fitness. Minimum MET_{max} capacity can be used as a measure of aerobic fitness to complement medical evaluations and swim-test performance. It has been argued that candidates should have a capacity of 13 MET to be allowed to dive.[18] The basis for this was US Navy research indicating that the maximum speed a fully equipped diver could attain was 1.3 knots (1.8 $km.h^{-1}$) at a work rate of approximately 13 MET. Realistically, this is a demanding standard given the normal level of effort involved with diving. Swimming at a more typical speed of 0.5 knots required an effort of only 3 MET in the same equipment. While emergent conditions may produce a transient demand for great power output, it is unclear if a capacity of 13 MET is a reasonable threshold.

How fit are divers?

While accident data suggest that inadequate fitness is a problem in the diving population, determining the typical fitness level of divers who do not have problems would be informative. Unfortunately, extremely limited data of this type are available. The best insight might come from published research studies involving actual divers for whom aerobic capacity was measured with maximal tests. Table 2 includes a sample of such studies. Representing a range from sport to professional divers, subjects were described as experienced in all but two of the studies.[19,20] The mean aerobic fitness ranged from 37–57 $mL.kg^{-1}.min^{-1}$ (10.6–16.3 MET). The threshold of 13 MET was exceeded by the group mean in only six of the 14 studies described.

The lowest individual $VO_{2\ max}$ score reported was 16 $mL.kg^{-1}.min^{-1}$ (4.6 MET).[24] Inclusion criteria for this study were age greater than 40 years, at least two years of diving experience, completion of at least 10 dives per year with depth greater than 20 metres' sea water, and no previous diving accidents. All of the subjects were French diving instructors. The lowest $VO_{2\ max}$ of a group of sport divers was reported as 16.4 $mL.kg^{-1}.min^{-1}$ (4.7 MET).[8,19] Estimating the minimal individual $VO_{2\ max}$ as the mean minus two standard deviations for the 12 studies in Table 2 that provide means but not minimum values yields a range of 21.0–43.0 $mL.kg^{-1}.min^{-1}$ (6.0–12.3 MET). The median value was 31.9 $mL.kg^{-1}.min^{-1}$ (9.1 MET)

The potential impact of saturation diving on aerobic capacity was evident in one study reporting a 15.2 per cent decline in $VO_{2\ max}$ following saturation dives ranging from 18–28 days total duration, which dropped from 14.8 ± 1.3 to 12.7 ± 1.4 MET.[23] Another study found no significant change in $VO_{2\ max}$ after 19.3 day saturation dives (from 12.1 ± 2.0 to 11.5 ± 2.1 MET).[29] The difference in the activity level of the two groups is unclear but this was likely a factor. Normally, diving does not typically include substantial aerobic demands, thus making it more difficult for active divers to maintain high aerobic capacities.

Table 1
Physical fitness categories[17]

Fitness capacity description	$VO_{2\ max}$ ($mL.kg^{-1}.min^{-1}$)	MET_{max}
Reasonable for inactive lifestyle	>25	>7
Reasonable for modestly active lifestyle	>35	>10
Optimal for lifetime fitness	>50	>14
Athletically competitive	>60	>17
Sub-elite to elite (sport-specific)	>70	>20

How aerobically fit should divers be?

Table 1 describes a capacity of 14 MET as optimal for lifetime fitness. This is a laudable goal for all to pursue. Aerobic fitness typically peaks in the early to mid-20s and will decline thereafter. Statistical models have predicted a decline of one per cent per year for both athletic and sedentary individuals.[32] A longitudinal study revisited individuals at 40 years of age who had been elite athletes at 25 years of age. Those remaining the most active in the intervening years experienced an average decrease in aerobic fitness of 0.7 per cent per year. Those who were least active experienced an average decrease of 1.6 per cent per year.[33] Combining the highest initial level of aerobic fitness with dedicated efforts to slow the rate of decline provides the best strategy for postponing the point at which reduced physical capacity will be incompatible with desired activities.

The age-related decline of fitness is an important issue since diving is a lifetime activity for many enthusiasts. The lack of expiration of basic diving certification allows individuals to continue diving through potentially significant physical changes. The physical capacity possessed in early years may erode substantially over time, though, in many cases, increased skill levels can partially compensate for declining fitness. A skilled diver who maintains excellent neutral buoyancy, good spatial awareness and use of currents, and a high degree of comfort underwater can require dramatically less energy to complete a given dive than a novice might. Not all divers, however, learn to dive early. Those coming

Table 2
Measured aerobic capacity of divers (mean ± SD or median or range)

Lead author (ref)	Divers	Subjects	$VO_{2\,max}$ ($mL.kg^{-1}.min^{-1}$)	MET_{max}	Mode	Age (yr)	BMI ($kg.m^{-2}$)
Thompson (1984a)[21]	Commercial	148 M	46 ± 9.0	13.1 ± 2.6	?	30 ± 5	25 ± 3
Thompson (1984b)[22]	Commercial	10 M	43.8 ± 4.5	12.5 ± 1.3	?	?	?
Thorsen (1990)[23]	Experienced	18	51.7 ± 4.6*	14.8 ± 1.3	Treadmill	28 (mean) (23–34)	24.3†
Carturan (2002)[19‡]	Sport	45 M	38.9 ± 10.8	11.1 ± 3.1	Cycle	37 ± 9.6	25.5†
Dujic (2004)[10]	Experienced	13 M	47.8 ± 4.7	13.7 ± 1.3	Treadmill	29.9 ± 5.0 (22–38)	21.5–29.0
Tripodi (2004)[24]	Experienced	27 M / 3 F	37 ± 8	10.6 ± 2.3	Cycle	48 ± 7.5	25.5†
Dujic (2005a)[25]	Military	10 M	41.2 ± 6.3§	11.8 ± 1.8	Cycle	35.1 ± 4.3 (29–41)	22.5–29.0
Dujic (2005b)[26]	Croatian Navy	12	41.2 ± 6.3	11.8 ± 1.8	Cycle	34.7 ± 4.1 (29–41)	25.8 ± 2.2 (22.2–29.0)
Almeling (2006)[20]	Certified	28 M	27.4–47.7‖	7.8–13.6	Cycle	39.9 (26–62)	24.4†
Dujic (2006a)[27]	Military	7	41.5 ± 9.6	11.9 ± 2.7	Cycle	30–39	25.9 ± 2.0
Dujic (2006b)[28]	Experienced	10 6	41.8 ± 6.4 47.3 ± 5.3	11.9 ± 1.8 13.5 ± 1.5	Cycle Cycle	34.4 ± 4.2 32.2 ± 5.2	25.9 ± 2.0 26.5 ± 2.4
Thorsen (2006)[29]	Commercial/ Saturation	8 M	42.3 ± 7.1	12.1 ± 2.0	Treadmill (6) Cycle (2)	41 (median) (29–48)	28.6 (median) (24.2–30.7)
Blatteau (2007)[30]	Trained military	16	51.7 ± 8.3	14.8±2.4	Treadmill	33.6 ± 3.7 (27–39)	21.0–27.1
Boussuges (2007)[31]	Trained military	20 M	57 ± 7	16.3 ± 2.0	Treadmill	33 ± 4	25 ± 2

* $VO_{2\,max}$ in $mL.kg^{-1}.min^{-1}$ derived from reported mean $L.min^{-1}$ by using the reported mean weight of subjects
† BMI derived from reported mean mass and mean height of subjects
‡ Study included individual data reported previously[8]
§ Mean $VO_{2\,max}$ reported in the text differed from the mean computed from the individual values also provided in the paper (the latter is shown here)
‖ $VO_{2\,max}$ imputed from minimum and maximum whole body measures ($L.min^{-1}$) converted to relative using the minimum and maximal body mass values, respectively, reported for the group

to diving later in life, when fitness may be more of an issue, may not benefit from the economy of experience. Similarly, other individuals may never develop high competence because of infrequent participation or personal limitations. Less physically competent individuals will be under much greater physiological and psychological stress.

The question of a reasonable requirement for aerobic fitness to dive is a difficult one when confounded by issues of experience. This was strongly debated more than 20 years ago when the recommendation for a minimum aerobic capacity of 50 $mL.kg^{-1}.min^{-1}$ (14.3 MET) for North Sea divers was suggested.[21,22,34,35] While the highest fitness possible is undoubtedly desirable, setting valid minimums is difficult. The French diving instructor with a $VO_{2\ max}$ of 16 $mL.kg^{-1}.min^{-1}$ (4.6 MET)[24] described previously certainly presented a fitness level that most would deem insufficient, but deciding on a single value is not as straightforward as it might seem. The available data suggest that at least some individuals are diving, presumably with an expectation of safety, with aerobic capacities far below the 13 MET threshold promoted by some.[18]

I posit that a skilled diver can probably safely conduct an uncomplicated dive under benign to modest conditions with a capacity of 7 MET. This is not optimal, but a skilled diver might be expected to have a reasonable reserve to meet most typical demands. Recognising the possibility of exceptional challenges, the benefit of substantial reserves and the greater challenge of less experience, I recommend that divers maintain a capacity of 10 MET or greater. The recommendation should be treated as a rule for anyone in a leadership position with responsibility over others. Interpreted conservatively this would include all buddy diving situations. For training purposes, I advocate accepting students with a capacity of 7 MET for confined water work and including conditioning in their training to help them achieve a 10 MET capacity before the open-water phase; advancement to open water may be delayed. MET scores should also be computed with gender-appropriate values, using assumed resting metabolic rates of 3.5 $mL.kg^{-1}.min^{-1}$ for males and 3.2 $mL.kg^{-1}.min^{-1}$ for females.

Physical fitness should receive more attention than is the current practice in dive training and diver evaluations. The risks of inadequate fitness and the benefits of enhanced levels of fitness for diving and general health should be more thoroughly discussed. All divers should be encouraged to pursue a capacity of 14 MET or higher as a personal goal (Table 1). At the same time, setting a threshold limit that is unrealistic serves no positive purpose. The majority of the studies described in Table 2 involved professional divers, one-third of them military divers with regular physical fitness training obligations. While the data are not available, it is likely that many divers, professional and non-professional, will have lower aerobic fitness than the described divers and will continue to dive safely. The point at which low aerobic fitness actually becomes a significant independent risk factor for diving safety requires additional study, but it is likely at the bottom of or below the range of 7–10 MET.

Practical strategies for testing of aerobic capacity

A major argument against aerobic capacity testing will be financial; testing in a professionally staffed facility can be expensive. One strategy to avoid the high cost is to volunteer for research studies that include $VO_{2\ max}$ tests as part of the protocol. These are typically available through the exercise or applied physiology programmes of many universities and colleges. Participants can have their performance evaluated while contributing to the greater good.

Practically, maximal testing may be required only for those with a poor history of regular physical exercise or those expected to be at risk of inadequate capacity. The pencil-and-paper tests mentioned earlier may not provide reliable estimates of aerobic fitness on an individual basis, but they can be a useful first step in discussing fitness questions. Some responses can be instantly reassuring. For example, any individual regularly completing a five kilometre run without breaking pace in less than 30 minutes is likely to have sufficient aerobic capacity. Conversely, responses that indicate less than regular involvement in physical activity with an aerobic component should prompt closer scrutiny. Unfortunately, many activities are more difficult to assess than running. Interpreting times and distances for cycling and swimming, for example, requires more caution. Fast swim times may say more about skill than aerobic fitness (and sometimes of fond memory). Outdoor cycling performance is affected by the bicycle, additional load and surface conditions, but participation weighs heavily in favour of the candidate. Reports of stationary cycling activity are the least informative. The resistance settings are selected by the rider, and frequently on non-standard scales. Cycling for even long periods of time against very low resistance will not maintain fitness. Giving too high a fitness credit for indoor cycling is a common problem. Reports of use of a wide range of other modern gym equipment can be similarly misleading.

The use of predictive tests to grade relative performance was discussed earlier. This approach can be useful for applied diver testing. Bias due to personal skill is likely to be less apparent in fin swimming than standard swimming strokes. A simple and relevant challenge is to have divers complete a surface fin swim with mask and snorkel alone or with mask and snorkel while wearing unused scuba gear. The time to complete the swim, heart rate at the end and heart rate one minute after ending exercise would all be recorded. Reduced swim times could indicate improvements in skill, economy or cardiorespiratory fitness. Increased difference in heart rate immediately after exercise and one minute post-exercise, i.e., better recovery of the heart rate, would primarily indicate improved cardiorespiratory fitness. Instituting such challenges to every training session may help divers establish the habit of regularly reviewing their own fitness.

Effectiveness of aerobic training

Aerobic capacity is sensitive to training. $VO_{2\,max}$ can generally be increased by 25 per cent or more in untrained individuals with modest effort. Additional gains can occur as body composition changes and the ratio of muscle-to-fat mass is improved. There are two primary stages of adaptation. The first comes after two to three weeks of training, primarily in the form of an increase in body fluid volume. The second begins after six weeks of training as the metabolic potential within the muscle cells is increased.

The primary adaptation to regular aerobic exercise is an increased work capacity. This increases the reserve potential to meet emergent needs and reduces the strain on the cardiorespiratory system from any submaximal work rate.

A similar pattern of detraining will be experienced if a training programme is suspended. The fluid volume increase will be lost within the first two weeks and a decline in the metabolic readiness will follow. Fortunately, the same degree of effort required to improve aerobic fitness is not required to maintain it. Maintenance training that is of lesser duration but similar intensity can protect aerobic capacity.

Training initiatives

The most basic goal should be to participate in some form of physical activity most days of the week, achieving training intensity efforts three times per week for 30–60 minutes per session. The simplest means of monitoring exercise intensity is based on the percentage of maximal heart rate (HR_{max}). Age-estimated maximum heart rate (assumed to be 220 minus age in years for land-based activities) is computed, and a target range of exercise intensity established (usually between 70 and 90 per cent of HR_{max}). This range corresponds to approximately 55–75 per cent of $VO_{2\,max}$. Immersed exercise heart rates will be lower than during land-based exercise, but similar training targets can be established by tolerance.

Individuals are encouraged to maintain their exercise intensity at the low end of the target range when they are starting out. The top end of the range represents the maximal safe intensity for sustained exercise. As fitness increases, it will take a greater intensity to generate the same relative effort (i.e., to produce the same heart rate or percentage of maximal ability). Employing a variety of aerobic activities is generally desirable to reduce the risk of overuse injury and boredom. A mix of activities that also incorporates strength, flexibility and agility will also produce benefits useful to meet the demands of diving.

The greatest absolute improvement in $VO_{2\,max}$ would likely be seen with running or cycling. Water-based activities, however, in addition to generally being less mechanically stressful, allow simultaneous improvement of physical fitness, watermanship and in-water psychological comfort. Fin swimming should be an important part of any programme given the diver's reliance on finning. Length fin swimming might be most appropriate at the entry level. Advancing to underwater hockey or underwater rugby introduces a dynamic component that can have a powerful training effect on fin power, technique and, as a separate but very relevant benefit, breath-hold ability. Regular swimming offers similar benefits in watermanship and develops upper-body strength, important for entry/exit requirements and many emergent situations. Advancing to water polo or canoe polo can again add more of a dynamic and social component to the fitness effort.

Conclusions

Physical fitness is necessary to ensure that the normal and emergent needs of diving can be met. Reserves of both strength and aerobic capacity are important. A minimum capacity in the range of 7–10 metabolic equivalents (MET) may be more appropriate than the capacity of 13 MET sometimes advocated. In recognition of the importance of physical fitness and the decline associated with normal ageing, training programmes should promote awareness of the problems associated with low fitness and the benefits of enhanced fitness. Possession of a healthy fitness reserve can make a huge difference in how well normal and emergent events will be managed. Such preparedness is clearly of value to divers operating in a medium (water) that at times and unexpectedly can be very unforgiving.

References

1 Physical activity and health: a report of the Surgeon General. National Center for Chronic Disease Prevention and Health Promotion. US Department of Health and Human Services; 1999. <http://www.cdc.gov/nccdphp/sgr/sgr.htm>.

2 Health Canada, 1997. <http://www.phac-aspc.gc.ca/pau-uap/paguide/back3e.html> (last accessed 24 September 2007).

3 National Center for Health Statistics, 2006 with chartbook on trends in the health of Americans. Hyattsville, MD: US Centers for Disease Control and Prevention; 2006. p. 54-7.

4 Ma AC, Pollock NW. Physical fitness of scientific divers: standards and shortcomings. In: Godfrey JM, Pollock NW, editors. *Diving for science 2007*. Proceedings of the 25th symposium. Dauphin Isl, AL: American Academy of Underwater Sciences; 2007. In press.

5 Glen S, White S, Douglas J. Medical supervision of sport diving in Scotland: reassessing the need for routine medical examinations. *Br J Sports Med*. 2000; 34: 375-8.

6 Taylor DD, O'Toole KS, Ryan CM. Experienced, recreational scuba divers in Australia continue to dive despite medical contraindications. *Wilderness Environ Med*. 2002; 13: 187-93.

7 Wisloff U, Brubakk AO. Aerobic endurance training

reduces bubble formation and increases survival in rat exposed to hyperbaric pressure. *J Physiol*. 2001; 537(Pt 2): 607-11.
8 Carturan D, Boussuges A, Burnet H, Fondarai J, Gardette B. Circulating venous bubbles in recreational diving: relationships with age, weight, maximal oxygen uptake and body fat percentage. *Int J Sports Med*. 1999; 20: 410-4.
9 Broome JR, McNamee GA, Dutka AJ. Physical conditioning reduces the incidence of neurological DCI in pigs. *Undersea Hyperb Med*. 1994; 21(S): 69.
10 Dujic Z, Duplancic D, Marinovic-Terzic I, Bakovic D, Ivancev V, et al. Aerobic exercise before diving reduces venous gas bubble formation in humans. *J Physiol*. 2004; 555: 637-42.
11 Astrand PO, Rodahl K. *Textbook of work physiology*, 2nd ed. New York, NY: McGraw-Hill; 1977.
12 Jackson AS, Blair SN, Mahar MT, Wier LT, Ross RM, Stuteville JE. Prediction of functional aerobic capacity without exercise testing. *Med Sci Sports Exerc*. 1990; 22: 863-70.
13 Kline GM, Porcari JP, Hintermeister R, Freedson PS, Ward A, et al. Estimation of $VO_{2\ max}$ from a one mile track walk, gender, age, and body weight. *Med Sci Sports Exerc*. 1987; 19: 253-9.
14 McArdle WD, Katch FI, Pechar GS, Jacobson L, Ruck S. Reliability and inter-relationships between maximal oxygen intake, physical work capacity and step-test scores in college women. *Med Sci Sports*. 1972; 4: 182-6.
15 Francis K, Brasher J. A height-adjusted step test for predicting maximal oxygen consumption in males. *J Sports Med Phys Fitness*. 1992; 32: 282-7.
16 Weller IM, Thomas SG, Corey PN, Cox MH. Selection of a maximal test protocol to validate the Canadian Aerobic Fitness Test. *Can J Sport Sci*. 1992; 17: 114-9.
17 Bennett PB, Cronje FJ, Campbell E, Marroni A, Pollock NW. *Assessment of diving medical fitness for scuba divers and instructors.* Flagstaff, AZ: Best Publishing; 2006.
18 Bove AA. Medical aspects of sport diving. *Med Sci Sports Exerc*. 1996; 28: 591-5.
19 Carturan D, Boussuges A, Vanuxem P, Bar-Hen A, Burnet H, Gardette B. Ascent rate, age, maximal oxygen uptake, adiposity, and circulating venous bubbles after diving. *J Appl Physiol*. 2002; 93: 1349-56.
20 Almeling M, Schega L, Witten F, Lirk P, Wulf K. Validity of cycle test in air compared to underwater cycling. *Undersea Hyperb Med*. 2006; 33: 45-53.
21 Thompson J, Barr D, McDonald DR, Rennie MJ. North Sea divers are no fitter than sedentary men. *Lancet*. 1984; II (8394): 107-8.
22 Thompson J, Barr D, Rennie MJ. Fitness of North Sea divers. *Lancet*. 1984; II (8406): 806.
23 Thorsen E, Hjelle J, Segadal K, Gulsvik A. Exercise tolerance and pulmonary gas exchange after deep saturation dives. *J Appl Physiol*. 1990; 68: 1809-14.
24 Tripodi D, Dupas B, Potiron M, Louvet S, Geraut C. Brain magnetic resonance imaging, aerobic power, and metabolic parameters among 30 asymptomatic scuba divers. *Int J Sports Med*. 2004; 25: 575-81.
25 Dujic Z, Palada I, Obad A, Duplancic D, Bakovic D, Valic Z. Exercise during a 3-min decompression stop reduces postdive venous gas bubbles. *Med Sci Sports Exerc*. 2005; 37: 1319-23.
26 Dujic Z, Palada I, Obad A, Duplancic D, Brubakk AO, Valic Z. Exercise-induced intrapulmonary shunting of venous gas emboli does not occur after open-sea diving. *J Appl Physiol*. 2005; 99: 944-9.
27 Dujic Z, Obad A, Palada I, Ivancev I, Valic Z. Venous bubble count declines during strenuous exercise after an open sea dive to 30 m. *Aviat Space Environ Med*. 2006; 77: 592-6.
28 Dujic Z, Palada I, Valic Z, Duplancic D, Obad A, et al. Exogenous nitric oxide and bubble formation in divers. *Med Sci Sports Exerc*. 2006; 38: 1432-5.
29 Thorsen E, Segadal K, Stuhr LEB, Troland K, Gronning M, et al. No changes in lung function after a saturation dive to 2.5 MPa with intermittent reduction in PO_2 during decompression. *Eur J Appl Physiol*. 2006; 98: 270-5.
30 Blatteau JE, Boussuges A, Gempp E, Pontier JM, Castagna O, et al. Haemodynamic changes induced by submaximal exercise before a dive and its consequences on bubble formation. *Br J Sports Med*. 2007; 41: 375-9.
31 Boussuges A, Riera F, Rossi P, Blatteau JE, Castagna O, Galland F. Echocardiography in military oxygen divers. *Aviat Space Environ Med*. 2007; 78: 500-4.
32 Rosen MJ, Sorkin JD, Goldberg AP, Hagberg JM, Katzel LI. Predictors of age-associated decline in maximal aerobic capacity: a comparison of four statistical models. *J Appl Physiol*. 1998; 84: 2163-70.
33 Marti B, Howald H. Long-term effects of physical training on aerobic capacity: controlled study of former elite athletes. *J Appl Physiol*. 1990; 69: 1451-9.
34 Cotes JE, Reed JW. North Sea divers are no fitter than sedentary men. *Lancet*. 1984; II (8398): 348-9.
35 Crosbie WA. Fitness of North Sea Divers. *Lancet*. 1984; II (8400): 471-2.

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Deep decompression stops

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Key words
Decompression, deep diving, technical diving, models, trimix, review article

Abstract

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Technical divers have adopted widely the practice of either adding deep decompression stops to their decompression profiles or using decompression models that incorporate deep stops in the belief that these techniques will reduce the incidence of decompression sickness. However, new evidence suggests that the gas kinetic model on which this practice is based is flawed. This paper reviews the historical precedence, controlled studies and theoretical background for and against deep stops.

Introduction

The last 25 years have seen a rapid development of recreational diving activities. Whereas previously recreational diving had been confined to 'no stop' limits, the development of new techniques and equipment has seen recreational 'technical' divers adopt decompression and mixed-gas strategies to access depths not often previously explored, even by bounce commercial or Navy divers. These depths are in many cases outside the well-established areas of decompression theory and practice. However, access to 'new' decompression models via the internet and computers has encouraged technical divers that such dives are not only possible but also can be conducted safely. Many of these new decompression models include so-called 'deep' stops. Despite the lack of evidence to support their efficacy, deep stops have been incorporated into many of the newer diving computer algorithms, notably those from Uwatec™, Suunto™ and Delta P™ (VR3 technical dive computer), and have been enthusiastically embraced by the recreational diving community. This paper will review the theoretical and practical evidence for deep decompression stops.

The introduction of deep decompression stops has been popularly ascribed to the American ichthyologist Richard Pyle.[1] Pyle's work often required him to collect specimens from considerable depth (> 300 feet sea water (fsw)). After successful forays, Pyle was forced to perform deep stops during his ascent to decompress the swim bladders of the specimens he had collected. Pyle noticed that he felt better after dives where he had made these additional deep stops than after dives where he had not collected any specimens and had made stops according to the normally prescribed decompression profile. Several internet articles, such as those by Baker,[2,3] have promoted the insertion of decompression stops deeper than those predicted by the Buhlmann or Workman tables. However, while some methods of deriving these stops may have some theoretical logic to back them up, others have merely resulted from some empirical rule.[4,5]

For the purposes of this paper, a deep stop will be defined as a decompression stop that is performed deeper than the initial decompression stop that would be predicted by the Buhlmann ZHL-16C decompression model.

Deep-stop profiles conducted by technical divers are usually produced by one of three methods:

1 Pyle stops: the first new stop is conducted for two to three minutes halfway between the maximum bottom depth and the first prescribed stop. The schedule is then recalculated and if the distance to the next stop is greater than 10 metres' sea water (msw) another stop is generated halfway between the first new stop and the next prescribed stop. The schedule is then recalculated and the process repeated. From a decompression modelling point of view, the first of the deep stops produced by this method is usually too deep, in that the calculated inert gas tensions are not sufficiently greater than ambient pressure to optimise decompression.

2 Proportional M-Value Reduction Method (PMVRM, popularly known as the Gradient Factor Method): the diver decides what proportion of the Buhlmann supersaturation gradients are to be allowed both at depth and in the shallows.[3] For example, a diver might select a maximum allowed gradient of 20% of the Buhlmann value at the maximum depth and perhaps 80% for surfacing (compared with the 90% of each value described by Buhlmann). A computer programme then proportionally alters the maximum allowed gradients between these two values depending on the depth. This results in deeper initial stops and generally longer decompression schedules than those of the native Buhlmann models. The depth of the initial stop will be controlled by the deep gradient factor value selected by the diver, i.e., the lower the value the deeper the initial stop. While there are no formal guidelines on how to select the gradients, the general method seems to be that the greater the depth/time profile, the lower the deep gradient value that is selected.

3 'Dual phase' or 'bubble' models: the most popular of these are the Variable Permeability Model (VPM) and Reduced Gradient Bubble Model (RGBM) and are

readily available as PC-based software. The programmes produce similar decompression schedules incorporating decompression stops deeper than those predicted by the Buhlmann model. The majority of these stops are of short duration (one minute or less). They generally produce longer decompression times within the recreational diving depth range but substantially shorter profiles for deep decompression type dives.

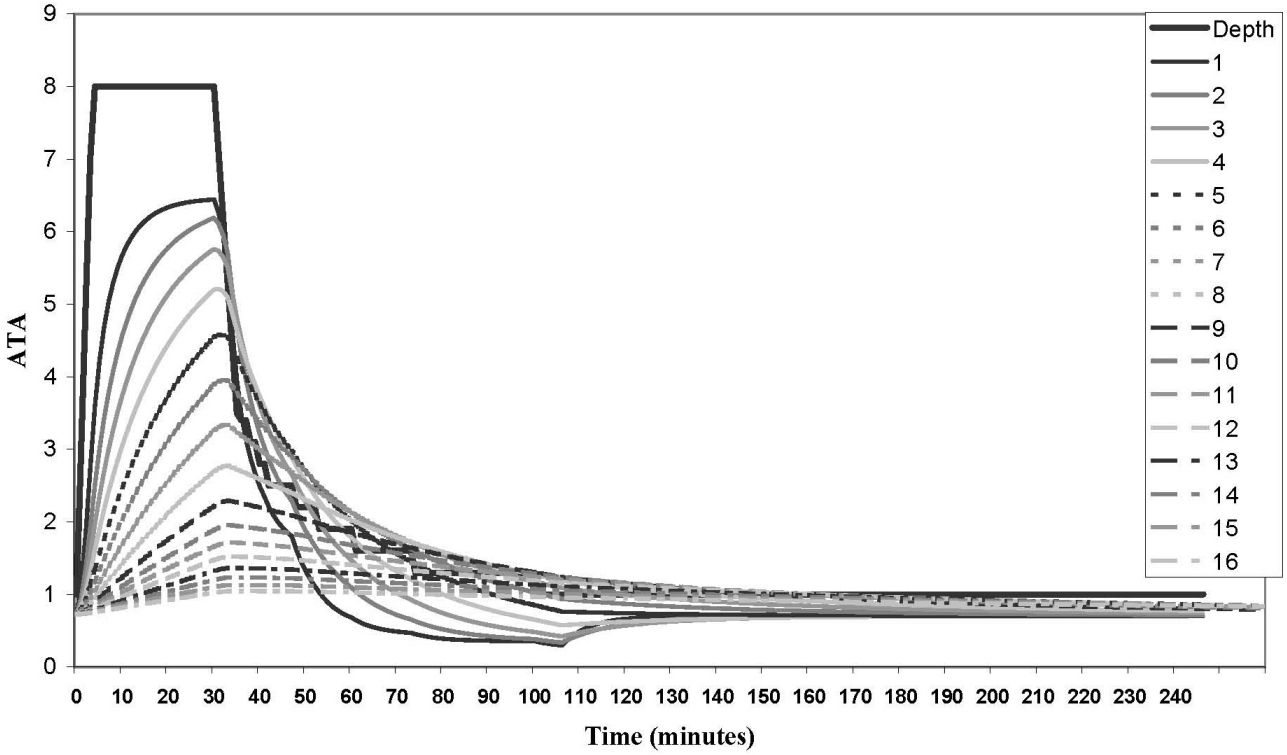
Decompression practices: historical perspective

Modern decompression practice is largely based on the work of JS Haldane.[6] Haldane's strategy for successful decompression was based on the concept that tissues could tolerate a finite level of supersaturation during decompression. If the ratio of ambient pressure to tissue pressure was kept below this level at all times during the decompression, no decompression sickness (DCS) should result. Haldane produced a gas kinetic model with five compartments and a single 'gradient' line, which limited the allowed over-pressure in all the compartments. This approach was combined with a stepped or staged decompression and resulted in the diver initially being brought as close to the surface as possible without exceeding the prescribed limit so as to minimise on-gassing at depth and to maximise the gradient for off-gassing inert gas from the tissues. While the development of Haldane's method was a vast improvement over previous practice, it was soon found that the schedules produced in this way were too conservative for shallow dives and too aggressive for deep dives. The Royal Navy dealt with the problematic profiles by (usually) adding time to the final stop. Over the subsequent years this resulted in a series of modified tables, which became progressively more empirical in derivation.

Buhlmann and Workman further developed the Haldanean model during the 1960s.[4,7] Buhlmann and Workman both conducted extensive manned experiments to find the maximum tolerated supersaturations for the various assumed tissue compartments (Figure 1). In contrast to Haldane's original model, each compartment was given its own unique gradient line with the slow compartments having very low allowed supersaturation limits and the faster compartments progressively higher limits (Figure 2).

The gradients in the various compartments in both Buhlmann and Workman's models resulted from the manned experiments and the need to fit their models to the known no decompression limits (NDLs). Both Workman and Buhlmann independently ended up with similar maximum allowed gradients for compartments of similar half-times with air dives. However, Workman found that for the deeper dives where heliox was being used as the breathing gas, a reduced gradient was required, producing deeper initial stops than for an air dive of equivalent depth.[5] This finding had some historical precedence, being consistent with the early studies on helium diving dating back to the 1930s.[8] In the Buhlmann tables, as the helium content of the breathing

Figure 1
Typical Buhlmann type tissue inert gas tensions versus time; 16 compartments with half-times from 2 min to 635 min; 30 min bottom time, dive to 70 msw utilising trimix 18:35 and nitrox decompression



gas increases, the allowed gradients are actually increased, though this is offset by the faster exchange of helium in this model and results in similar overall profiles.

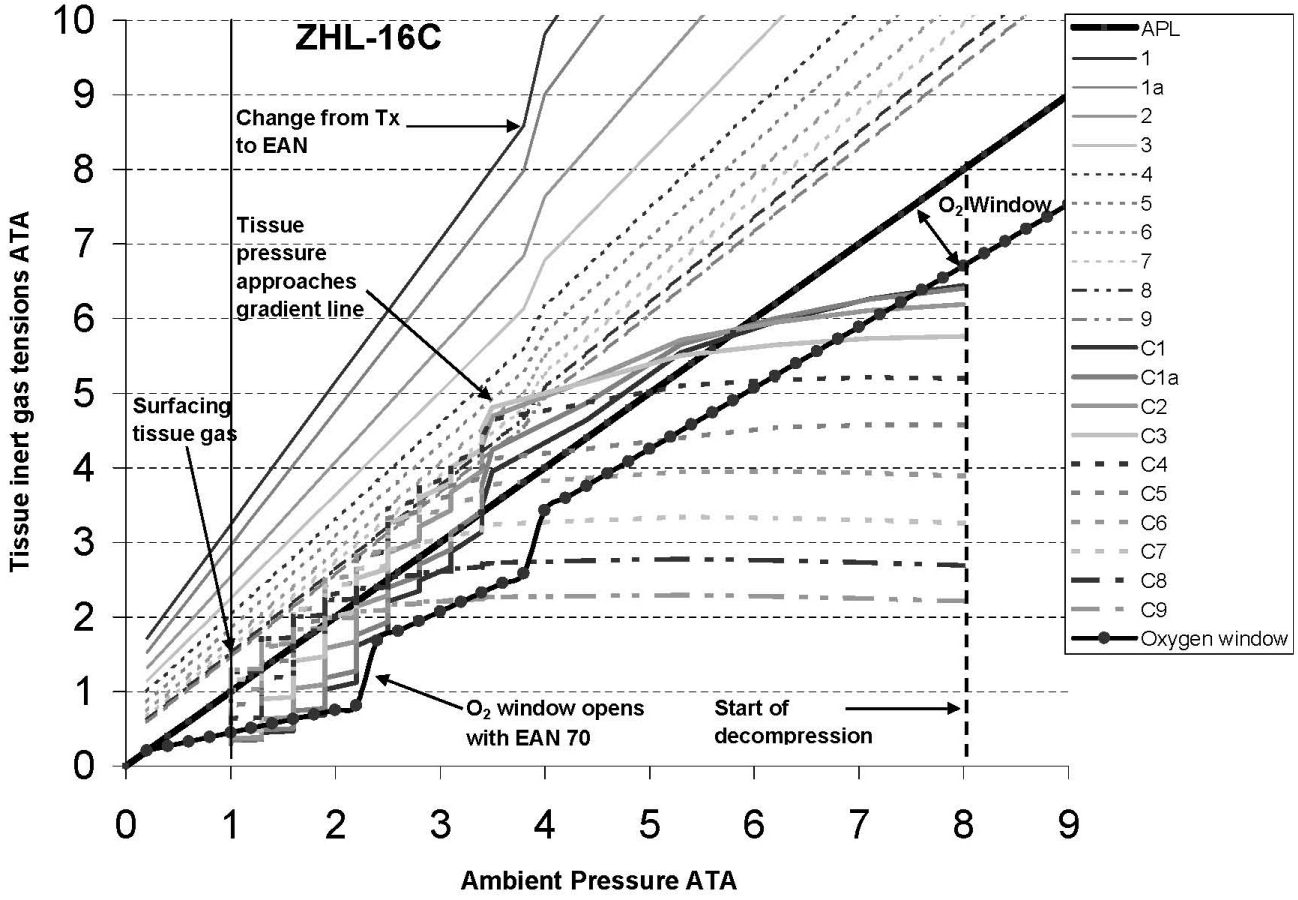
The result of this approach from both Buhlmann and Workman is that profiles produced using their methodology have a characteristic long initial ascent towards the surface and long periods in the shallow to complete the decompression. In general, in the air range, the Buhlmann method produces deeper initial stops than the USN (Workman). However, this difference is relatively small compared with the more recently invoked 'deep stop' type profiles.

Buhlmann's ZHL-12 and later ZHL-16 models have proven very successful and have been widely applied in electronic diving computers, becoming the de-facto standard against which other models are compared in recreational diving. Their rate of DCS, when used in the recreational range, is quoted as somewhere between 1:1,000 and 1:10,000. One feature of the Buhlmann model that was different to earlier models was the adoption of a 10 $m.min^{-1}$ ascent rate. Marroni et al have demonstrated that this ascent rate has a lower decompression stress as measured by venous bubble scores than either faster or slower ascent rates.[9]

Historical precedent

Deep decompression stops have been practised empirically for probably close to 80 years. In the 1960s, Le Messurier and Hills studied the decompression practices of the pearl divers off the Torres Straits in Northern Australia.[10] These divers had developed their diving practice without any knowledge of decompression theory over some 80 years and at the cost of some 2,000 lives. The profiles they had developed had a similar rate of DCS to the USN tables then in use, about 5–7%, but featured deeper initial stops, a final stop at 6 msw (versus 3 msw for the USN) and generally an overall reduction in decompression time to about two-thirds of that for similar USN profiles. Interestingly, other groups of native divers in other parts of the world (e.g., Hawaii) had also empirically ended up with similar deep-stop dive profiles. However, the likelihood of a considerable 'healthy

Figure 2
70 msw dive as per Figure 1, but looking at inert gas tensions in each compartment versus ambient pressure; as each tissue nears its gradient line, a decompression stop is forced. Typically high supersaturation levels are allowed early in the dive, progressively reducing as the longer half-time tissues control the dive. Note the allowed higher gradients for the short half-time tissues and the increased gradients when helium is present in the breathing mix.



worker' effect associated with these native divers (given the harsh selection process) may significantly limit the applicability of these types of profiles to the wider diving community.

Hills' investigations into the practices of the Torres Strait pearl divers led him to believe that the USN type profiles were causing bubble formation to occur during the long initial ascent to the surface, the time in the shallow being prolonged to allow for bubble reabsorption before surfacing. This was in contrast to the popular view that no DCS meant no bubbles. Hills tested his hypothesis with a series of experiments on goats and demonstrated that by performing the final stop at 6 msw rather than 3 msw, the total decompression time could be reduced for the same incidence of DCS.[11]

Hills theorised that this reduction in decompression time was possible because the bubbles that had formed would have a smaller radius at the deeper final stop, hence their internal pressure would be higher, favouring reabsorption and gas elimination. He described traditional models as "bend then mend" and theorised that by avoiding the formation of free gas, by performing deeper initial stops, decompression could be both faster and safer. Hills went on to develop the Thermodynamic Decompression Model, a diffusion-based tissue model in which gas was prevented from coming out of solution by utilising a decompression profile conducted within the oxygen window or inherent unsaturation of tissues.[12–14] A feature of the Hills profiles, compared with the Buhlmann or USN profiles, is that the initial stops are much deeper. The mathematical complexity of his model limited its further development and implementation.

Vann et al were able to demonstrate in goats that profiles from thermodynamic models did not produce detectable venous bubbles until the final ascent from the last stop to the surface.[15] Similar depth/time profiles decompressed using the USN table produced detectable bubbles from 40 fsw. Hills' profiles also had some success in the commercial oil industry.[16] Hills found that the addition of a few minutes' 'deep' often prevented DCS symptoms without the need to add time in the shallow (personal communication, BA Hills, 2005). Krasberg claimed considerable success using Hills type profiles for deep diving in the North Sea; however, precise data on these profiles and the implementation of Hills' model have remained elusive.[16]

1970s bounce diving experience

During the 1970s, prior to the development of safe saturation techniques, there was much interest in deep bounce diving. Despite deep stops being advocated by some researchers, others were equally (or more) successful with more traditional types of profiles. Whereas Cabarrou et al were able to produce workable profiles with traditional Haldanean type supersaturation limits, Bennett et al had only limited success for similar depth/time profiles despite drastic reductions in the deep supersaturation limits by adding deep stops.[17] Their initial approaches, which produced very similar profiles to the modern 'bubble' models, were unsuccessful, with a high incidence of vestibular and Type I DCS, and it was not until they adopted a mixed perfusion/diffusion-based gas kinetic model that they were able to produce successful profiles. However, these profiles were substantially longer than those of Cabarrou for the same depth/time exposure without deep stops.[18]

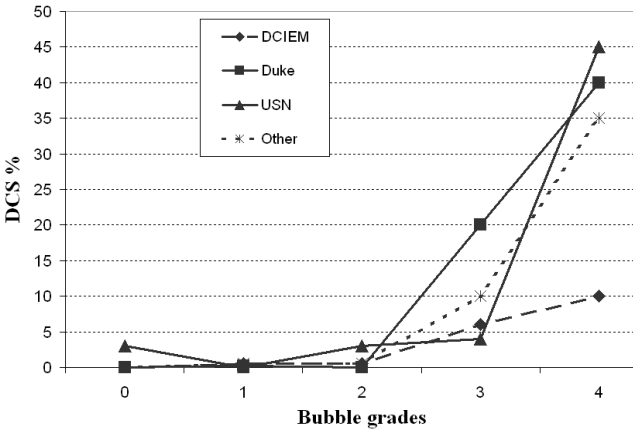
Bubble models

The development of ultrasonic bubble detection devices and their application to decompression practice demonstrated that despite the low observed incidence of DCS in traditional profiles, venous bubbles were present after most dives.[19–21] One explanation for the presence of these bubbles in the absence of symptoms is the 'critical volume hypothesis'.[11,22] This hypothesis states that the body can tolerate a certain amount of gas coming out of solution into one or more critical tissues. As long as the volume of evolved gas during a dive is kept below this amount, no symptoms of DCS should result. For the Buhlmann type gas model, the fast compartments control the ascent profile for shallow, short depth/time profiles. Since little gas has been taken up in the critical tissue(s) during such dives, relatively large apparent supersaturations can be justified for the same critical volume of gas to come out of solution.

Based on the observed behaviour of bubbles in gelatine,[23] Kunkle and Yount applied the critical volume hypothesis in an attempt to marry the empirically derived Buhlmann type supersaturation limits with some basic theories on bubble mechanics.[24,25] The resultant VPM model predicted deeper initial decompression stops than the Buhlmann model with similar decompression times within the recreational range.[25] The model amortises the calculated evolution of gas over the dive such that calculated evolved volume of gas is restricted to less than a critical volume as defined by the model. This produces supersaturation restraints that are then overlaid to the Buhlmann gas model. The effect of this is to substantially lower allowed compartment supersaturations at depth (and hence deeper stops) but paradoxically may produce shorter overall decompression times and higher predicted compartment surfacing gas tensions in the mid to slow compartments. In dives of greater than 70 msw, these surfacing compartment tensions commonly exceed the limits derived experimentally by Hempleman and Buhlmann, which were shown to produce clinical symptoms.[26]

Wienke has developed a model (RGBM) similar to the VPM model.[27] This has been enthusiastically adopted by both the dive computer manufacturers and the technical diving community despite limited field validation. Like the VPM, the RGBM predicts deeper initial stops than the Buhlmann or Workman type profiles.

Figure 3
Ultrasound-derived venous bubble scores versus DCS incidence from various institutions[33]



Limitations of gas kinetic models

The VPM, RGBM and Buhlmann models all share the same basic compartment model. A major feature of this base model that has made it popular with technical divers is its ability to deal with gas mixtures other than air. The model assumes that helium is on-and off-gassed 2.65 times faster than nitrogen.[4,28] The partial pressures for each inert gas in each compartment are calculated, and then added together and compared with the prescribed limit (which in the Buhlmann model is itself varied depending upon the fraction of each gas). Buhlmann derived the 1:2.65 ratio from a fairly weak data set of human experiments.

However, recent experiments actually measuring helium and nitrogen elimination at 1 ATA (101.3 kPa) would tend to indicate that the true ratio is probably closer to 1:1.2.[29,30] In these studies, helium kinetics were best described by a model incorporating perfusion, diffusion and an element of counter-diffusion between arterioles and venules. The proportional contribution of each component was dependent upon the blood flow and the inert gas involved. Doolette's experiments were carried out at 1 ATA. Extrapolation of this work to higher pressures involving tissue supersaturation and decompression must be performed with caution as the rate of inert-gas washout has been described to vary with decompression.[31]

To confuse matters further, the ability of venous bubbles to enhance inert-gas washout during decompression may also substantially alter the kinetics of gas exchange.[32] This may result in an acceleration of the elimination of helium when bubbles are present, as retention of gas in venous bubbles may significantly affect arterio-venous gas exchange. Thus, while there is evidence that helium kinetics may be slower than predicted in traditional models, a mechanism may exist whereby elimination may be accelerated in the presence of venous microbubbles.

Therefore, while it is unlikely that the Buhlmann model of gas exchange is physiologically accurate, it is quite plausible that it might still provide workable decompression solutions within certain ranges when used in conjunction with its original decompression rules. However, if the decompression ascent rules are altered, the relationship may not necessarily hold.

This may be particularly pertinent for technical recreational divers who often utilise inert-gas switching to accelerate decompression in association with the insertion of deep decompression stops. Such practices may actually reduce gas exchange as outlined previously, while the decompression model predicts a reduced decompression obligation. The result may well be an inadequate decompression solution. Recently, at least one of the technical diving training agencies has recommended the maintenance of a constant fraction of helium in decompression mixtures to minimise the risks of inner ear decompression sickness.

While the presence of venous gas emboli (VGE) in USN and Buhlmann profiles was taken as a marker of inadequate decompression (on the basis that no DCS should equal no bubbles), it should be noted that low-grade venous bubbles scores do not correlate with incidence of DCS (Figure 3). Even Spencer Grade IV bubbles correlate only approximately to a 45% risk of clinical DCS.[33] Thus, while the presence of venous bubbles might be a marker of inadequate decompression they may paradoxically also enhance tissue nitrogen elimination and decompression. If this were so, the presence of *low-grade* VGE may represent optimised decompression rather than indicate a *significant* level of decompression stress. This would bring into question the use of low-grade VGE scores as justification for the adoption of deep decompression stops and reduced decompression gradients, as happened in the 1980s.

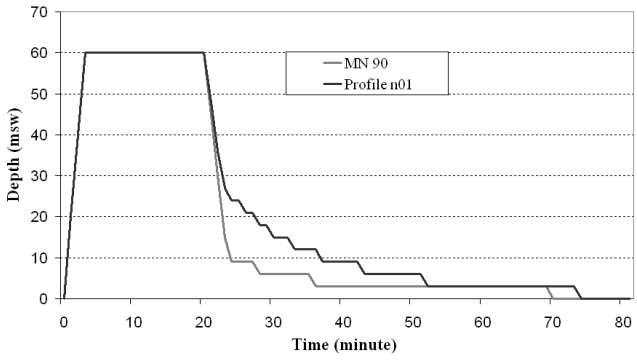
Studies on deep decompression stops

To date, two papers have been published and two abstracts reported that have specifically addressed the issue of the value of deep initial decompression stops.

Marroni et al conducted a series of sea dives to 25 msw.[34] The durations of these dives were within the no decompression limits and the following profiles were followed:

- 3 m.min^{-1} ascent rate without stops
- 3 m.min^{-1} ascent rate with stops
  - stop for 5 min at 6 msw
  - stop for 5 min at 15 msw
  - stops for 5 min at both 6 msw and 15 msw
- 10 m.min^{-1} ascent rate without stops
- 10 m.min^{-1} ascent rate with stops
  - stop for 5 min at 6 msw
  - stop for 5 min at 15 msw
  - stops for 5 min at both 6 msw and 15 msw
- 18 m.min^{-1} ascent rate without stops

Figure 4
French Navy experimental deep-stop profile n^{0}1 versus the standard MN 90 profile. In the repetitive series, the n^{0}1 profile produced higher bubble grades and symptomatic DCS[35]



- 18 m.min^{-1} ascent rate with stops
  - stop for 5 min at 6 msw
  - stop for 5 min at 15 msw
  - stops for 5 min at both 6 msw and 15 msw

Divers were monitored for venous bubbles post dive using a Doppler ultrasound device. Highest bubble scores were seen in dives that had a 3 m.min^{-1} ascent rate with a 5 min stop at 6 msw even though the total decompression time was similar to that for the lowest scoring group (10 m.min^{-1} ascent rate with stops at 6 msw and 15 msw). The addition of the deep stop at 15 msw seemed to substantially reduce the bubble scores in all profiles irrespective of ascent rate. This reduction in bubble scores was far larger than that seen with the addition of the 6 msw stop. However, statistical analysis of the results was not provided, thus their significance is unknown.

It is interesting to note that the longest decompression time (3 m.min^{-1} ascent with stops at 6 msw and 15 msw) did not produce the lowest bubble scores. While the addition of the deep stop to a given ascent rate did seem to reduce the venous bubble scores, this generally occurred in the setting of an increased total decompression time. Unfortunately this paper failed to answer the more important question of whether the addition of a deep stop for a given total decompression time alters decompression stress.

Blatteau et al looked at the issue of deep stops in a way more relevant to technical decompression diving.[35] They compared the standard French Navy profile MN 90, for a dive to 60 msw for 20 min followed by a 50 msw dive for 13 min (3 hour surface interval), with a profile where they reduced the ascent rate to 12 m.min^{-1} (as opposed to 15 m.min^{-1} for the standard profile) and added deep stops beginning at half the maximum depth. The subsequent ascent rate was reduced to 3 m.min^{-1} from 6 m.min^{-1} for the standard table. This profile was designated n^{0}1 (Figure 4).

A second profile was also tested where they used their standard ascent rates and added a single deep stop at half the maximum depth for two minutes, n^{0}2. This latter profile was tested only for a dive of 60 msw/15 min bottom time. Evaluation was conducted using Doppler ultrasonic monitoring and venous bubble grading. Peak bubble scores were seen 60 minutes after surfacing in all dives. Similar bubble scores were seen in both the deep-stop profiles and the standard profiles; however, in the multiple dive series, the deep-stop profile n^{0}1 produced higher bubble grades (which persisted for more than three hours) and symptomatic DCS. The n^{0}2 profile did not produce significantly different bubble scores to the native Haldanean type Navy schedule (MN90). This paper would appear to confirm the observation from Marroni's paper that prolonged decompression times *per se* do not necessarily reduce decompression stress.

Both of the papers discussed utilised Doppler venous bubble scoring as a marker of decompression stress.

ABSTRACTS

Two abstracts were reported at the 2007 Undersea and Hyperbaric Medicine Society meeting in Hawaii.[36,37]

In the first study, air dives to 51 msw for 30 min bottom time (including descent time) were conducted.[36] Decompression was then carried out according to either a deterministic gas content model or a probabilistic bubble model (BVM). In the former the first stop was at 40 fsw and the latter 70 fsw. In both cases the total dive time was 174 min. The trial was terminated at the mid-point interim analysis with 11 cases of DCS (including two CNS cases) in the deep-stop group and only three in the shallow group (198 and 192 dives in each group respectively). It should be pointed out that the profiles produced with the BVM model in this study correlate poorly with the profiles generated with the 'bubble' models used by the technical diving community.

The second study reported the results of a series of experiments on pigs.[37] In this study, the pigs received either a long shallow profile (30 msw/70 min) or a short deep profile (65 msw/20 min). Decompression was then carried out using either a Buhlmann model or a new 'bubble' model incorporating deep stops. The animals were monitored using ultrasound to detect the development of venous gas emboli (VGE). For the long shallow profile the addition of the deep stops reduced VGE scores; however, in the deep profile, the addition of the deep stops produced a dramatic increase in bubble formation and the experiment was aborted. A revised profile with the deep stops removed produced a significant reduction in vascular bubble formation.

Summary

The availability on the internet of decompression software for mixed-gas diving has seen an explosion of technical diving over the last 10 years. It is now common for recreational divers to conduct dives to over 50 msw, and dives to in excess of 100 msw are regularly reported in the popular

dive literature. Many of the divers conducting these dives have little formal training or education in decompression theory beyond the basics taught in their technical diving courses and yet may be extremely opinionated and vocal in internet forums on the subject, based solely on their diving experience. It is rather alarming to see the almost zealous way in which deep stops have been incorporated into the recreational market given the paucity of good evidence as to either the benefit of such stops or a validated method of incorporating them into diving practice.

While there is some theoretical reasoning behind the adoption of deep decompression stops and some empirical and historical evidence that they may be of value, the available studies do not support their introduction. Problems encountered with deep mixed-gas dives may be as much related to the inadequacies of the base compartment model to accurately describe inert-gas kinetics as to the presence or otherwise of deep stops. Finally, the lack of correlation between DCS and low bubble scores and the increased nitrogen elimination described with venous microbubbles makes the interpretation of Doppler ultrasound bubble scores (used to justify the adoption of deep stops) difficult.[20]

It would seem that, from the available evidence, decompression profiles where more time is spent deep do not always reduce decompression stress as might be expected. This may be especially true of dives involving mixed gases and inert-gas switching. While accepting that stops deeper than those prescribed by the Buhlmann model may be optimal for safe decompression from significant depth, several workers in the field are now questioning the validity of deep stops as generated by 'bubble' models.[36–9] Further studies are needed to better define the value of deep stops and the best method of optimising decompression schedules, before deep stops are routinely incorporated into dive profiles.

Recommendations

- Decompression diving to depths of less than 80 msw using the Buhlmann ZHL-16C model would appear to have a relatively low incidence of DCS.
- Conducting the final decompression stop at 6 msw may allow for reduced decompression requirements.
- The use of helium as a breathing gas may necessitate deeper initial stops than those predicated in the USN air diving tables for an equivalent depth.
- The inclusion of half maximum depth (Pyle) type empirical stops is not supported at present by the available literature or decompression modelling.
- The inclusion of deep stops in association with gas switches involving large changes in concentrations of inert gas may result in inadequate decompression.
- Newer 'bubble' models incorporating deep stops have not been formally validated. Recent evidence would suggest that this approach produces initial stops that are too deep and may result in an increased rate of DCS.
- Further formal studies looking at deep stops in mixed-gas decompression diving need to be conducted.

References

1 Pyle R. The importance of deep safety stops: rethinking ascent patterns from decompression dives. *Deep Tech*. 1996; 5: 64.
2 Baker EC. Clearing up the confusion about deep stops. *Immersed*. 2000; issue 4: 23-31.
3 Baker EC. Understanding M-values. *Immersed*. 1998; issue 3: 23-7.
4 Buhlmann AA. *Decompression – Decompression sickness*. English ed. Berlin: Springer-Verlag; 1984.
5 Workman RD. American decompression theory and practice. In: Bennett PB, Elliott DH, editors. *The physiology and medicine of diving and compressed air work*. London: Tindall & Cassell; 1969. Chapter 12.
6 Boycott D, Damant G, Haldane J. The prevention of compressed-air illness. *J Hyg*. 1908; 8: 342-443.
7 Workman RD. *Calculation of diving tables for nitrogen-oxygen and helium-oxygen dives*: USN Experimental Dive Unit; 1965. Report No. RDU 6-65.
8 Momsen C. *Report on the use of helium oxygen mixtures for diving*. Washington, DC: Experimental diving unit; 1939. Report No. 2.
9 Marroni A, Bennett PB, Cali-Corleo R, Balestra C, Germonpre P, et al. What ascent profile for the prevention of decompression sickness? A field model comparing Hill and Haldane ascent modalities, with an eye to the development of a bubble safe algorithm. In: Germonpre P, Balestra C, editors. *Proceedings of the 28th Annual Scientific Meeting of the European Underwater and Baromedical Society*; 2002. Brugge, Belgium: EUBS, DAN; 2002. p. 44-8.
10 Le Messurier DH, Hills BA. Decompression sickness, a study of the diving techniques in the Torres Strait. *Hvaldradets Skrifter*. 1965; 4: 54-84.
11 Hills B. *Decompression sickness, volume 1, the biophysical basis of prevention and treatment*. New York: John Wiley & Sons; 1977.
12 Hills BA. A thermal analogue for the optimal decompression of divers: construction and use. *Phys Med Biol*. 1967; 12: 445-54.
13 Hills BA. A thermal analogue for the optimal decompression of divers: theory. *Phys Med Biol*. 1967; 12: 437-44.
14 Hills BA, LeMessurier DH. Unsaturation in living tissue relative to the pressure and composition of inhaled gas and its significance in decompression theory. *Clin Sci*. 1969; 36: 185-95.
15 Vann RD, Widell PJ, Youngblood D, Hills BA. Decompression of widely differing profiles monitored by ultrasonic bubble detector. In: *Symposium on blood bubble detection*; 1973; Seattle; 1973.
16 Krasberg A. Part VII. Inert gas transport: Discussion. In: Shilling CW, Backett MW, editors. *Underwater Physiology VI: Proceedings of the Sixth Symposium on*

Underwater Physiology; 1978. Bethesda, Maryland: FASEB; 1978. p. 404.
17 Bennett PB, Vann RD, Roby J, Youngblood D. Theory and development of subsaturation decompression procedures for depths in excess of 400 feet. In: Shilling CW, Backett MW, editors. *Underwater Physiology VI: Proceedings of the Sixth Symposium on Underwater Physiology*; 1978. Bethesda, Maryland: FASEB; 1978. p. 367-82.
18 Cabarrou P, Muller KG, Fust HD, Oser H, Krekeler H, Finkeldey U. Development and testing of heliox dives in excess of 100 meters. In: Shilling CW, Backett MW, editors. *Underwater Physiology VI: Proceedings of the Sixth Symposium on Underwater Physiology*; 1978. Bethesda Maryland: FASEB; 1978. p. 383-8.
19 Nishi R, Kisman K, Eatock B, Buckingham I, Masurel G. Assesment of decompression profiles and divers by Doppler ultrasonic monitoring. In: Bachrach A, Matzen M, editors. *Underwater Physiology VII: Proceedings of the Seventh Symposium on Underwater Physiology* 1971. Bethesda, Maryland: Undersea Medical Society; 1981. p. 717-27.
20 Nishi RY. Ultrasonic detection of bubbles with doppler flow transducers. *Ultrasonics*. 1972; 10: 173-9.
21 Brubakk AO, Eftedal O. Comparison of three different ultrasonic methods for quantification of intravascular gas bubbles. *Undersea Hyperb Med*. 2001; 28: 131-6.
22 Yount DE, Hoffman DC. Decompression theory: a dynamic critical volume hypothesis. In: Bachrach AJ, Matzen MM, editors. *Underwater physiology VIII*. Bethesda, Maryland: Undersea Medical Society; 1984. p. 131-45.
23 Kunkle TD, Yount DE. Gas nucleation in gelatin. In: Shilling CW, Backett MW, editors. *Underwater Physiology VI: Proceedings of the Sixth Symposium on Underwater Physiology*; 1978. Bethesda Maryland: FASEB; 1978. p. 459-68.
24 Yount DE. Application of bubble formation model to decompression sickness in fingerling salmon. *Undersea Biomed Res*. 1981; 8: 199-208.
25 Yount DE. Application of a bubble formation model to decompression sickness in rats and humans. *Aviat Space Environ Med*. 1979; 50: 44-50.
26 Hempleman HV. *Investigation into the decompression tables*. Report for the Royal Navy Perfusion Laboratories. London: Medical Research Council; 1952.
27 Wienke BR. Reduced gradient bubble model. *Int J Biomed Comput*. 1990; 26: 237-56.
28 Buhlmann AA, Frei P, Keller H. Saturation and desaturation with N_2 and He at 4 atm. *J Appl Physiol*. 1967; 23: 458-62.
29 Doolette DJ, Upton RN, Grant C. Perfusion-diffusion compartmental models describe cerebral helium kinetics at high and low cerebral blood flows in sheep. *J Physiol*. 2005; 563 (Pt 2): 529-39.
30 Doolette DJ, Upton RN, Zheng D. Diffusion-limited tissue equilibration and arteriovenous diffusion shunt describe skeletal muscle nitrous oxide kinetics at high and low blood flows in sheep. *Acta Physiol Scand*. 2001; 172: 167-77.
31 Hills BA. Effect of decompression per se on nitrogen elimination. *J Appl Physiol*. 1978; 45: 916-21.
32 Lundgren C, Bergoe G, Olszowka A, Tyssebotn I. Tissue nitrogen elimination in oxygen-breathing pigs is enhanced by fluorocarbon-derived intravascular microbubbles. *Undersea Hyperb Med*. 2005; 32: 215-26.
33 Rogers RE, Powell MR, Spencer MP. Doppler ultrasound monitoring of gas phase formation following decompression and repetitive dives. Santa Ana, California: Diving Science and Technology Corporation; 1988.
34 Marroni A, Bennett PB, Cronje FJ, Cali-Corleo R, Germonpre P, et al. A deep stop during decompression from 82 fsw (25 m) significantly reduces bubbles and fast tissue gas tensions. *Undersea Hyperb Med*. 2004; 31: 233-43.
35 Blatteau JE, Hugon M, Gardette B, Galland FM. Decompression profiles with deep stops: comparative doppler study with procedures of the French Navy. In: Grandjean B, Meliet J-L, editors. *Proceedings of the 30th Annual Scientific Meeting of the European Underwater Baromedical Society*; 2004 Sep; Ajaccio, Corsica. France: EUBS; 2004. p. 31-5.
36 Gerth WA, Gault KA, Doolette DJ. Empirical evaluation of the efficacy of deep stops in air decompression dives. *Undersea Hyperb Med*. 2007; 34 (suppl): 231-2.
37 Gutvik CR, Mollerlokken A, Brubakk AO. Difference in bubble formation using deep stops is dependent on length of bottom time; experimental findings and theoretical support. *Undersea Hyperb Med*. 2007; 34 (suppl): 230-1.
38 Brubakk AO, Gutvik C. Optimal decompression from 90 msw. In: Lang MA, Smith EB, editors. *Proceedings of the Advanced Scientific Diving Workshop*; 2006 February 23-24. Washington DC: Smithsonian Institution; 2006. p. 39-46.
39 Imbert JC. Commercial diving: 90 msw operational aspects. In: Lang MA, Smith NE, editors. *Proceedings of the Advanced Scientific Diving Workshop*; 2006 February 23-24. Washington DC: Smithsonian Institution; 2006. p. 103-18.

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Hyperbaric oxygenation in the patient with malignancy: friend or foe?

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Key words
Hyperbaric oxygen, malignancy, outcome, review

Abstract
(Macdonald HM. Hyperbaric oxygenation in the patient with malignancy: friend or foe? *Diving and Hyperbaric Medicine.* 2007; 37: 133-8.)

Background: Hyperbaric oxygen (HBO) affects angiogenesis and cellular regeneration and is used to revitalise irradiated tissue. Assuming similarity in pathophysiology it is believed that tumour growth can be stimulated by HBO, and overt or suspected malignancy is considered a contra-indication to HBO by referring specialties.
Objectives: To determine whether the existing evidence supports or refutes this concept and whether the level of evidence utilised affects current published results and conclusions.
Methods: The critical appraisal worksheet for Harm/Aetiology from the Oxford Centre for Evidence Based Medicine was applied to studies of evidence level 3b and above, where a comparison was made of outcomes of malignancy between groups where HBO was used and similar groups without such exposure. Numbers in the treatment arms of selected trials were combined for outcomes which were worse, unchanged or better than those of controls.
Results and Conclusion: Fourteen human trials contributed to the final review. 3,434 patients were represented of whom 1,713 were not subjected to HBO and 1,721 were subjected to HBO as part of either a radiosensitisation protocol or for recovery of late radiation treatment injury. In 195 patients (11.3%) outcomes were worse than controls, 483 patients (28.1%) no difference was detected and in 1,043 patients (60.6%) outcomes were better with HBO. Comparison with existing reviews revealed differences in results but no difference in the trends noted and supports the conclusion that the balance of evidence in the existing literature refutes the perception that HBO is a risk factor for the patient with malignancy.

Introduction

World Health Organization statistics for 2005 estimate that 10 million people are diagnosed with cancer every year.[1] Approximately 50% will receive radiotherapy and of these 50% will be long-term survivors. Radiotherapy injures normal tissue in the field of radiation. Though improvement follows the acute phase, serious radiation-induced complications, known as late radiation tissue injury (LRTI), will affect 5–15% of long-term survivors.[2] With LRTI, progressive reduction in microvascular density and increasing fibrosis result in cellular hypoxia and inability to sustain normal function. This hypoxic, hypovascular and hypocellular situation is exacerbated by infection, surgery or dental extraction and can progress to a critical point where tissue breaks down causing soft-tissue radionecrosis or osteoradionecrosis.[3]

Hyperbaric oxygen (HBO) affects angiogenesis and cellular regeneration and is used to revitalise irradiated tissue.[3] Assuming similarity in pathophysiology, it is believed that tumour growth can be stimulated by HBO, and overt or suspected malignancy is considered a contra-indication to HBO by some referring specialties.[4]

Prior to HBO, the management of LRTI has been less than effective.[5] Surgical or dental intervention in an irradiated area can precipitate disfigurement, poor healing and infection. HBO stimulates angiogenesis in an irradiated field. Typically the treatment regime involves pressures of 243 kPa (2.4 ATA) using 100% oxygen for 90 minutes for 30–40 (usually daily) treatments. Another regime is applied to reduce hypoxia in solid tumours prior to radiotherapy, which involves pressurisation to 202–405 kPa (2.0–4.0 ATA), breathing 100% oxygen for 20–30 minutes for pre-oxygenation, during or following which radiotherapy is delivered.

Radiosensitisation was designed to *decrease* tumour recurrence and metastasis. Concerns that HBO might have cancer-enhancing effects were published by Johnson and Lauchlan in 1966, generating animal and human trials to clarify the effect of HBO on tumour growth.[6] Due to the technical difficulties of combining HBO and radiotherapy, this approach has been largely abandoned despite promising results.

Though the purpose and regimen of HBO as a radiosensitiser differ from the treatment of LRTI, it remains important to consider whether HBO affected tumour recurrence and spread beneficially or adversely. Therefore the outcomes

recorded in these trials have been combined for the purposes of this study. Longer-term follow up of tumour growth was recorded in the trials for radiosensitisation. A challenge in the interpretation of data from treatment for LRTI is the lack of long-term follow-up data recording specifically the presence or absence of tumour recurrence/metastasis. An assumption has to be made, therefore, that if this was not recorded it did not occur. Though apparently a logical conclusion, given the seriousness of recurrence or metastasis, assumptions do not accord well with the scientific method and the tenets of evidence based medicine.[7]

A review by Feldmeier et al included published articles, text books and conference proceedings from 1966–1993 including human and animal data – evidence level 1 (randomised controlled trial) to 5 (case report).[8,9] He concluded that "*the published literature on tumour angiogenesis mechanisms and other possible mechanisms of cancer causation or accelerated growth provides little basis for hyperbaric oxygen to enhance malignant growth or metastases. A history of malignancy should not be considered a contra-indication for hyperbaric oxygen therapy.*"[8] Conclusions, no matter how valid, based on such heterogeneity of evidence should be questioned by practitioners in the disciplines for which this information is critical in terms of referrals. In contrast, two Cochrane reviews selected randomised and quasi-randomised controlled trials only.[10,11]

Whereas trials of drug therapy accommodate large numbers, narrow entry criteria and well-matched placebo controls, practical considerations around generating a valid placebo arm for HBO leads to trials of high-level evidence being much smaller. Surgical disciplines share the challenge of creating valid placebo arms and, in the effort to generate rigorous entry criteria, numbers in such trials are usually small. Meta-analysis of a series of trials with low individual power can lead to confusion about appropriate therapeutic decisions and is associated with the possibility of a Type II error.[7] A case can be made for the inclusion of cohort prospective (level 3) evidence in disciplines where the treatment modality is technical in nature and reviews represent either high numbers with uncritical inclusion, or few numbers with critical appraisal.

Objectives

Assessments were made of human studies from evidence level 3b and higher to answer the questions:

1 Does hyperbaric oxygen treatment pose risks to patients who have known or occult malignancy at the time of their treatment in terms of tumour activation or metastatic spread?
2 Does the evidence level utilised in a review affect trends and clinical conclusions already published?

Search strategy

The following search strategy was used, with an English-language restriction:

- Electronic searches (January 1966–June 2006) were undertaken of the Cochrane Library (CENTRAL Issue 3, 2005); MEDLINE; CINAHL; EMBASE and the database of randomised controlled trials in hyperbaric medicine (DORCTHIM).
- Hyperbaric textbooks and journals (*Undersea and Hyperbaric Medicine*, *South Pacific Underwater Medicine Society Journal* and *Aviation, Space and Environmental Medicine*)
- The reference lists of relevant articles were searched manually.

CRITERIA FOR INCLUSION OF STUDIES

The types of studies included were meta-analyses and reviews, randomised and quasi-randomised controlled trials, cohort and case-controlled studies. The participants and interventions chosen were diagnosed cancer patients with radiotherapy and treated with HBO, compared with similar regimens excluding HBO.

OUTCOME MEASURES

Studies were included if they reported *or were expected to report* the following outcome measures at any time:

- mortality rate
- local recurrence or growth rate
- metastatic disease occurrence or spread.

TRIAL IDENTIFICATION

Abstracts identified by the initial search were assessed and the full text of suitable articles retrieved electronically or from the libraries of the universities of Auckland and Otago or private collections of experts in the field.

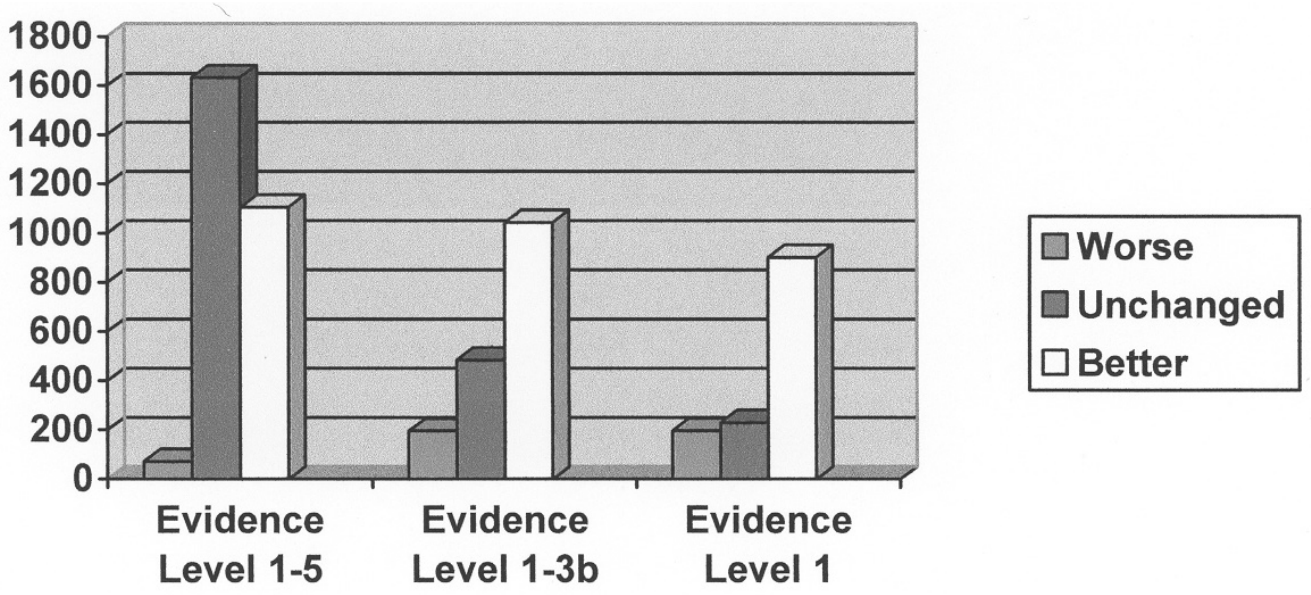
QUALITY ASSESSMENT

Study quality assessment utilised Schulz's method (1995).[12] The reference from a recent comprehensive review was included, giving the rationale for exclusion of duplicate trials.[13]

ANALYSES AND METHODOLOGICAL QUALITY OF INCLUDED STUDIES

The critical appraisal worksheet for Harm/Aetiology from the Oxford Centre for Evidence Based Medicine (EBM) was applied to studies of evidence level 3b and above, where a comparison was made of outcomes of malignancy between groups where HBO was used and similar groups without such exposure.[14] Numbers in the treatment arms of selected trials were combined for outcomes which were worse, unchanged or better than controls. This simple index

Figure 1
Comparison of the numbers of patients in three reviews of the effect of HBO on malignancy, based on different levels of evidence. Patients are categorised as worse off, unchanged or better off than control patients who did not receive HBO.[3,15–27]



outcome was chosen not (as was the original intention of the studies of radiosensitisation and treatment for LRTI) to determine whether HBO was beneficial, but to ascertain whether an adverse outcome in terms of malignancy had occurred that could be attributed to HBO as part of a treatment process.

Computation of risk ratio (RR), confidence interval (CI) and numbers needed to harm was not possible due to the heterogeneity of study types, malignancy types, anatomical sites and levels of evidence.[3,15–27] Whereas RR and CI have been calculated for each randomised controlled trial individually and are not repeated here,[10,11] the combined totals from all trials did not represent figures that could be analysed in this fashion. Data included case-controlled studies which can provide only prevalence of exposure and causation and an odds ratio cannot be calculated. The measure of risk obtained from a case-controlled study is an *estimate* of the RR only.[28] The original review by Feldmeier presented the same difficulties in statistical analysis and as one objective of this review was to provide comparative data, the same type of analysis would need to be applied to all those reviews being compared.

Because of the simplicity of the index endpoint the numbers from the various studies could be combined and percentages of the total calculated to provide comparative data, and histograms for graphic representation of trends. In order to establish whether a causal relationship could be assumed between HBO and changes in the behaviour of malignancy, the Bradford-Hill criteria of causation were applied.[29]

Results are portrayed as histograms from the Feldmeier, Cochrane and the present reviews as the actual numbers of patients combined, where HBO preceded outcomes that were:

- worse than those in the untreated group
- no different from those in the untreated group
- better than those in the untreated group (Figure 1).

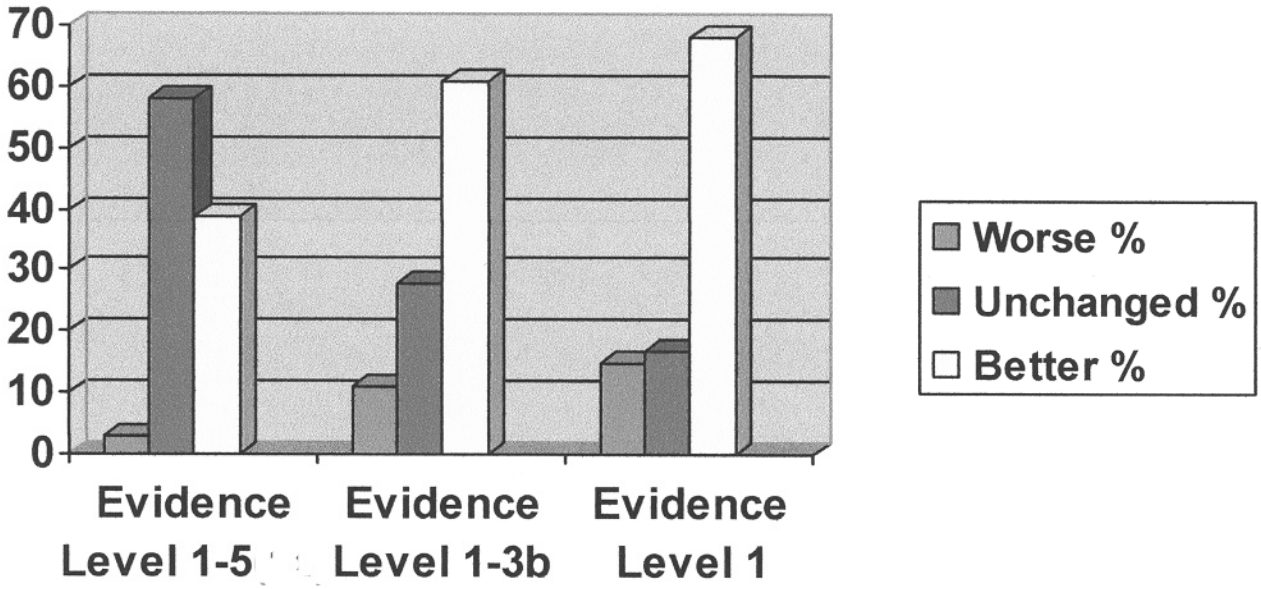
Results

Fourteen human trials from 1966 to July 2006, with level of evidence 1–3b and above contributed to the final review.[3,15–27] A total of 3,434 patients were represented of whom 1,713 were not subjected to HBO and 1,721 were subjected to HBO as part of a radiosensitisation protocol or for recovery of LRTI. Of the latter, 195 had outcomes worse than those of controls, in 483 no difference in outcomes was detected and in 1,043 the outcomes in the HBO group were better than those for controls (Figure 1).

In the review by Feldmeier,[8] combining all studies from 1966 to 1993, 2,808 patients underwent HBO. In 72 patients outcomes were worse with HBO than for controls, in 1,632 no difference in outcomes was seen and in 1,104 exposed to HBO improved long-term outcomes were seen compared to the control group (Figure 1).

Results from the Cochrane reviews,[10,11] from randomised and quasi-randomised controlled trials to 2004, showed that of 1,325 patients treated with HBO, in 195 the outcomes were worse than for controls, in 228 there was no demonstrable difference in outcomes and in 902 the outcome in the HBO group was better than in the control group (Figure 1).

Figure 2
Comparison of the percentage of patients (to allow normalisation of the data) in the three reviews of the effect of HBO on malignancy. Patients are categorised as worse off, unchanged or better off than control patients who did not receive HBO.[3,15–27]



A second graph represents the three outcome groups from the Feldmeier, Cochrane and present reviews expressed as percentages of the combined patient totals, providing a common denominator by which trends can be demonstrated (Figure 2).

Discussion

The use of HBO as an adjunct in head and neck surgery is well established and is increasing for treatment of other irradiated anatomical sites. Such patients usually undergo extensive surgical resections with disruption of vascular supply and resultant chronic hypoxic wounds, radionecrosis or fistula formation adding to the risk of infection. HBO stimulates leukocyte bactericidal activity, angiogenesis, fibroblast activity, and collagen formation creating a favourable environment for healing and resisting infection.[3] HBO in conjunction with radiotherapy has been studied since the 1950s on the assumption that tumour cell hypoxia directly influences radiation. Concern has been expressed over the possibility of increased risk of distant metastases with combination radiation therapy and HBO, whereas the balance of evidence appears to refute this concern.

In all three reviews represented above, where differing levels of evidence were included in each review, the numbers and percentages of patients having received HBO and being perceived as worse off remained in the minority, i.e., 2.6%, 11.3% and 14.7% respectively. It is noteworthy that the higher the level of evidence utilised, the more sensitive the data became to detect evidence of harm. This may reflect a potential positive publication bias particularly in literature of lower evidence level.

However, in terms of those patients perceived to be better off, the same trend to improved outcomes was evident the higher the level of evidence used, i.e., 39.3%, 60.6% and 68.1% respectively. The biggest difference was apparent in the percentages where no change was noted between the hyperbaric group and controls, i.e., 58.1%, 28.1% and 17.2% respectively.

No definite hypothesis can be invoked to account for this. Despite the reversal of trend in terms of 'no difference' to 'better off', the aim of the review remains to ascertain whether the balance of evidence confirms or refutes the perception of harm to patients undergoing HBO with overt or covert malignancy. In this respect the figures of no difference and improved outcomes can be combined as both represent a lack of harm to the patient. From Table 1 it would appear, therefore, that the balance of evidence refutes the perception of harm to the patient with malignancy.

Table 1
Relationship between the levels of evidence used in clinical reviews and the risk of cancer being worsened with HBO. The 'no harm' group combines studies reporting no difference in cancer rates and those with reduced rates in the HBO groups.

Level of evidence	Harm (% patients)	No harm (% patients)
1-5	2.6%	97.4%
1-3	11.3%	88.7%
1	14.7%	85.3%

The second point is that the level of evidence used in the various reviews, though differing in the actual percentages, maintains the same trend and conclusion through all levels. The closest correlation was between the higher levels of evidence. The comparison is not without bias, however, as the uncritical review utilised literature from 1966 to 1993 whereas the other reviews utilised literature from the same starting date, but ended at 2006 (the present study) and 2004 (Cochrane reviews) respectively. Literature that would have been included in the uncritical review had it covered similar dates, was identified and would have changed the figures and percentages to include more in the harm category though still without altering the trends and conclusion.[30]

Despite the weight of evidence in favour of no harm with HBO treatment to the patient with malignancy, the fact that concerns have been raised, and that some outcomes are documented as worse following such treatment, raises the possibility that certain tumour conditions could favour a less than desirable outcome. McMillan et al studied the effect of HBO on developing tumours in a hamster model and found two apparent effects of HBO.[31] They found fewer tumours in the group treated with HBO, but tumours that did develop were larger. They postulated two independent effects: inhibition of tumour growth during induction and enhancement of growth of pre-existing tumours. The inhibitory effects of HBO appear to predominate to some critical tumour bulk/size, at which point a tumour-enhancing effect may be observed. This is postulated to result from angioneogenesis.

In a very similar animal model Marx and Johnson also noted inhibition of tumourigenesis where tumour regression was noted with HBO alone.[32] Lindenschmidt et al observed the tumouricidal effect of HBO in lung tumours as possibly due to the interaction of cell membranes with free oxygen radicals such as peroxide and superoxide.[33] Where protective enzymes such as superoxide dismutase are in limited supply these radicals are demonstrated to destroy cell membranes with a mechanism similar to the action of some antineoplastics.[34]

Conclusions

IMPLICATIONS FOR CLINICAL PRACTICE

In the existing literature, the balance of evidence refutes the perception that HBO poses a risk for the patient with malignancy. Those patients for whom HBO is indicated for the treatment of LRTI should not have this therapy denied them because of the fear of increased tumour recurrence or metastatic spread. The level of evidence used in the synthesis of data produced results that differed between the reviews, in particular between no observed difference and improved outcome. The clinical conclusion, however, remained the same for all reviews. Despite the predominance of a predicted favourable outcome for patients with malignancy undergoing HBO, a better understanding is warranted to be able to identify minority groups with a measure of risk.

IMPLICATIONS FOR RESEARCH

A strong case has been made for large randomised trials of high methodological rigour to define the extent of benefit from the administration of HBO for patients with late radiation tissue injury.[19] By extending follow up on an existing trial the prerequisites of power to detect expected differences, suitable patients, consistency of dose and effective sham therapy will all ideally have been met. Elucidation of any adverse effects would need longer follow up, ideally to five years. Mortality data, quality-of-life scores and long-term cost utility could be gathered concurrently.

References

1 WHO. World Health Organization website: Cancer. <http://www.who.int/cancer/en/2005>

2 Anscher MS. The irreversibility of radiation induced fibrosis: Fact or folklore? *J Clin Oncol*. 2005; 23: 8551-2.

3 Marx RE. Radiation injury to tissue. In: Kindwall EP, Whelan HT, editors. *Hyperbaric medicine practice*. Flagstaff: Best Publishing; 1995. p. 665-718.

4 Feldmeier JJ, Heimbach RD, Davolt DA, Brakora MJ. Hyperbaric oxygen and the cancer patient: a survey of practice patterns. *Undersea Hyperb Med*. 1993; 20: 337-45.

5 Stone HB, Coleman CN, Anscher MS, McBride WH. Effects of radiation on normal tissue: consequences and mechanisms. *Lancet Oncology*. 2003; 4: 529-36.

6 Johnson RJR, Lauchlan SC. Epidermoid carcinoma of the cervix treated by 60CO therapy and hyperbaric oxygen. In: *Proceedings of the third international congress on hyperbaric medicine*. 1966. p. 648-52.

7 Sachett DL, Straus SE, Richardson WS, Rosenberg W, Haynes RB. *How to practice and teach evidence-based medicine*, 2nd edition. Edinburgh: Churchill Livingstone; 2000. p. 29-67.

8 Feldmeier J, Carl U, Hartmann K, Sminia P. Hyperbaric oxygen: Does it promote growth or recurrence of malignancy? *Undersea Hyperb Med*. 2003; 30: 1-18.

9 Oxford Centre for Evidence Based Medicine. *Levels of Evidence* (May 2001). <http://wwwcebm.net/levelsofevidence.asp>

10 Bennett MH, Feldmeier J, Smee R, Millross C. Hyperbaric oxygen for tumour sensitization to radiotherapy. In: *The Cochrane Database of Systematic Reviews*. 2005. 3.

11 Bennett MH, Feldmeier JJ, Smee R, Hampson N. Hyperbaric oxygen therapy for late radiation tissue injury. In: *The Cochrane Database of Systematic Reviews*. 2005. 3.

12 Schulz KF, Chalmers I, Hayes RJ, Altman DG. Empirical evidence of bias: dimensions of methodological quality

associated with estimates of treatment effects in controlled trials. *JAMA*. 1995; 273: 408-12.
13 Bennett MH. *The evidence basis of diving and hyperbaric medicine – a synthesis of the high level clinical evidence with meta-analysis*. Australasia Digital Thesis Program. <http://adt.caul.edu.au/2006>
14 Oxford Centre for Evidence Based Medicine. *Critical Appraisal Worksheet for Harm /Aetiology*. 2001; <http://www.cebm.net/worksheet_harm.asp>
15 VandenBrenk HAS, Madigan JP, Kerr RC. An analysis of the progression and development of metastases in patients receiving x-radiation in hyperbaric oxygen. *Clin Radiol*. 1967; 18: 54-61.
16 Cade IS, McEwen JB. Megavoltage radiotherapy in hyperbaric oxygen. A controlled trial. *Cancer*. 1967; 20: 817-21.
17 Johnson RJR, Walton RJ. Sequential study of the effect of the addition of hyperbaric oxygen on the 5-year survival rates of carcinoma of the cervix treated with conventional radiation fractions. *Am J Roentgenol Ra*. 1974; 120: 111-7.
18 Henk JM, Kunkler PB, Smith CW. Radiotherapy and hyperbaric oxygen in head and neck cancer. Final report of first controlled clinical trial. *Lancet*. 1977; 8029: 101-3.
19 Henk JM, Smith CW. Radiotherapy and hyperbaric oxygen in head and neck cancer. Interim report of second clinical trial. *Lancet*. 1977; 8014: 104-8.
20 Bennet MB, Sealy R, Hockly J. The treatment of stage III squamous cell carcinoma of the cervix in air and in hyperbaric oxygen. In: *Proceedings 6th International congress on Hyperbaric Medicine*; 1977. p. 247-52.
21 Perrins DJD, Wiernik G. Controlled trials in carcinoma of the bladder. In: Smith G, editor. *Proceedings of the sixth international congress on hyperbaric medicine*. Abderdeen: University Press; 1977. p. 253-8.
22 Watson ER, Halnan KE, Dische S, Saunders MI, Cade IS, et al. Hyperbaric oxygen and radiotherapy: a medical research council trial in carcinoma of the cervix. *Br J Radiol*. 1978; 51: 879-87.
23 Dische S. Hyperbaric oxygen: the Medical Research Council trials and their clinical significance. *Br J Radiol*. 1979; 51: 888-94.
24 Brady LW, Plenk HP, Hanley JA, Glassburn JR, Kramer S, et al. Hyperbaric oxygen therapy for carcinoma of the cervix stages – IIB, IIA, IIIB and IVA: results of a randomized study by radiation therapy oncology group. *Int J Radiat Oncol Biol Phys*. 1981; 7: 991-8.
25 Eltorai I, Hart GB, Strauss MB, Khonsari R, Montroy RE. Does hyperbaric oxygen provoke an occult carcinoma in man? In: Kindwall EP, editor. *Proceedings of the eighth international congress on hyperbaric medicine*. San Pedro: Best Publishing; 1987. p. 18-29.
26 Denham W, Yeoh EK, Wittwer G, Ward CG, Ahmad AS, et al. Radiation therapy in hyperbaric oxygen for head and neck cancer at Royal Adelaide Hospital – 1964 to1969. *Int J Radiat Oncol Biol Phys*. 1987; 13: 201-8.
27 Xin PJ, Miao GC, Zong WC, Rong WC, Rong WS, et al. The influence of hyperbaric oxygenation on chemotherapy effects in patients with malignant lymphoma. In: Wen-ren Li, *Proceedings of the eleventh international congress on hyperbaric medicine*. Flagstaff: Best Publishing; 1993. p. 44-7.
28 Friedman GD. Introduction to epidemiology. In: *Primer of epidemiology*. New York: McGraw-Hill; 1994. p. 55-63.
29 Hill AB. The environment and disease: association or causation? *Proc R Soc Med*. 1965; 58: 295-300.
30 Bradfield JJ, Kinsella JB, Mader JT, Bridges EW, Calhoun KH. Rapid progression of head and neck squamous carcinoma after hyperbaric oxygenation. *Otolaryngol Head Neck Surg*. 1996; 114: 793-7.
31 McMillan T, Calhoun KH, Mader JT, Stiernberg CM, Rajaraman S. The effect of hyperbaric oxygen therapy on oral mucosal carcinoma. *Laryngoscope*. 1989; 99: 241-4.
32 Marx RE, Johnson RP. Problem wounds in oral and maxillofacial surgery: the role of hyperbaric oxygen. In: Davis JC, Hunt TK, editors. *Problem wounds: the role of oxygen*. New York: Elsevier; 1987. p. 107-9.
33 Lindenschmitt RC, Tryka AF, Witschi HP. Inhibition of mouse lung tumour development by hyperoxia. *Cancer Res*. 1986; 46: 1994-2000.
34 McCord JM, Fridovich I. The biology and pathology of oxygen radicals. *Ann Intern Med*. 1978; 89: 122-7.

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Case report

Transient prosopagnosia resulting from a cerebral gas embolism while diving

Colin M Wilson, Martin DJ Sayer and A Gordon Murchison

Key words
Cerebral arterial gas embolism (CAGE), arterial gas embolism, decompression illness, prosopagnosia or visual agnosia, treatment, case reports

Abstract
(Wilson CM, Sayer MDJ, Murchison AG. Transient prosopagnosia resulting from a cerebral gas embolism while diving. *Diving and Hyperbaric Medicine*. 2007; 37: 139-42.)
A 33-year-old male was diving in excess of 50 metres of water in a marine tunnel at a remote location off the west coast of Scotland when he ran out of air. He made an emergency ascent and was recovered unconscious on the surface; he was not breathing, had no pulse and had missed a significant amount of decompression. Following resuscitation he was transferred by helicopter to the Dunstaffnage Hyperbaric Unit where he received hyperbaric oxygen therapy for a cerebral arterial gas embolism. He made a quick recovery but during the treatments he demonstrated a number of neurological abnormalities including visual disturbances which were diagnosed as prosopagnosia, the inability to recognise faces. The effects were transient and the patient went on to make a full recovery. Prosopagnosia is a rare affliction and this is thought to be the first reported case that appears to have occurred as a direct result of a diving accident. The case is described in detail and prosopagnosia is reviewed.

Introduction

Arterial gas embolism (AGE) is a recognised serious problem related to diving, caused when gas passes through the alveolar walls and into the pulmonary vasculature.[1] There are a wide number of signs and symptoms related to AGE depending where the bubbles are distributed in the brain.[1] Prosopagnosia is a rare condition in which individuals are unable to recognise faces. More unusually transient prosopagnosia has been reported following ischaemic cerebral vascular accidents, as part of normal pressure hydrocephalus and following temporal surgery for epilepsy.[2–4] It does not appear to have been reported previously in relation to diving. This report gives details of a rare case of transient prosopagnosia following a cerebral gas embolism while diving.

Case report

JP was a 33-year-old television engineer who was an experienced sports scuba diver for 16 years, with no past medical history of note or history of any diving-related medical problems. He was taking part in a liveaboard expedition which had departed from Oban (on the west coast of Scotland) to the isolated volcanic archipelago of St Kilda, lying in the Atlantic 45 miles to the west of the Outer Hebrides, 170 miles from Oban (see postscript).

The diving expedition started with a "shake down" dive on the first afternoon to a maximum depth of 30 metres' sea water (msw) for a total duration of 18 minutes. Following that dive, the liveaboard vessel began the 170 mile journey to the St Kilda islands; the boat arrived there late in the evening of the following day. On the day following arrival, JP carried out a single dive to a maximum depth of 61.4 msw with a total dive time of 34 minutes.

After a surface interval of 22 hours, he carried out his third dive, the incident dive, at 1112 hr to a maximum depth of 51.8 msw for about 30 minutes. He entered the water with twin air tanks of unknown capacity and descended on his own. He entered a cave at about 50 msw and prepared to wait for his companions with his camera. While he waited he began exploring, on his own, one of the tunnels and swam along to its end. During this initial exploration he dipped into a couple of dead-end holes. Still at the end of the tunnel he was aware that he still had not been joined by his companions and so he decided to return to the entrance. However, on his way back he encountered a strong current and remembered having to swim with considerable effort, finning hard and pulling himself along the cave wall. At the same time he remembered that he was "dragging" on his demand valve. He also remembers being relieved to have made it back to the entrance and seeing the sun filtering down through the water. Unfortunately because of the excessive effort used, JP had run out of air and he was still at a depth of approximately 50 msw. He had no recollection of surfacing but he had appeared to have inflated his buoyancy jacket and surfaced rapidly. He had some vague recollection of being in a helicopter but really remembered nothing for the next three hours until he was being examined at the hyperbaric unit at Dunstaffnage, near Oban.

According to the divers on the scene he was seen floating on the surface at 1145 hr. He was recovered into a dinghy, when he was noted to not be breathing and have no pulse, so they commenced cardiopulmonary resuscitation, carrying it out for 5–10 minutes as he was taken back to the dive boat. His twin tanks were noted to be empty as his gear was removed; his drysuit was cut off and he was placed in the recovery position as he was by now breathing and had a pulse.

He was taken ashore to the Ministry of Defence facilities on St Kilda and was attended by an army medic. The emergency services were contacted and Stornoway Coastguard tasked their helicopter for urgent evacuation from St Kilda to Dunstaffnage. He remained deeply unconscious for a further 30 to 40 minutes, was given high-flow oxygen via closed re-breathing trauma mask and the medic started an intravenous infusion. During the helicopter transfer oxygen and IV fluids were continued as his conscious level started to improve; he arrived at the Dunstaffnage Hyperbaric Unit at 1430 hr.

Examination on admission found him confused, with poor recollection of recent or past events, and with disorientation in time and place. Finger–nose coordination was poor and he was noted to have dysdiadochokinesia. He had generalised bilateral weakness of the thigh and calf muscles (grade 3). Reflexes were brisk in both knees and ankles with sustained clonus in both ankles. Clonus was also triggered by having his reflexes tested. He had been incontinent of faeces and possibly of urine. His cranial nerves were intact.

Recompression commenced at 1445 hr to 18 metres (283 kPa) following the Royal Navy treatment table 62 protocol. At the first air break he was less confused. At the second air break he was able to stand unsupported, carry out a heel-shin slide while standing and carry out accurate serial sevens. As he reported to be feeling "absolutely normal" and examination showed no abnormality the treatment table was continued without extensions, surfacing at 1930 hr. He later admitted, however, having difficulty in distinguishing or recognising the faces of those who were treating him.

On completion of the treatment, he was admitted to the local hospital for post-treatment monitoring. He kept asking as to what had happened to him and if he had inflated his buoyancy jacket. He also now reported some problem with his vision. Though he had a history of an amblyopic left eye since childhood, his near and distance vision were normal, he was able to recognise objects, and could see people but had difficulty in recognising the faces of those who had been closely involved in his recompression.

He slept well overnight with further recollections of the previous day's events. Full examination the next day was normal except for the 'visual disturbance' with continuing problems in "seeing faces as to who they were". As there was incomplete resolution of his symptoms he was recompressed using a Comex 12 hyperbaric oxygen (HBO) table (150 minutes at a maximum pressure of 222 kPa, 12 msw depth). Although apparently more relaxed after this he described faces as being indistinct around the mouth, nose and eyes, appearing "cartoon-like", and found recognising people by their faces as being difficult. He realised that he was recognising people by their voices or the glasses they were wearing rather than by their faces.

The following day his condition remained unchanged and so he was referred to the specialist neurology unit in Glasgow for assessment and CT scanning. Prior to transfer he underwent a further Comex 12 HBO treatment, with seemingly complete resolution of his ongoing problem. Clinical assessment by the neurologist found him to be completely normal and likewise the CT scan they carried out was normal. He remained well and was discharged the following day to his home in Glasgow. He returned the following day to the pier in Oban to welcome back the dive boat after completion of the trip, showing his colleagues that he had recovered completely and thanking them for their life-saving help.

JP was contacted seven years after his incident and was found to be in good health with no ongoing problems and without any return of his prosopagnosia. He had returned to diving two months after his incident, though he had restricted his diving to 30 metres or less. There had been no further episodes of decompression illness and he remained a happy man following this near-death experience.

In summary, following a rapid ascent from depth with omitted decompression, this man suffered a near-fatal arterial gas embolism (AGE) with cardiorespiratory arrest, after running out of air. He had neurological abnormalities as a result, which all resolved with recompression, including a visual problem diagnosed as prosopagnosia.

Discussion

Anyone suffering an AGE will have a rapid onset with variation in symptoms. Five per cent of patients suffering a catastrophic AGE will experience apnoea, unconsciousness and cardiac arrest immediately and many are unresponsive to CPR.[5] Having survived the initial event, with successful CPR in this case, the most appropriate assistance was sought with intravenous fluid resuscitation and high-flow oxygen and rapid evacuation. Recompression was commenced three hours after surfacing with apparent rapid resolution of symptoms. The resultant visual problem of prosopagnosia became apparent only during the time following the patient's first treatment, but completely resolved after two further HBO treatments.

Agnosia is "*the loss of the ability to recognise objects, persons, sounds, shapes or smells while the specific sense is not defective, nor is there a specific memory loss*." Usually agnosia is associated with brain injury or neurological illness, particularly after damage to the temporal lobe. Specifically

there is a group of visual agnosias, with prosopagnosia being the inability to recognise faces. The name originates from the Greek '*prosopon*' – 'face' and '*agnosia*' – 'non-knowledge'. It was described by both Jackson and Charcot in the 19th century but the term was first used by Bodamer in 1947.[6]

Patients suffering from prosopagnosia have the ability to recognise all objects except faces, including their own when viewed in a mirror. The patient recognises people instead by their clothing, hair colour, body shape, voice, spectacles, etc. There have been some peculiar cases of prosopagnosia reported: for example, a dairy farmer who developed prosopagnosia lost the ability to recognise the individual cows in his herd and a prosopagnosic bird-watcher reported "all birds look the same".[7] Evidence shows that prosopagnosia is usually associated with extensive damage to the temporal and occipital lobes, particularly in the region of the right occipitotemporal or fusiform gyrus.[8] Recent research, however, suggests that there are individuals who may also have a congenital or heritable cause.[9]

Early researchers of agnosias theorised that visual agnosia was the result of a reduction in low-level visual processing with impairments to mental abilities. This was often termed the sensory-deficit account.[10] There were, however, problems with this theory and the more recent "peppery mask" account offers a better explanation.[11] This theorises that the presence of random visual noise is caused by obstructing air bubbles circulating in the blood or the presence of blood clots in an intact blood vessel. Such impairment makes it difficult to organise information in its totality. It is highly probable that, in this case, the bubbles created by the embolism and the resultant tissue reaction have affected the area of the brain that supports the function of face recognition. This supposition is supported by the resolution of the visual disturbance following the third HBO treatment, after which it might be expected that any remaining bubbles would have been significantly reduced in size or eradicated and the tissue reaction improved or resolved.

Oliver Sacks, the eminent neurologist, wrote a short story entitled "*The man who mistook his wife for a hat*" about a music professor with a visual agnosia and prosopagnosia.[12]

"*Sometimes a student would present himself, and Dr P would not recognise his face. The moment the student spoke, he would recognise his voice … For not only did Dr P increasingly fail to see faces, but saw faces when there were no faces to see; genially, Magoo-like, when in the street, he might pat the heads of hydrants and parking meters, taking these to be the heads of children; he would amiably address carved knobs on furniture and be astounded when they did not reply.*

He also appeared to have decided that the examination was over and started to look round for his hat. He reached out his hand and took hold of his wife's head, tried to lift it off, to put it on his own head. He had apparently mistaken his wife for a hat! His wife looked as if she was used to such things."

References

1 Neuman TS. Arterial gas embolism and pulmonary barotraumas. In: Brubakk AO and Neuman TS, editors. *Bennett and Elliott's physiology and medicine of diving*, 5th ed. Philadelphia: Saunders; 2004. p 557-7.

2 Lang MD, Baudewig J, Kallenberg K, Antal A, Happe S, et al. Transient prosopagnosia after ischemic stroke. *Neurology*. 2006; 66: 916.

3 Otani N, Nawashiro H, Ishihara S, Fukui S, Katoh H, et al. Normal pressure hydrocephalus manifesting as transient prosopagnosia, topographical disorientation and visual objective agnosia. *J Clin Neurosci*. 2004; 11: 313-7.

4 Mesad S, Laff R, Devinsky O. Transient postoperative prosopagnosia. *Epilepsy Behav*. 2003; 4: 567-70.

5 Neuman T, Pulmonary barotrauma. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 185-94.

6 Bodamer J. Die Prosop-agnosie. *Archiv fur Psychiatrie und Nervenkrankheiten*. 1947; 179: 6-53.

7 Farrah M. *Visual agnosia: Disorders of object recognition and what they tell us about normal vision*. Cambridge, MA: MIT Press, Bradford books; 1990. p. 202.

8 Barton JJS, Press DZ, Keenan JP, O'Connor M. Lesions of the fusiform face area impair perception of facial configuration in prosopagnosia. *Neurology*. 2002; 58: 71-8.

9 Behrmann M, Avidan G. Congenital prosopagnosia: face-blind from birth. *Trends Cogn Sci*. 2005; 9: 180-97.

10 Vecera SP, Gilds KS. What processing is impaired in apperceptive agnosia: Evidence from normal subjects. *J Cogn Neurosci*. 1998; 10: 568.

11 Farah MJ, Monheit MA, Wallace MA. Unconscious perception of extinguished visual stimuli: Reassessing the evidence. *Neuropsychologia*. 1991; 29: 949-58.

12 Sacks O. *The man who mistook his wife for a hat*. London: Picador; 1985.

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Postscript concerning St Kilda

St Kilda is currently one of the few double World Heritage sites in the world.[1] The fragile community that inhabited these islands for 4,000–5,000 years lived by a system of what was described as feudal communism. The community was sustained predominantly by obtaining food by scaling the cliffs and sea stacks and collecting seabirds and their eggs. The inhabitants had little or no contact with the outside world until the 18th century when curious visitors made their way there, unfortunately bringing diseases unknown to the local inhabitants. In 1724 smallpox reduced the population of St Kilda from 124 to 30. Between 1830 and 1843, 80% of the babies born on the islands died of neonatal tetanus. In 1852, 36 of the islanders emigrated to Australia and, although 20 died on the voyage, the suburb of St Kilda in Melbourne was established. On St Kilda, no-one paid taxes, no-one was registered to vote and no-one was called up to the armed services; they were left to their own devices. After petitioning the UK government in 1930, the remaining 36 islanders were evacuated to the Scottish mainland, as life had become unsustainable on St Kilda.[2]

Today, St Kilda is owned by the National Trust for Scotland (NTS), who maintain a small presence there in the summer months. St Kilda has one of the most extensive groups of vernacular building remains in Britain.[3] The layout of the 19th-century village remains to this day and over 1,400 stone-built cleitean for storing food and fuel are scattered all over the islands, and even on the sea stacks. The Ministry of Defence lease part of the islands and maintain a missile tracking station there. The islands have few visitors, from yachtsmen, diving groups and volunteer work parties for the NTS. Diving at St Kilda is considered to be the finest in the UK with clear water of 50–60 metre visibility, sea caves and arches, and interesting marine life, though the diving there is challenging because of the depths of the dives and the near continuous sea swell and currents.

References

1 UNESCO World Heritage web site. <http://whc.unesco.org/en/list/387> (last accessed 29 August 2007).
2 MacLean C. St *Kilda: Island on the edge of the world*. Edinburgh: Canongate Books Ltd.; 2006.
3 National Trust for Scotland (NTS) St Kilda. <http://www.kilda.org.uk/> (last accessed 29 August 2007).

This case report was presented for discussion by Dr Wilson at the SPUMS Annual Scientific Meeting in New Zealand, 2007.

Short communication

Effects of a single hyperbaric oxygen exposure on haematocrit, prothrombin time, serum calcium, and platelet count

Harold W Hibbs, Myroslav P Harasym, Dheeraj Bansal and James Stewart

Key words
Hyperbaric oxygenation, oxygen, platelets, coagulation, haematology, research

Abstract

(Hibbs HW, Harasym MP, Bansal D, Stewart J. Effects of a single hyperbaric oxygen exposure on haematocrit, prothrombin time, serum calcium, and platelet count. *Diving and Hyperbaric Medicine*. 2007; 37: 143-5.)
We investigated whether administration of hyperbaric oxygen (HBO) affects platelet counts and some components of the haemostatic system. Ten test subjects were treated with 100% hyperbaric oxygenation at 253 kPa (2.5 ATA) for 60 minutes. A comparison was made between pre-exposure and post-exposure measurements of haematocrit, prothrombin time (PT), serum calcium concentration, and platelet counts. While no significant changes were detected in haematocrit, PT, or serum calcium concentration, platelet levels demonstrated significant decreases with a mean pre-HBO platelet count of 283 x 10^3 mm^3 $\pm$ 32 compared to a mean post-HBO platelet count of 255 x 10^3 mm^3 $\pm$ 33 (P = 0.001). This is consistent with previous studies modelling coagulation pathways and suggests that HBO may activate components of the haemostatic system. Characterisation of the mechanisms associated with this decrease in platelet count by future studies may provide insight into platelet adhesion and aggregation properties during exposure to high oxygen tensions, and may find applicability towards the vascular component of disorders in which oxidant stress and coagulation are prominent, such as hypertension, diabetes, and coronary artery disease.

Introduction

The effects of hyperbaric oxygen (HBO) upon the haemostatic system have not been entirely determined although previous studies indicate several haemostatic factors are affected by HBO. Olszanski et al found a decrease in platelet count and factors I, X, and XII after air, but not helium, diving.[1] Ersoz et al reported that, in New Zealand rabbits, HBO results in decreased collagen-induced platelet aggregation.[2] Yamami et al found differences in human fibrinolytic activity after HBO.[3] More recently, studies by Thom et al and Puthucheary et al demonstrate respectively unaltered platelet function and decreased exhaled nitric oxide (NO) after HBO.[4,5] This study was designed to test the null hypotheses that HBO would have no effect upon: 1) haematocrit, 2) prothrombin time (PT), 3) serum calcium concentration, or 4) platelet count.[1]

Subjects and methods

The study design was a non-randomised, before-and-after study that was evaluated by the Saba University School of Medicine (SUSOM) Ethics Committee, Human Subjects Review Board. Informed consent was obtained from all subjects. All testing was completed within 90 minutes for each subject, eliminating possible diurnal variations. Baseline laboratory values for each subject were ascertained before HBO and served as the control measurements for post-HBO exposure.

Inclusion criteria were: 1) at least 18 years of age, 2) no bleeding disorders, 3) no prescription medications, and 4) no contra-indications for HBO. Eight male and two female volunteers were recruited from SUSOM, aged between 24 and 50 years. Subjects were instructed to avoid products containing aspirin for at least four days, and to fast for four hours prior to the study. Subjects were then compressed in pairs in a Dräger 5.19 m^3 multiplace hyperbaric chamber to 253 kPa (2.5 ATA) for 60 minutes, breathing 100% oxygen continuously through a tight-fitting oxygen delivery mask that covered the nose and mouth. As the risks for oxygen toxicity and decompression illness were minimal, there were no oxygen breaks and both compression and decompression were accomplished within five minutes.

Blood samples were drawn from the right median anticubital vein immediately prior to entering the chamber and from the left median anticubital vein within 15 minutes of exiting the chamber. Each set of blood samples was examined to measure haematocrit, PT, serum calcium and platelet count.[6] Blood was collected into a 4.5 mL EDTA tube (haematocrit and platelet count), 4.5 mL sodium citrate tube (serum calcium concentration), and 5 mL SST tube with gel and clot activator (PT).

Haematocrit was measured using a standard capillary tube centrifugation technique and expressed as a percentage of the total column height.[6] PT was performed using a manual technique. Following centrifugation and separation, the

plasma was incubated for three minutes at 37 +/- 0.5^{0}C. Dade Thromboplastin C plus was reconstituted with 10 ml distilled water and incubated in the same manner as the plasma. Three parts of thromboplastin were added to one part plasma. The time to achieve visible coagulation in two aliquots was recorded and averaged.[6] Platelet count was measured using Unopipette; one drop was pipetted onto each side of a haemocytometer and allowed to stand for at least three minutes before the platelet count was read.[6] Serum samples for calcium concentrations were separated, frozen and analysed six days later. Serum was added to a pretreated slide and allowed to stand for at least three minutes. After the incubation period, total calcium was measured using an automated spectrophotometer.[6]

Data were evaluated using a paired T-test with the statistical software Minitab version 15; a P-value less than 0.05 was considered significant. A pre-study power analysis was not calculated.

Results

There were no significant differences in haematocrit, PT and serum calcium following HBO compared with before exposure (variation < 1%, Table 1).

Platelet counts (Table 1 and Figure 1) demonstrated significant changes post-HBO. All subjects demonstrated a decreased platelet count with a mean pre-HBO platelet count of 283 x 10^3 mm^3 $\pm$ 32 and mean post-HBO platelet count of 255 x 10^3 mm^3 $\pm$ 33 (P = 0.001).

Discussion

The responses of healthy volunteers may not represent those of patients undergoing HBO therapy. No external control group was used because there is no evidence that platelet counts or the other parameters typically change over 90 minutes in healthy individuals. Large numbers of subjects would have been needed to improve the power of the study for parameters other than the platelet count. There was no measurement of a dose/response relationship since only one exposure of HBO was administered. There were no follow-up samples collected to measure the duration of any changes observed.

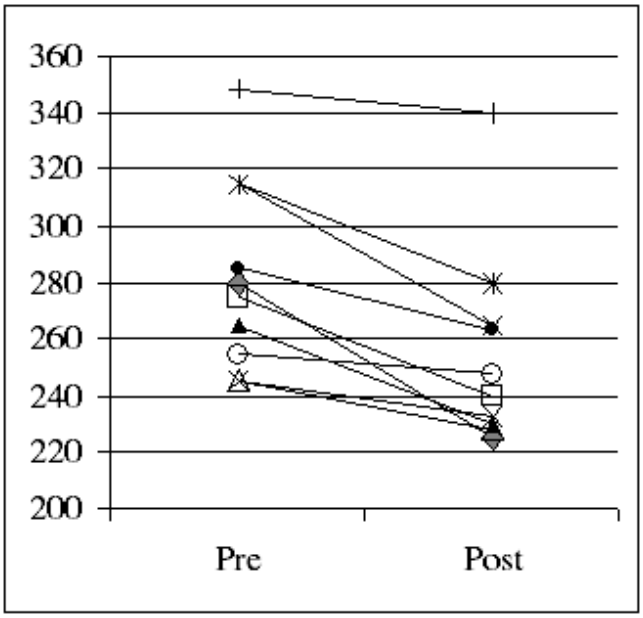
The data reveal a decrease in platelet count soon after a single exposure to HBO. This decrease could be attributed to the modulating effects of reactive oxygen species (ROS) inhibiting and scavenging NO production and bioavailability during HBO. Jaimes et al suggest that ROS significantly impair endothelial nitric oxide synthase (eNOS) activity, while Krotz et al suggest that superoxide anion may induce platelet aggregation.[7,8] Clutton et al suggest that platelet-derived NO contributes to an inhibition of platelet activation.[9]

Table 1
Haematocrit, prothrombin time, total calcium and platelet count pre- and post-hyperbaric exposure to 253 kPa pressure oxygen for 60 mins; mean $\pm$ SD; *P = 0.001

Sample	Pre-HBOT	Post-HBOT	Percentage difference
Haematocrit (Per cent %)	42%	42%	0%
Prothrombin (time, seconds)	12.1 $\pm$ 0.4	12.2 $\pm$ 0.4	< 1%
Total calcium (mm.L^{-1})	2.45 $\pm$ 0.14	2.46 $\pm$ 0.05	< 1%
Platelet count (x100,000 mm^3)	283 $\pm$ 32	255 $\pm$ 33*	~ 10%

There has been recent research that suggests inflammatory reactions may modulate NO levels by up-regulating inducible nitric oxide synthase (iNOS). Puthucheary et al reported no changes in platelet counts for healthy attendants breathing air at 243 kPa or breathing oxygen at 101.3 kPa.[5] What Puthucheary et al suggest is an up-regulation of iNOS in patients with inflammatory reactions that becomes significantly reduced after HBO. Interestingly, the inflammatory up-regulation of iNOS may explain the conclusion of Thom et al that HBO had no effect upon platelet count, especially since the subjects in that study

Figure 1
Platelet counts pre- and post-exposure to hyperbaric oxygen at 253 kPa pressure for 60 mins



were undergoing prophylactic HBO for osteoradionecrosis and potentially had disease processes that promoted inflammatory responses.[4] This could explain the difference in results as compared with the present study, which utilised healthy volunteers.

Is the platelet reduction revealed in this study a temporary manifestation or a longer-lasting degradation? One proposed mechanism suggests that temporary platelet adhesion to the endothelium lasts only a few hours after HBO as levels of ROS become reduced and normal levels of NO become replenished by eNOS. However, a second possible mechanism proposes that micro-aggregates form within the vasculature and eventually become sequestered and degraded by the spleen, which would result in a longer duration of decreased platelet count. Additionally, if micro-aggregates are formed, this poses a potentially dangerous side-effect for patients with increased risk factors for thrombus formation, such as those with vascular disease and diabetes. The data from this study cannot reliably suggest which mechanism may be responsible for the observed decrease in platelet count.

Conclusion

A single exposure to HBO at 253 kPa for 60 minutes was shown to lower platelet levels in healthy individuals. The exact mechanism by which this is accomplished is unknown; however, the possibilities include permanent degradation of platelets by the spleen or simply a temporary binding of platelets to the endothelium.

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References

1 Olszanski R, Radziwon P, Baj Z, Kaczmarek P, Giedrojc J, et al. Changes in the extrinsic and intrinsic coagulation pathways in humans after decompression following saturation diving. *Blood Coagul Fibrinolysis*. 2001; 12: 269-74.
2 Ersoz G, Ocakcioglu B, Bastug M, Ficicilar H, Yavuzer S. Platelet aggregation and release function in hyperbaric oxygenation. *Undersea Hyperb Med*. 1998; 25: 229-32.
3 Yamami N, Shimaya K, Sera AM, Fujita H, Shibayama M, et al. Alterations of fibrinolytic activity in human during and after hyperbaric oxygen exposure. *Appl Human Sci*. 1996; 15: 239-42.
4 Thom SR, Fisher D, Stubbs JM. Platelet function in humans is not altered by hyperbaric oxygen therapy. *Undersea Hyperb Med*. 2006; 33: 81-3.
5 Puthucheary ZA, Liu J, Bennett M, Trytko B, Chow S, Thomas PS. Exhaled nitric oxide is decreased by exposure to the hyperbaric oxygen therapy environment. *Mediators Inflamm*. 2006; Article ID 72620: 1-6.
6 Henry, J. *Clinical diagnosis and management by laboratory methods*, vol I, 16th ed. Philadelphia: Saunders; 1979. p. 289-90, 872-74, 882-3, 1156-7.
7 Jaimes EA, Sweeney C, Raij L. Effects of the reactive oxygen species hydrogen peroxide and hypochlorite on endothelial nitric oxide production. *Hypertension*. 2001; 38: 877-83.
8 Krötz F, Sohn HY, Pohl U. Reactive oxygen species: players in the platelet game. *Arterioscler Thromb Vasc Biol*. 2004; 24: 1988-96.
9 Clutton P, Miermont A, Freedman JE. Regulation of endogenous reactive oxygen species in platelets can reverse aggregation. *Arterioscler Thromb Vasc Biol*. 2004; 24: 187-92.

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$200,000 fine for diving death

A Melbourne diving company was fined $200,000 after failing to ensure the safety of an inexperienced diver who drowned after experiencing problems with his rented diving equipment during a dive out of Portsea in January 2004. Found guilty by a jury of failing to ensure the victim's care under the terms of the Occupational Health and Safety Act, the judge condemned the company's breach of duties as 'profound' and said that the fatality could have easily been avoided had the company and its employees followed good safety procedures. The judge also said there were many warnings and indications that the victim lacked the skills and experience to dive in Victorian waters. In fining the company an unprecedented $200,000, the judge said that while the company was now in receivership and the fine would, therefore, not be paid, it was important to send a clear message to the diving industry about the responsibility operators had towards their customers.

Opinion

Diving medical courses: a primer

Carl Edmonds

Key words
Underwater medicine, training, general interest

Background

During 2006, being involved in a number of diving medical courses, I contemplated the difficulties of upgrading these courses. I initiated discussions with some of the organisers in the hope of improving the quality and logistics of the educational training we were offering. To my pleasant surprise, most were amenable to this proposal, but simply did not have the time to implement it themselves. As a consequence, I have prepared this paper, which I make available for discussion, and perhaps even implementation in some cases. This paper documents some of the items for consideration by course organisers.

Anyone could have prepared this document. I do it because I have the time and the experience; I do not have the evidence. But, in this context there is a general agreement about the need for the courses and the intended outcomes. We all want more knowledgeable diving clinicians who can make reasonable provisional diagnoses, administer appropriate initial treatments, conduct astute diving medical examinations and refer if appropriate. In this I am running with the pack, not leading it.

I claim no copyright on the suggestions made here. On the contrary, most have come from a variety of sources, including the invaluable critiques of students, the observations of my diving medical colleagues and the exasperation of organisers who did not achieve the outcomes they had contemplated. It is easy to blame the quality of the students for these shortcomings, but often it is the course that has failed them, not vice versa. I also do not blame the lecturers. In this field, most are enthusiastic and competent.

I have lectured at, or directed, more than 150 diving medical courses over the last four decades, and I am grateful to many organisations for demonstrating, to both me and their students, what not to do. My first course was in 1967, at the Royal Australian Navy School of Underwater Medicine. I was allocated two weeks to prepare it. Since then it has not changed much and some of the changes are not necessarily improvements. Nevertheless, it is still one of the best courses available, which does not say a lot about many of its successors.

To define our terms. I am not referring to the three-month US Navy diving medical course, which trains doctors to be divers, diving supervisors and chamber operators. Nor the one-week Royal Navy course that merely titillates the interest of national health service bureaucrats. Nor the 'mickey mouse' diving medicine holiday courses, held on a Pacific island or in the Caribbean for doctors who dive.

I refer to the two-week, introductory, full-time courses carried out in Australia (Royal Australian Navy and Royal Adelaide Hospital) and devised as background for more comprehensive diving medical instruction, performance of diving medical examinations, or training of general practitioners who could perform initial assessments. Those who graduate usually do not operate hyperbaric treatment chambers, and use diving medical consultants for the more serious diving medical problems.

Some differentiate the course into a first 'basic' week and a second 'advanced' week. This has the advantage of permitting delegates to undertake the course over two separate time periods, not requiring a continuous two-week absence from their gainful employment. The depiction of the second week as 'advanced' is presumptuous. It is not necessary to designate it so untruthfully. Using the terms Part 1 and Part 2 of a 'Basic' or 'Introductory' course would serve the same purpose. Claiming that Part 2 is advanced is hypocritical on the part of the organisers and misleading to the students. It is analogous to the recreational diving industry's use of the term 'advanced diving' when referring to the courses that encompass a diver's fifth to eighth open water dives, when the diver is really still a novice.

About 30 is the maximum number of students that can be dealt with by lecturers before teaching becomes purely dictatorial, and thus could be as well done over the internet. Personal interaction is as necessary for the class questioners as it is for the more timid students who do not ask the questions they should. I refer to them all, in class, as 'delegates' but I treat them as students. You cannot underestimate the knowledge of general-trained doctors in this field of diving medicine.

Instructors

A course requires at least three full-time lecturers, if it is to have sufficient variety to avoid over-exposure of lecturers and boredom from their voices. Other specialist lecturers may be required for lectures on respiratory, otological, pathological and marine toxicological topics, depending on the orientations of the full-time lecturers. Preferably even the specialists will have diving experience, to ensure their presentations have relevance and validity. The lecturers are usually:

- divers
- non-diving medical specialists
- medical specialists who are also divers
- diving physicians.

Rule 1 for any lecturer is to keep the class awake. To some degree all lecturers are entertainers or performers – but there must be more to the presentations. Some are fascinating and captivating but, at the end of the performance, one is left with a single 'bottom line' that the lecturer is a fantastic diver, a brilliant researcher or a lovely person. There may be little practical diving medical knowledge imparted. Interesting as these lecturers are to divers and medicos alike, they occupy valuable instructional time and should be allocated to after-dinner speaking, if at all. Researchers should be advised that they are there to teach, not to promote their own hobbyhorse research, unless it is directly relevant to the topic.

A full-time course chaperone, preferably a diving physician, is necessary to analyse the information supplied, note omissions in this, remedy these, integrate the information presented, explain discrepancies and supply continuity. He is also of value in ensuring that back-up logistics (morning and afternoon teas, social functions and lecture technology) are functioning appropriately. A high-class teacher, administrator and 'goffer'.

Syllabus

Assume nil knowledge, especially from new graduates and the older specialists. Otologists do not necessarily understand respiratory function tests, and respiratory physicians may not recognise an audiogram if they tripped over it. New graduates may have a superb theoretical knowledge of functional MRIs, but not be able to read a chest X-ray.

Unfortunately a syllabus, once designed, often becomes the default template for all future years – sometimes 40 of them! New directors may not feel confident enough to alter their predecessors' course, and they often are not aware of previous critiques, or do not take these into account.

The sequence of the lectures is of major importance. Moving sequentially from the simple to the complex is preferred, with dogmatic, easily understood, presentations for the earlier, more basic part of the course. Unless one plans the syllabus so that the student is sequentially instructed from basic physics, physiology, and equipment, to aquatic environment, and finally diving hazards the less sophisticated will become confused. One should evolve from the known and recognised into the more exotic. Jumbling the lectures up according to the availability of lecturers will be disadvantageous to many of the non-diving students trying to comprehend new concepts. Lecturers should fit in with the course, not the other way around.

The principles of a typical course syllabus are as follows.

The first two days are often best presented by diving instructors, as they are accustomed to giving precise lectures, understandable to all, and bereft of medical terminology.

The first day would encompass a review of relevant physics and physiology. There are some excellent but outdated films produced by the Royal Navy and the US Navy, depending on whether you are using metric or imperial measurements. These also introduce the student to the diving diseases, but not in any significant clinical detail. They can be followed by brief oral reiterations of the major facts of the laws of physics, with responses to questions. Examples of the laws of physics should be worked through, as a class. Respiratory and otological physiology may be refreshed during these lectures, but the focus should be strictly on relevant diving physics and physiology. Diseases and pathology should only be touched on, to acquaint the students with the future direction of the lectures. Basic free-diving equipment can be discussed and illustrated. If time permits, to introduce diving medicine and entice the doctors' interest, a lecture on free-diving, by a diving physician, may be incorporated. This is a wonderful introduction to the remainder of the course and it is applicable even to non-divers.

The second day can be devoted to equipment, from free-diving to chambers. Without the availability of films (DVDs now), I would occasionally do a whirlwind "Cook's tour of diving accidents" at the end of the day.

The third day should be devoted to the hazards of the marine environment, applicable to all fishermen, seafarers and water-sports enthusiasts. This expands the range of the course and its relevance to 'non-divers'. These lectures include drowning syndromes, thermal disorders, marine animal injuries, seasickness, etc.

The fourth day is initially devoted to diving fatalities, including the statistics and overviews of the contributing factors. This not only introduces the students to all the major diving diseases, but also gives them some perspective of their relative importance. So often doctors graduate from these courses with no concept of the frequency of stress factors such as panic and fatigue, the significance of drowning and cardiac deaths, or the relative infrequency of decompression sickness (DCS) and contaminated gases in recreational diving fatalities. They have presumed that the time allocated to these subjects is a reflection of their frequency and significance, whereas it really reflects the interests and commitments of the organisers.

The afternoon leads us finally to the very specific diving diseases, starting with the simplest gas law to understand. This day should be "Boyle's Law Day" and kept totally isolated from any discussion on decompression or DCS, otherwise it will cause confusion to the less bright diving medical neophytes.

The fifth day can be "Henry's Law Day" and devoted to the

various gas toxicities and decompression. A temptation to combine decompression with decompression sickness must be resisted. Otherwise some students are going to get very confused. One involves physiological theories, the other a specific medical disease. They are worlds apart. How often do the poor students confuse flying-after-diving restrictions (decompression) with medivac altitudes (DCS)?

The sixth day (hopefully with a weekend away from decompression calculations) is for DCS and other serious or specific diving diseases. First aid and resuscitation fits in well, as does the epidemiology of non-fatal diving accidents.

The whole seventh day should be spent on fitness for diving, with lectures (asthma, diabetes, drugs, etc.), interactive questioning and explanations of investigations (audiometry, tympanometry, respiratory spirometry, cardiac risk assessments, radiology and indications for more specific investigations). Introduction of diving medical fitness examinations earlier in the course is totally illogical, although it is frequently scheduled there. The students are not *au fait* with the real problems the medical must assess – and these must be covered before confronting fitness assessments. The latter relies entirely on a foundation of fatality and morbidity statistics

The eighth day could cover large topics related to diving medicine, depending on the orientation of the organisation. These could include hyperbaric medicine, submarine medicine, aviation medicine, etc. As these are self-enclosed topics, they could be included almost anywhere in the second week, depending on availability of the resources.

The ninth day deals with a miscellaneous group of problems, some quite complex and extending into contentious areas. Vertigo and disorientation, the long-term effects of diving, technical diving and indigenous divers are all covered in the morning session. The afternoon is spent discussing case histories supplied by the lecturers, and engaging the class in diagnosis and treatment decision making. It is important to include non-diving problems that can present during diving activities.

A written examination is best performed on the tenth or last day. A multiple-choice questionnaire with machine marking is preferable, as this can be performed rapidly, with the remainder of the morning devoted to discussion of the questionnaire results (essential as a teaching procedure). The afternoon is spent on the verbal critique, a written critique if required by credit-supplying authorities, and the issuing of certificates of attendance and/or success.

Interactive techniques

There are various interactive techniques used by different lecturers, but there are also the more formal ones, such as student presentations and evolving case histories. These are usually performed for half an hour at the start of each day.

The student presentations are used to reinforce basic knowledge and are thus more effective during the first week. They include laws of physics, effects and treatment of barotrauma, decompression calculations, etc. If, at the end of each day, students are given a list of five or so basic questions, and are informed that they will be chosen at random to give a presentation of less than five minutes without using notes or lecture aids, to their peers on the following morning, they are inspired to at least learn this essential and non-controversial material. I inform them that these presentations will be graded and the results integrated into their final exam mark.

The use of evolving case histories for general discussion, is obvious in the second week. These are both interesting and instructive, and introduce a practical element into the proceedings. They are of little value in the first week, and are often counter-productive, as the students do not have the background to analyse and comprehend the complexity of the cases.

Literature

There are various attitudes to the question of literature. Two of the best courses I attend (the Diving Medical Centre DDME and the annual Malaysian Navy course) send a simple, easily readable text to the students a couple of weeks before the course, for their perusal. It is also useful during the course, to review the basic factual material. *Diving medicine for scuba divers* is ideal for this, even though it was originally designed for divers and dive instructors.[1] It is also of value for students whose first language is not English, besides being inexpensive.

Another two very impressive courses (the South African and Australian Navy courses) supply a comprehensive text for the duration of the course. *Diving and subaquatic medicine* is used as an authoritative and clinically orientated text, but is hardly bedtime reading.[2] A relatively inexpensive paperback version is now available.

Whichever of the texts are used, a specific medical fitness booklet is recommended. We tend to use Parker's *The sports diving medical*, even though it is not comprehensive and certainly not applicable to occupational divers.[3]

Often copious literature is supplied to support or extend the lectures. This includes Australian Standards medical forms, other official regulations, and handouts to be copied and used by diving physicians for diver education.

Some supply large manuals, often collected from previous years' lecturers. This is not as valuable as it may at first seem. The lectures have often been changed, with different lecturers and different content. The chapters vary with the writing capability of the various lecturers, and repetition is

annoying. There is often conflicting advice, and rarely is there an adequate index supplied.

What is of great value to the students is a selection of the major slides (about 10) from the PowerPoint presentation of each lecture. Do not deluge the students with the total number, sometimes up to a hundred a lecture, as this is overload.

Diving and recompression-chamber exposure

It is my belief that a diving medical course that does not result in students experiencing both diving and the compression chamber is dull and boring. These guys and gals need something to boast about in their dinner party conversations, so that others are enticed to apply.

Of course, medical examinations must be performed, not only to illustrate fitness to dive, but also to teach about medicals. Most students can be made fit to swim underwater with scuba, attached to a buddy (instructor), at least in a shallow swimming pool. Experience with ear equalisation in a chamber is invaluable, and even exposure to inert gas narcosis may be possible.

The more exciting diving medical courses will have available diving experiences conducted by professional dive instructors during the weekend, in the middle of the course. This should not be a required or integral part of the course, or be commercially related to it.

Investigations

Practical experience of performing an audiogram on a colleague, and spirometry and tympanometry on themselves, will produce results that will have much more meaning and encourage interpretation during the relevant lectures, when these investigations are subsequently described. The first time I introduced this 'testing' apparatus to the lecture room, with a paramedic to illustrate and correct the technique, I was amazed at its ability to promote interest and understanding. Much more than a passive PowerPoint presentation.

Examinations

These are often copied from previous exams, dealing with questions not covered in the current course! This is one of the reasons for using a full-time diving doctor as observer and chaperone at the course. This will ensure that all the questions and answers are covered during the course.

Dr Bob Thomas, in his three-day Designated Diving Medical Examiners course, has taught me how to approach examinations constructively. The exams are used as a teaching opportunity, engaging all the full-time lecturers. They are essentially discussions about the most important subjects covered by the course (and reflected by the exam questions). The lecturers' varying attitudes demonstrate the complexity of the subject, and the students' attempts to rationalise their incorrect answers allows us to focus on the failure of our instruction. These omissions can then be corrected.

Qualifications

Bestowing of qualifications on attendees is beyond the scope of this discussion. Many delegates receive elaborate certificates that decorate consulting rooms and academic offices. Some declare successful completion of the course, others merely attendance. Some certificates confer the designation of Diving Medical Examiner (DDME), approved by the Australian Standards Association (and SPUMS) for either professional or recreational divers. Others imply an expertise in assessing and treating diving accidents.

The DDME qualification needs to be addressed, but not here and now. The relative value of the three-day specialised courses and the two-week more generalised diving medical courses may result in surprising outcomes in producing competent DDMEs. The surveys carried out in Queensland by Mark Marshall (364 respondents in the 1970s), as a master's thesis for the University of Queensland (reference not available), and Simpson and Roones' more recent questionnaire (52 respondents) are relevant.[4] In both there are claims that are, in my opinion, unsupportable, but both unquestionably demonstrate the widespread inadequacies of diving medical physician training and the implementation of accepted medical standards. The unpalatable conclusion is that the quality or focus of our instruction is lacking.

Critiques

This is where we are taught what we really need to know. I find that during this time, when each student is required to say his piece and to criticise, not praise, it is best that the lecturers (all the full-time ones at least) should shut up and listen. Attempted rationalisations and even explanations are irrelevant. How the student experiences the course, and his wishes for improvement, are subjects on which he is the undoubted expert, not the lecturer.

Conclusion

Of course, I do not expect most established diving medical courses to actually change, based on the above observations. That is why they are called 'established'. What I do hope for is that the younger, new diving medical physicians – the ones who have sought my views privately – will have the opportunity to peruse the suggestions in print, as opposed to the barely remembered snippets offered over a barbecue or bar, and compare them to the format of the courses currently available.

This paper is not the final answer. It is a work in progress, and if some, students or lecturers, dissent from my observations, I welcome their objections and suggestions.

References

1 Edmonds C, McKenzie B, Thomas R. *Diving medicine for scuba divers,* 2nd ed. Melbourne: JL Publications; 1996.
2 Edmonds C, Lowry C, Pennefather J, Walker R. *Diving and subaquatic medicine,* 4th ed. Oxford: Arnold; 2002.
3 Parker J. *The sports diving medical*, 2nd ed. Melbourne: JL Publications; 2002.
4 Simpson G, Roones D. Scuba diving medical examinations in practice: a postal survey. *Med J Aust*. 1999; 171: 595-8.

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Editor's comments

A comparison between the 2007 Royal Australian Navy, and Royal Adelaide Hospital two-week diving medicine course programmes and Dr Edmonds' recommendations is provided in Table 1. The overall times are for structured components of each course and do not allow for travel time between venues, etc., nor for the informal interaction that occurs during refreshment/meal breaks and at the end of the day.

Not all components could be categorised in the same way for each course, and it is likely that some components that appear to be missing from one or other are actually embedded within other headings. This is probably particularly the case with the RAH course where a much greater time is spent on the broad category of 'diving equipment and techniques' that encompasses a number of items otherwise not specified in their programme. Also, the physiology component of the RAH and Edmonds programmes appears light, but is likely to be incorporated into most of the specific topics.

There is a similarity between the three with emphasis varying depending on the interests of individual lecturers. All three devote about 12–15% of the course time to interactive sessions, largely based on clinical issues such as evolving clinical problems and 'fitness-to-dive' scenarios. These interactive components are undoubtedly an important part of the learning process in pressure-cooker programmes like this. In looking at the actual timetables (not shown here), a logical sequence such as Dr Edmonds advocates is broadly followed at both venues.

The timetables for the most recent RAN and RAH courses and the hypothetical by Dr Edmonds are available from the journal office on request (<spumsj@cdhb.govt.nz>).

Table 1
A comparison of the components and the time (hours) devoted to each of the Royal Australian Navy (RAN) and Royal Adelaide Hospital (RAH) two-week diving medicine courses and those suggested by Edmonds

Component	RAN hrs	RAH hrs	Edmonds hrs
History of diving	0.75	1.5	1
Physics	1.5	1	2
Physiology	1	2.5	0.5
Breath-hold diving	1		1
Dive accidents and fatalities	1.5	2.5	3
Diving equipment & techniques	2.75	9	3.5
Technical diving		0.75	1
Diving environment			1.5
ENT problems and diving	1	1	1
Barotrauma: lung, other	1	1	3
Decompression theory/ tables/computers	3.75	1.5	3
DCI: pathophysiology	1	1	1
DCI: clinical presentation	1	1	1
DCI: treatment incl. tables	2	2	1
DCI: treatment in remote areas	1	1	
Oxygen: delivery systems	1		
Gas toxicities & contamination	1.75	1	3
Carbon monoxide poisoning	0.5	1	
Inert gas narcosis & HPNS		1.25	0.5
Hypothermia & hyperthermia	1	0.75	1
Resuscitation & CPR update	3	1	1
SWAS/near drowning/ sudden death	1	0.75	2.5
Dangerous marine animals	3	1	2.5
Long-term effects of diving	1	1	1
Women and diving			1
Indigenous diving			1
Fitness to dive: general principles	2.5	1	0.5
Fitness to dive: all aspects	3	1	4
Fitness to dive: medical forms	0.75		1.5
Recompression chambers	1	1.75	1
RCC experience	1.25	3.25	3
Diving medicine organisations	0.25		
Wet acquaint	3.5		
Submarine medicine & escape	3		1
Submarine familiarisation	1.5		
Hyperbaric medicine	2.25	5	2
Hyperbaric nursing		1	
Interactive-ECPs, fitness, etc	7.5	8.75	9.75
Examination: pre-course	0.75		
Examination: final	2	2	1.5
Examination: reviews/critiques	1.25	1.5	2
Introduction/course critique	1.25	1	1.5
Total course time	63.25	59.75	64.75

Dive to walk

Most of us dive for pleasure, to experience the underwater world, to swim with the fish or to explore a wreck, but one British diver has a unique reason to venture underwater, he dives so that he can walk. To prove his point, he has just completed his 300th dive with Red Sea Waterworld in Taba Heights.

Mark Chenoweth was born with spina bifida. He was able to lead a relatively normal life until 1996, when he lost the feeling in both legs and was forced to start using a wheelchair. Mark's first experience of diving came while on holiday in Rhodes with his wife. He enjoyed a try-dive in the hotel pool so much that he set out to obtain a medical in order to do an open-water dive.

Unable to find a doctor willing to sign, he returned to England, where his search proved no more fruitful. Six doctors, diving and drug specialists, all gave reasons why Mark could not dive, one even telling him that it would be the last thing he ever did! Mark refused to give up. On his next holiday, to Minorca, he saw a doctor but this time 'forgot' to mention a few things (six to be precise – not a step normally to be recommended) and received the certificate he needed!

After two days of pool and theory training, the first two open-water dives went well, any anticipated problems easily overcome. Then, on the Saturday, a slight hitch with weightbelt replacement required the third dive to be repeated. The re-run third dive went well, and back on the dive boat, Mark realised that his legs felt "different". Amazingly, especially as he was on a boat, he stood up! Every night Mark's father-in-law had brought his wheelchair to take him from the transfer bus into the hotel. But that night he was amazed to see Mark stroll past him, straight into the arms of his stunned wife.

Since this momentous day, Mark has learnt more about his phenomenal recovery. It is only a temporary cure, and dives to 16 metres' sea water (msw) depth have no effect, but at 17 msw, the atmospheric pressure seems to be enough to alter his spinal cord. The deeper he dives, the longer he regains the use of his legs, but the gases are not the trigger, as they return to normal in a matter of hours. A 20 msw dive will see him return to his chair within days, two weeks at most, but a deep dive will keep him mobile for nearly six months!

Mark has gone on to complete many PADI specialities, including Deep Diver, Rescue Diver and Master Scuba Diver. A hyperbaric medical team has carried out chamber dives with him, and because of the high oxygen levels these gave the best results, leading Mark to complete his nitrox qualification.

The team suggested that he might have a bubble on his spinal cord that was reduced under pressure, but after a CAT scan ruled this out no other theories emerged and the reason remains a mystery. Mark reckons himself extremely lucky to have found a way to defy medical science, especially as the answer is diving, which he describes as "the most wonderful hobby anyone could ever imagine".

At recreational diving limits, the lack of sensation in parts of Mark's body means that if he were to suffer a bend he would not necessarily realise it. He reduces this risk by diving well within limits and ascending at a rate far slower than the recommended 18 metres per minute. Mark Chenoweth is looking forward to the next 300 dives, and hopes to complete some technical training in the near future.

Reprinted with minor editing from a news item in *Diver*. August 2007. p. 22.

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates

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

1 The candidate must be medically qualified, and be a 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 Hyperbaric Medicine Unit.
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, and in a standard format, for approval by the Academic Board 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.

Additional information

The candidate must contact the Education Officer to advise of their intended candidacy, seek approval of their courses in Diving and Hyperbaric Medicine and training time in the intended Hyperbaric Medicine Unit, discuss the proposed subject matter of their research, and obtain instructions before submitting any written material or commencing a 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.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of:
Dr Fiona Sharp, Education Officer, Professor Des Gorman and Dr Chris Acott.

All enquiries should be addressed to the Education Officer:
Dr Fiona Sharp,
249c Nicholson Road
Shenton Park, WA 6008
Australia
E-mail: *<sharpief@doctors.org.uk>*

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

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Approved extracts of minutes of the SPUMS Executive Committee Meeting, held on 19 April 2007 at Oceans Resort, Tutukaka, New Zealand

Opened: 1945 hr

Present: Drs C Acott (President), G Hawkins (Acting Secretary), F Sharp (Education Officer), D Smart (ANZHMG Representative), M Davis (Editor)

Apologies: Drs S Sharkey (Secretary), G Williams (Treasurer), C Lee (Committee Member), V Haller (Public Officer), D Vote (Committee Member), M Walker, R Walker (Immediate Past-President)

1 Minutes of previous meeting
Minutes accepted for AGM 2006. Proposed by F Sharp, seconded by C Acott.

2 Matters arising from previous minutes
2.1 Availability of R Walker for SPUMS representation at the EUBS attendance to be confirmed.
2.2 G Hawkins attended the Asian Hyperbaric and Diving Medical Association meeting in Bali, Indonesia and discussed the use of the SPUMS Journal *Diving and Hyperbaric Medicine* as the journal of AHDMA. This was accepted by the members of AHDMA and the President

of AHDMA will forward a letter to the President of SPUMS to initiate formal discussions. At the meeting was Professor E Sanchez (President of Latin America Chapter of UHMS) who stated that the Latin America Chapter of UHMS would also like to initiate talks regarding the use of *Diving and Hyperbaric Medicine* as its official journal.

3 ASM 2007

Dr Michael Davis suggested that the members of Dive Tutukaka be offered an invitation to the 2007 ASM dinner at SPUMS' expense. It would not affect the overall profitable outcome of the meeting significantly. The proposal was accepted by the committee members.

4 ASM 2008

4.1 The 2008 ASM is to be held at Liamo Resort, Kimbe, PNG.

4.2 Payment of guest speakers was discussed. The Committee agreed to a proposal that invited guest speakers would be given AU$ 5,000.00 per speaker in lieu of airfare as this provides them greater flexibility in deciding how to travel and arrange their diving and accommodation.

4.3 The 2008 ASM will have a slightly different format with lectures in the morning and diving in the afternoon to ensure optimal conditions.

5 ASM 2009

5.1 Three locations had been proposed by Dr D Smart. Fiji was accepted by the Committee as the best option.

5.2 Due to issues regarding political tensions in Fiji in the recent past, a second location is to be investigated by the ASM Convenor in conjunction with the dive travel company.

5.3 It was also proposed that every ASM should be put out to tender process with two companies in the future and this was thought to be acceptable. For further consideration by the Committee.

6 ASM 2010

It was suggested by G Hawkins that the 2010 ASM could be a joint ASM with the Asian Hyperbaric and Diving Medical Association in South East Asia. He will investigate further and report back to the Committee at the next meeting.

7 Editor's report

7.1 Dr Davis will continue to investigate the linkages to be developed between SPUMS and EUBS with regard to the Journal and he has a preliminary set of criteria for EUBS to consider at their ASM.

7.2 Dr Davis has requested a review of the documentation for the role of Editor of the Journal and he has distributed proposed changes. This is for consideration by all the committee members.

8 Education report

8.1 Main report as presented at AGM.

8.2 Dr M Davis has resigned from the Education Committee stating a potential conflict of interest with respect to his role in the Auckland Graduate Diploma Course. This was reluctantly accepted.

8.3 Dr F Sharp will clarify the role of Professor Des Gorman and poll people for any interest in becoming members of the Education Committee.

9 Other business

Nil

Closed: 2044 hr

Dates and venues of the SPUMS Annual Scientific Meetings

1972	Australia, Heron Island
1973	AGM only, no ASM
1974	Fiji
1975	Vanuatu
1976	Fiji
1977	Truk
1978	Fiji
1979	Vanuatu
1980	Malaysia, Tioman
1981	Papua New Guinea (PNG), Madang
1982	Philippines
1983	Fiji
1984	Thailand, Phuket
1985	The Maldives
1986	French Polynesia, Morea
1987	Solomon Islands
1988	Fiji
1989	Vanuatu
1990	Palau
1991	The Maldives
1992	Australia, Port Douglas
1993	Palau
1994	PNG, Rabual
1995	Fiji
1996	The Maldives
1997	New Zealand
1998	Palau
1999	Layang Layang
2000	Fiji
2001	PNG, Madang
2002	Vanuatu
2003	Palau
2004	Noumea
2005	The Maldives
2006	Fiji
2007	New Zealand

Greetings from the new SPUMS Webmaster

Hello to all members!

I just wanted to inform you of the developments to the Website that may be of some interest to SPUMS members.

First, I have taken over the duties of Webmaster from Dr Robyn Walker, who has made the Website a great source of information, particularly for members who can now use it to pay for both membership and next year's ASM. Yes, that's right, you can now pay for your ASM online and this has made a significant improvement to the organisational running of the Society for now and the future.

Second, I have added to the Website some papers from the SPUMS Journal on diving (and general) travel by Dr Trish Batchelor. They involve things like traveller's diarrhoea, prophylaxis for malaria and other features such as constructing a travel medical kit. All these papers are in pdf format and can be seen in the Travel section on the main menu. Thanks must go to both Dr Batchelor and Dr Mike Davis for allowing the publication of these articles.

Third, we will start to establish a webmail send-out capacity from the site so that any new events or changes in the Society and the Website can be sent to people judiciously.

Finally, we will be endeavouring to place our journal, *Diving and Hyperbaric Medicine*, online for members in full-format capacity. This is an evolving project that will allow those with member access the ability to search and read through all issues of the Journal at their leisure. In conjunction with this we have decided to make the Journal available to the general public via the Rubicon Foundation, which has a mandate to make diving and hyperbaric papers available for people to access around the world. Several 'classic' papers have been placed online at <http://rubicon-foundation.org/>, which has over 4,000 articles and papers already available. We will endeavour to make papers from the SPUMS Journal available up until the last three years (so that members retain privileged access to the most recent papers).

The idea behind this is to make the work that we have done in this part of the world more accessible to people in the northern hemisphere as well as lifting the profile of the whole organisation on a worldwide basis.

So there are several exciting things happening and I would like to thank Dr Robyn Walker and Steve Goble for their efforts in setting up the SPUMS Website. I hope that I can continue to build it from the strong base that they have developed.

Glen Hawkins (Webmaster – In training)

UHMS fitness-to-dive course now certified in Europe

The Diving Medical Advisory Committee, the European Diving Technology Committee, the European College of Baromedicine and the European Committee for Hyperbaric Medicine have all recently approved the Undersea and Hyperbaric Medical Society (UHMS) Medical Assessment for Fitness to Dive Course. This brings an important academic component to the course for European post-graduate education in underwater and hyperbaric medicine. The Immediate Past-Executive Director of UHMS asks "*What's next for our course*?" – the answer: Australia and the South Pacific. After reading an e-mail exchange between Dr Michael Bennett and Dr Mario Lanza concerning an American physician performing physical examinations for Australian divers, the UHMS will be following advice from Dr Bennett and the Chair of its Diving Committee, Dr Simon Mitchell, as the UHMS course is submitted for consideration by SPUMS to be included in a list that would qualify a physician who attends this course to perform Australian diving medicals according to the Australian recreational and occupational diving standards.

Auckland University Post-graduate Diploma in Medical Science – Diving and Hyperbaric Medicine

The ANZCA Council has agreed that successful completion of the "Medicine 719" component of the University of Auckland Post-graduate Diploma in Medical Science – Diving and Hyperbaric Medicine, will be accepted as an alternative to the ANZCA DHM Formal Project.

Additionally, successful completion of the "Medicine 714 and 715" components of the University of Auckland Post-graduate Diploma in Medical Science – Diving and Hyperbaric Medicine, will be accepted as an alternative to the formal course requirements of the ANZCA Certificate in Diving and Hyperbaric Medicine.

It was further agreed that up to six months of the training towards the University of Auckland Post-graduate Diploma in Medical Science – Diving and Hyperbaric Medicine, may be credited towards the six-month practical experience component of the ANZCA Certificate in Diving and Hyperbaric Medicine which is not required to be undertaken in an accredited unit.

L F Wilson
Chair, Hospital Accreditation Committee

Editor's note: Sadly the Auckland programme in diving and hyperbaric medicine has been abandoned by the University mainly on financial grounds. Therefore, the above applies only to existing graduates and current students. As the programme director, the writer is extremely disappointed that this attempt to link diving and hyperbaric medicine to an academic institution in Australasia at a more basic level than that of the ANZCA Certificate has failed.

The Australian and New Zealand College of Anaesthetists Special Interest Group in Diving and Hyperbaric Medicine (SIG-DHM)

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 The "Medicine 714 and 715" components of the University of Auckland Post-graduate Diploma in Medical Science – 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. Successful completion of the "Medicine 719" component of the University of Auckland Post-graduate Diploma in Medical Science – Diving and Hyperbaric Medicine will be accepted as an alternative to the ANZCA DHM Formal Project.

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

Report from the ANZCA SIG-DHM

There was a highly successful ANZCA SIG-DHM scientific meeting held at the ANZCA Annual Scientific Meeting (ASM) in Melbourne on 28 May 2007. A cutting-edge scientific programme organised by Dr Mike Bennett included international and local experts, and attracted an audience of over 100 enthusiastic listeners. Simon Mitchell kicked off the session with an update on the treatment of decompression illness in the 21st century. Professor Bruce Speiss then gave a fascinating presentation about the role of perfluorocarbon emulsions in modern medicine, with applications ranging from blood replacement to the treatment of decompression illness. Glen Hawkins presented an update on the chronic hypoxic wound database, and David Smart closed with a presentation of the current state of the evidence regarding the role of transcutaneous oximetry as a tool to predict outcomes in hypoxic wounds.

There were many questions from the floor, with a high level of interest, especially in the information presented by Professor Speiss. Bruce Speiss 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 his major research interests are in the areas of blood substitutes, the pathophysiology of coagulation and inflammatory abnormalities in shock. Professor Speiss also conducts research into decompression sickness and submarine escape with the United States Navy. He was a fascinating speaker, and is keen to return for another session, perhaps in 2009.

The next SIG-DHM scientific meeting will be held in Sydney during the May 2008 ANZCA ASM, and all interested practitioners are welcome to attend.

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

Articles reprinted from other sources

Endothelial microparticles in vascular disease and as a potential marker of decompression illness

Leigh A Madden and Gerard Laden

Key words
Reprinted from, decompression illness, endothelium, bubbles, microparticles, review article

Abstract

Micro-gas emboli are known to be present within the venous circulation following routine hyperbaric exposure. Emboli can be identified/quantified using Doppler and 2D ultrasound, thus functioning as an index of decompression stress. In relation to decompression illness this technique has low sensitivity and specificity. A biological marker of decompression stress would prove a useful tool. Such a marker could be used to gauge the efficacy of prophylaxis. Endothelial cells are known to shed microparticles during activation and apoptosis. Since microparticles in general express the antigens of the cells from which they were derived, the origin of them can be determined and their phenotype can lead to an insight as to the state of the parental tissue. Microparticles have been studied in many vascular diseases, and reviewed here, and we hypothesise that micro-gas emboli have a capacity to damage the endothelium and thus cause a change in the circulating microparticle population.

Decompression illness

When humans breathing air sojourn to high- or low-pressure environments they are exposed to the risk of acute decompression illness (DCI, the bends). The population at risk is varied and growing, e.g., sport scuba divers, submarine escapees and space explorers. The number of sports diving related cases of DCI has steadily risen in both North America and the UK over the last 10 years. Advancement in techniques for both prophylaxis and treatment of DCI is warranted.

Decompression illness is the result of gas phase forming in tissue and blood, following pressure changes, i.e., return to atmospheric pressure (surfacing) or a drop in pressure as in astronaut extravehicular activity. Modern technology has allowed the detection of venous gas following routine (believed) safe dive profiles. Contrary to the thinking 100 years ago, venous gas does not routinely lead to overt DCI. Venous bubbles often referred to as 'silent' appear to be filtered by the lungs and fail to reach the arterial circulation where their presence is significantly more problematic.

Historically decompression procedures (tables) have been validated following mathematical calculation, animal modelling and human trials. More recently, detection of venous gas, accepted as an index of decompression stress has been used to help validate decompression procedures, as has probabilistic modelling.

Vascular gas can be identified using Doppler and 2D imaging; however, these techniques have limitations. For example they tend to use short time weighted sampling (typically 5 minutes) over increasingly extended periods post exposure; they poorly quantify vascular gas. Their sensitivity and specificity in relation to DCI is poorly defined. There are numerous reports of divers with no or low bubble scores becoming ill and asymptomatic divers with high bubble scores.

With the now wide acknowledgement that DCI often attacks the central nervous system, human trials with DCI as one possible endpoint have become ethically and practically problematic. Finally the postulate that venous gas bubbles are 'silent' or biologically inactive regardless of their quantity is naïve. Accordingly a method of biologically describing a subclincial dose response to the effect of a decompression procedure would be helpful in all aspects of decompression modelling. A potential biological marker relating to the stress of a dive is the endothelial microparticle. Gas bubbles released into the circulation will interact with the surface of the vascular endothelium and may give rise to a measurable response, linked to the state of the endothelium.

Endothelial function

Vascular endothelium plays a role in the mechanisms of haemostasis, being involved with the vessel itself, platelet interaction and the functions of the plasma. Endothelial function also is implicitly involved in inflammation and repair of damaged tissue. Disruption of the endothelium, either physically or characteristically, due to a disease state such as post-ischaemic reperfusion, inflammation, hypertension and others, results in a potentially prothrombotic endothelial function. In this state the endothelium expresses von Willebrand factor (vWF), P-selectin (CD62P), ICAM-1 (CD54), VCAM-1 (CD106), IL-8 and promotes the activation and adhesion of platelets, via PECAM-1 (CD31).

Thrombin formation is also observed, along with expression of tissue factor and fibrin deposition. Vascular permeability increases and the production of cytokines, chemokines, and growth factors and the expression of cellular adhesion molecules are upregulated. These responses are usually part of endothelial function rather than dysfunction and are a prerequisite for tissue repair and wound healing subsequent to the disruption.

Endothelial dysfunction usually results from various disease states and is characterised by a reduction in dilatory capacity and decreased NO capacity, as a result of increased oxygen radical production, which reacts with NO to form peroxynitrate. NO and prostacycline (PGI_2) are vasodilators, involved in maintaining an antithrombotic state by preventing the formation of platelet aggregates. NO controls endothelium-dependent vasodilation, leucocyte adhesion, platelet aggregation, expression of adhesion molecules, synthesis of endothelin and inhibition of vascular growth and inflammation.[1] NO is produced in endothelium by nitric oxide synthase (eNOS) and is inactivated by oxygen radicals. Production of NO is dependent upon the presence of cofactors (such as tetrahydrobiopterin) and the availability of the substrate L-arginine. Oxygen radicals can be produced by eNOS under conditions of tetrahydrobiopterin or L-arginine deficiency and elevated concentrations of LDL-cholesterol. NADH/NADPH oxidase also produces oxygen radicals when stimulated by TNFα, and is located in the arterial wall, where extracellular superoxide dismutase acts to remove such radicals.

Endothelial dysfunction can be measured and is implicated in arteriosclerosis,[2] in which oxygen radical formation is enhanced.

Oxidative stress results in endothelium-mediated vessel dilation and a subsequent increase in cell turnover and death. Endothelial dysfunction is normally reversible.[3]

Cytokine activation of endothelial cells results in increased ability to bind circulating leucocytes, by as much as 400%.[4] This increase is due to new or increased expression of adhesion molecules E-selectin (CD62E), ICAM-1 and VCAM-1. Under normal physiological conditions endothelial cells bind leucocytes only briefly, but once activated low affinity interactions are formed, which are then disrupted by shear forces, to be reformed once again, causing a rolling of the leucocytes on the cell surface.

This in turn, with the involvement of chemokines, causes firm attachment of leucocytes to the endothelium, where they crawl to the endothelial cellular junctions and extravasate into the tissue space causing inflammation. Adhesion molecule expression follows a defined path; E-Selectin expression occurs early in the process of inflammation (around 2–4 hours after activation) and VCAM-1 expression later (12–24 hr). The pattern of expression can be modified by various chemokines, such as INFγ and IL-4.[5,6] E-Selectin, ICAM-1 and VCAM-1 possess DNA sequences that bind transcription factors NfκB and activator protein-1 and these are essential for the TNFα-mediated activation of endothelial cells,[7] showing that these transcription factors are able to modulate the adhesion molecule expression.

Endothelial microparticles and markers of endothelial perturbation

Established markers of endothelial cell (EC) damage/activation are traditionally soluble, and are measured from circulating blood. Such markers include ICAM-1 and VCAM-1 amongst many others. However, measurement of these markers may well include membrane-bound forms, as they can be removed by filtration.[8] This has led to a wide variation in measurements determined by ELISA techniques. These membrane-bound markers are constituents of microparticles (MP). Endothelium-derived MP (EMP) and the markers they express are indicative of the state of the endothelium, i.e., activated or apoptotic. They were first described as being released from cultured human umbilical vein endothelial cells (HUVECS) upon stimulation with complement,[9] and have been studied as an *in vitro* model for release of MP during activation or apoptosis.[10] MP released from HUVECS are phenotypically distinct and have been proposed to be a useful marker for endothelial injury,[11] and are presumed to be procoagulant due to the expression of anionic phospholipids.

Microparticles in humans

MP are released by unstimulated endothelium in healthy subjects, and so a basal concentration exists in the circulation. This suggests that endothelial vesiculation occurs under normal physiological conditions.[12] They have been postulated to maintain a balance between cell activation, proliferation and death and be involved in the maintenance of homeostasis.[13] Plasma membrane vesiculation is part of remodelling and there is evidence that MP can illicit a response in remote cells via their expressed antigens.[13] An increase above the basal levels of MP may lead to pathologic disorders; however, basal levels are not detrimental. As MP numbers vary according to the method used no comparable intra-study is available, although research to date has been compared to levels found in healthy controls under the same detection conditions.

Microparticles in disease

MP in the blood circulation have been described in many disease states as either increased in their numbers or being of altered composition, reviewed by Horstmann et al.[8] They were first identified from platelets by Wolf,[14] and have been shown to be released by many different cells in response to activation or cell death, recently reviewed by Nieuwland and colleagues in relation to their role in cardiovascular disease.[15] Release of MP from activated cells is time and calcium dependent,[16] whereas those released from cells

undergoing apoptosis are formed by membrane blebbing, and are positive for annexin V binding. In both cases MP carry the proteins specific to the parent cell from which they were derived, thus allowing identification of relative MP populations. This is particularly useful when the parent cell may have become activated and expressed proteins specific to the activated state.

Increased numbers of circulating MP have been studied in acute coronary syndromes,[17] multiple sclerosis,[18] arteriosclerosis,[15] diabetes,[19,20] hypertension,[21,22] pre-eclamplsia,[23,24] and sepsis,[25] amongst others. MP have been shown to be either elevated or of an altered composition in patients with cardiovascular disease that show impaired endothelial function.[15] MP released from endothelial cells may act as a marker for vessel wall injury.[10]

Microparticles in thrombocytopenic purpura

EMP, released from perturbed endothelium, were elevated in thrombotic thrombocytopenic purpura (TTP),[11] a disease where platelet activation is established. Plasma from TTP patients was found to induce a three-fold increase in ICAM-1 and a 13-fold increase in VCAM-1 expression on *in vitro* culture of renal microvascular endothelial cells (MVECS). EMP were elevated in patients with TTP, but not when the disease was in remission and therefore were stated to have the potential to be a useful marker of endothelial injury. CD62E and CD54 expression on EMP from TTP patients was found to be increased significantly[26] and of CD62E-positive EMP, 55% displayed expression on vWF. The authors concluded that the EMP were released from activated endothelium in TTP patients. EMP counts returned to normal upon remission. EMP were analysed from cultured brain and MVECS. CD31 and CD42b were used to identify MP of endothelial origin. EMP were found to be pro-coagulant when the cells were stimulated with TNFα (activation) or mitomycin C (apoptosis).[11]

Research into EMP markers by the same group showed that they possess different proteins, which were determined by whether the MP were formed by activation or apoptosis pathways in the endothelial cell of origin.[27] The expression of the inducible markers CD54, CD62E and CD106 was found to be increased in MP from activated cells, compared with those from apoptotic cells and control samples. Annexin V binding to MP was found to be increased in both activation and apoptosis.

Microparticles in coronary disease

EMP were found able to bind platelets *in vitro*, forming aggregates (EMP-P) with a potential involvement in thrombus formation.[28] MP were isolated from HUVECS by ultracentrifugation and incubated with isolated platelets before being labelled with CD105 and CD41a. Flow cytometry confirmed aggregates expressing both the endothelial (CD105) and platelet (CD41a) markers had been formed. Similar aggregates could be isolated from healthy subjects and almost all of those which were CD105 positive were also found to express CD31 and two markers of endothelial activation (MCP-1 and CD62E). Patients with stable coronary disease were found to have a significantly higher concentration of EMP-P (16.7 per μL whole blood) than healthy controls (7.1 per μL). A significant decrease in EMP-P concentration was observed during acute myocardial infarction, which was hypothesised to be due to involvement of these aggregates in thrombus formation in the infarct-related vessel. Levels of circulating EMP-P returned to pre-event concentration at 48 hr. A previous study observed an increase in EMP within blood of subjects with acute myocardial infarction;[17] however, the MP were higher measured days after onset and when compared to healthy controls.

It has also been demonstrated that MP isolated from patients with myocardial infarction have the potential to cause further endothelial dysfunction.[29] Rat aortic rings were incubated with MP isolated by ultracentrifugation from patients with myocardial infarction. It was concluded that these MP caused a high degree of endothelial dysfunction in healthy vessels by affecting the NO transduction pathway. The MP significantly decreased relaxations in response to acetylcholine in the aortic rings, and this observation was eliminated upon endothelium removal or the addition of a NO synthase inhibitor. The actual MP, if indeed there was a particular type responsible, were not analysed as to their cellular origin.

Microparticles in multiple sclerosis

The presence of CD31 on endothelium is a prerequisite for extravasation of leucocytes,[30] and was found to be increased in the serum of MS patients where brain gadolinium-enhancing lesions were present.[31] Circulating EMP were analysed for CD31 and CD51 expression in MS patients and were found to be elevated in disease exacerbation but not when the disease was in remission. The amounts of EMP were found to be 2.45, 0.58 and 0.86 x 10^6 per mL in MS exacerbation, remission and normal controls, respectively. The median value for all collected EMP was surpassed by 93% of patients with MS in exacerbation and 90% were below the median when in remission, suggesting strong evidence for a role of endothelial damage in the disease process.

Microparticles in sickle cell anaemia

Circulating EC have been analysed in sickle cell anaemia, a disease in which the vascular endothelium has a role in pathogenesis.[32] A correlation was established between acute painful episodes and circulating EC. Also, the circulating EC were found to express CD54, CD106, CD62E+P, suggesting the endothelium is in an activated state in the illness. MP

in sickle cell disease subjects were found to be elevated in crisis and steady-state conditions, when compared to normal controls,[33] although MP were defined as less than 1 μm in size and able to bind annexin V. MP were isolated by ultracentrifugation; however, this method was previously shown to vastly underestimate the true number of platelet-derived MP within the plasma.[34] These MP were released by endothelial, monocytes, erythrocytes and platelets and a proportion were found to be tissue factor (TF) positive, but 13/21 patients had no detectable EMP expressing TF. The majority of TF expression was found to be on monocyte-derived MP (20/21). The expression of TF on MP in sickle disease may be of importance in thrombosis, potentially causing an activation of clotting pathways and the production of thrombin.[33] The authors concluded that the presence of monocyte- and endothelial-MP were a marker of parent cell activation in the disease; however, such markers of activation were not measured.

Microparticles in paroxysmal nocturnal haemoglobinuria

A further study focused upon EMP in paroxysmal nocturnal haemoglobinuria (PNH) and sickle cell disease (SCD) in comparison to healthy controls.[35] Thrombosis is the major cause of morbidity and mortality in PNH, and is always associated with EC activation and damage. PNH is clinically manifested by haemolysis, which releases free haemoglobin, toxic to EC. The number of endoglin (CD105) positive EMP was elevated in PNH (0.4 x 10^9/L plasma) and SCD (0.57 x 10^9/L) when compared to controls (0.18 x 10^9/L). A subpopulation of CD105+CD54+ EMP were also elevated in PNH (0.24 x 10^9/L) and SCD (0.25 x 10^9/L) compared to controls (0.11 x 10^9/L). This was thought to show that the vascular EC show an inflammatory phenotype. No correlation between EMP and thrombotic events in the disease states was evaluated; however, since thrombosis is a major source of mortality in PNH a link was intimated and the EMP phenotype was said to be a marker of severity of vascular disease and of diagnostic use.

Microparticles in meningococccal sepsis

Meningococccal sepsis is caused primarily by the release of endotoxin by bacteria. Plasma from patients (n = 7) with this disease were analysed for MP and compared to healthy controls (n = 5).[36] Elevated levels of various MP, with procoagulant properties (TF positive) were found, although CD62E+ defined EMP were elevated but not significantly so. EMP concentration was 61 x 10^6 per L plasma in patients, compared with 18 x 10^6/L in controls. All cell-derived MP showed large variations in the 36-hour testing period. Interestingly, TF activity was identified in a non-surviving patient, and was found to occur on monocyte-derived MP, as identified by CD14/TF dual staining. Also, thrombin generation was seen to occur when MP from this patient were incubated with normal plasma, and furthermore this generation was delayed significantly by pre-incubation with antibodies against TF or factor VII.

Microparticles in diabetes

In type I diabetes EMP levels were significantly raised (26 x 10^9/L plasma), when compared with control subjects, and patients with type II disease did not show an increase.[37] Healthy controls were found to have 14 +/-16 x 10^9 EMP per L plasma. Platelet-derived MP were also elevated in type I disease, and annexin V positive MP were elevated in type II disease. Leucocyte-derived MP were found to be elevated in both type I (38 x 10^9/L) and type II (37 x 10^9/L), when compared to controls (14 x 10^9/L). The results were thought to suggest that EMP were a marker of endothelial damage associated with diabetic nephropathy in type I diabetic patients. The authors speculated that MP in diabetes, which possess a pro-coagulant function, exacerbate cell activation and contribute further to the disease progress and therefore may be a target for therapeutic intervention.[37]

EMP, as with other soluble cytokine receptors, may act to neutralise ligands destined for their parent cells, effectively removing or at least decreasing their potential effect. Therefore EMP may have a protective function, preventing further EC activation.

In summary EMP may provide a valuable insight into the state of vascular endothelium in many disease states, some of which have been highlighted here.

EMP and decompression

We recently conducted a randomised crossover trial of divers (n = 24) subject to 2.8 atm for 78 min bottom time and decompressed using USN standard air tables. Endothelial MP were identified from blood samples taken pre-, during and up to 24 hr post-hyperbaric exposure, using flow cytometry, after labelling with the relevant fluorescent-tagged antibodies PECAM-1, CD34, CD42b, CD51, CD54, CD62E+P and VCAM-1.

Having established a coefficient of variation for mean fluorescence values of CD markers of approximately 50% (n = 46 samples), no analysis between groups was thought meaningful. Wilcoxon's signed-rank test was used for within-group analysis, with pre-dive values used as a baseline control. In the dive group significant increases were observed in some markers 5 min following decompression (CD54: P = 0.030, n = 20; CD106: P = 0.013, n = 23). After 12 hr decompression most markers studied had significantly increased (CD34: P = 0.041, n = 15; CD51: P = 0.007, n = 20; CD54: P = 0.005, n = 21; and CD106: P = 0.001, n = 24); however, no significant change was observed in CD62E+P expression.

The significant increase in endothelial-specific markers within the circulating MP population is indicative of endothelial damage. There is evidence of possible EC

activation following hyperbaric exposure, as indicated by the increase in CD106. Further investigations are necessary to correlate changes in MP population with diving stress.

References

1 Hornig B, Drexler H. Reversal of endothelial dysfunction in humans. *Coron Artery Dis*. 2001; 12: 463-73.

2 Galle J, Quaschning T, Seibold S, Wanner C. Endothelial dysfunction and inflammation: What is the link? *Kidney Int*. 2003; 63: S45-9.

3 Buckley CD, Rainger GE, Nash GB, Raza K. Endothelial cells, fibroblasts and vasculitis. *Rheumatology*. 2005; 44: 860-3.

4 Bevilacqua MP, Pober JS, Wheeler ME, Cotran RS, Gimbrone MA. Interleukin-1 activation of vascular endothelium – effects on procoagulant activity and leukocyte adhesion. *Am J Pathol*. 1985; 121: 394-403.

5 Briscoe DM, Cotran RS, Pober JS. Effects of tumor-necrosis-factor, lipopolysaccharide, and Il-4 on the expression of vascular cell-adhesion molecule-1 in vivo – correlation with Cd3+ T-Cell infiltration. *J Immunol*. 1992; 149: 2954-60.

6 Doukas J, Pober JS. Ifn-gamma enhances endothelial activation induced by tumor-necrosis-factor but not Il-1. *J Immunol*. 1990; 145: 1727-33.

7 Collins T, Read MA, Neish AS, Whitley MZ, Thanos D, Maniatis T. Transcriptional regulation of endothelial-cell adhesion molecules - Nf-Kappa-B and cytokine-inducible enhancers. *FASEB J*. 1995; 9: 899-909.

8 Horstman LL, Jy W, Jimenez JJ, Ahn YS. Endothelial microparticles as markers of endothelial dysfunction. *Front Biosci*. 2004; 9: 1118-35.

9 Bernal-Mizrachi L, Jy W, Jimenez JJ, Pastor J, Mauro LM, et al. High levels of circulating endothelial microparticles in patients with acute coronary syndromes. *Am Heart J*. 2003; 145: 962-70.

10 Simak J, Holada K, Vostal JG. Release of annexin V-binding membrane microparticles from cultured human umbilical vein endothelial cells after treatment with camptothecin. *BMC Cell Biol*. 2002; 3: art. no.-11.

11 Jimenez JJ, Jy W, Mauro LM, Horstman LL, Ahn YS. Elevated endothelial microparticles in thrombotic thrombocytopenic purpura: findings from brain and renal microvascular cell culture and patients with active disease. *Brit J Haematol*. 2001; 112: 81-90.

12 Combes V, Simon AC, Grau GE, Arnoux D, Camoin L, et al. In vitro generation of endothelial microparticles and possible prothrombotic activity in patients with lupus anticoagulant. *J Clin Invest*. 1999; 104: 93-102.

13 Freyssinet JM. Cellular microparticles: what are they bad or good for? *J Thromb Haemost*. 2003; 1: 1655-62.

14 Wolf P. The nature and significance of platelet products in human plasma. *Br J Haematol*. 1967; 13: 269-88.

15 VanWijk MJ, VanBavel E, Sturk A, Nieuwland R. Microparticles in cardiovascular diseases. *Cardiovasc Res*. 2003; 59: 277-87.

16 Miyoshi H, Umeshita K, Sakon M, ImajohOhmi S, Fujitani K, et al. Calpain activation in plasma membrane bleb formation during tert-butyl hydroperoxide-induced rat hepatocyte injury. *Gastroenterology*. 1996; 110: 1897-904.

17 Mallat Z, Benamer H, Hugel B, Benessiano J, Steg PG, et al. Elevated levels of shed membrane microparticles with procoagulant potential in the peripheral circulating blood of patients with acute coronary syndromes. *Circulation*. 2000; 101: 841-3.

18 Minagar A, Jy W, Jimenez JJ, Sheremata WA, Mauro LM, et al. Elevated plasma endothelial microparticles in multiple sclerosis. *Neurology*. 2001; 56: 1319-24.

19 Diamant M, Nieuwland R, Berckmans RJ, Pablo RF, Smit JWA, et al. Cell-derived microparticles expose tissue factor in patients with early uncomplicated type 2 diabetes mellitus. *Diabetologia*. 2000; 43: 295.

20 Diamant M, Nieuwland R, Berckmans RJ, Pablo RF, Smit JWA, et al. Circulating cell-derived microparticles in recent-onset type 2 diabetes: A mediator of atherogenesis? *Diabetes*. 2000; 49: 1551.

21 Preston RA, Jy W, Jimenez JJ, Mauro LM, Horstman LL, Ahn YS. Elevated endothelial microparticles (EMP) and platelet activation in severe hypertension. *Blood*. 2001; 98: 3839.

22 Preston RA, Jy W, Jimenez JJ, Mauro LM, Horstman LL, et al. Effects of severe hypertension on endothelial and platelet microparticles. *Hypertension*. 2003; 41: 211-7.

23 Gonzalez-Quintero VH, Jimenez JJ, Jy W, Mauro LM, Hortman L, et al. Elevated plasma endothelial microparticles in preeclampsia. *Am J Obstet Gynecol*. 2003; 189: 589-93.

24 Bretelle F, Sabatier F, Desprez D, Camoin L, Grunebaum L, et al. Circulating microparticles: a marker of procoagulant state in normal pregnancy and pregnancy complicated by preeclampsia or intrauterine growth restriction. *Thromb Haemost*. 2003; 89: 486-92.

25 Volk T, Kox WJ. Endothelium function in sepsis. *Inflamm Res*. 2000; 49: 185-98.

26 Jimenez JJ, Jy W, Mauro LM, Horstman LL, Soderland C, Ahn YS. Endothelial microparticles released in thrombotic thrombocytopenic purpura express von Willebrand factor and markers of endothelial activation. *Brit J Haematol*. 2003; 123: 896-902.

27 Jimenez JJ, Jy WC, Mauro LM, Valle M, Horstman LH, Ahn YS. Endothelial cells (EC) release phenotypically distinct endothelial microparticles (EMP) in activation vs. apoptosis: Findings in TTP patients. *Blood*. 2001; 98: 1045.

28 Heloire F, Weill B, Weber S, Batteux F. Aggregates of endothelial microparticles and platelets circulate in peripheral blood. Variations during stable coronary disease and acute myocardial infarction. *Thrombos Res*. 2003; 110: 173-80.

29 Boulanger CM, Scoazec A, Ebrahimian T, Henry P, Mathieu E, et al. Circulating microparticles from patients with myocardial infarction cause endothelial

dysfunction. *Circulation*. 2001; 104: 2649-52.
30 Muller WA, Weigl SA, Deng XH, Phillips DM. Pecam-1 is required for transendothelial migration of leukocytes. *J Exp Med*. 1993; 178: 449-60.
31 Losy J, Niezgoda A, Wender M. Increased serum levels of soluble PECAM-1 in multiple sclerosis patients with brain gadolinium-enhancing lesions. *J Neuroimmunol*. 1999; 99: 169-72.
32 Solovey A, Lin Y, Browne P, Choong S, Wayner E, Hebbel RP. Circulating activated endothelial cells in sickle cell anemia. *N Engl J Med*. 1997; 337: 1584-90.
33 Shet AS, Aras O, Gupta K, Hass MJ, Rausch DJ, et al. Sickle blood contains tissue factor-positive microparticles derived from endothelial cells and monocytes. *Blood*. 2003; 102: 2678-83.
34 Kim HK, Song KS, Park YS, Kim CM, Lee KR. Method comparison of flow cytometric assay of platelet microparticles and changes of platelet microparticles during cancer chemotherapy. *Thromb Haemost*. 2002; 87: 547-8.
35 Simak J. Elevated circulating endothelial membrane microparticles in paroxysmal nocturnal haemoglobinuria. *Brit J Haematol*. 2004. In press.
36 Nieuwland R, Berckmans RJ, McGregor S, Boing AN, Romijn F, et al. Cellular origin and procoagulant properties of microparticles in meningococcal sepsis. *Blood*. 2000; 95: 930-5.
37 Sabatier F, Darmon P, Hugel B, Combes V, Sanmarco M, et al. Type 1 and type 2 diabetic patients display different patterns of cellular microparticles. *Diabetes*. 2002; 51: 2840-5.

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Reprinted with kind permission from Madden LA, Laden G. Endothelial microparticles in vascular disease and as a potential marker of decompression illness. *European Journal of Underwater and Hyperbaric Medicine*. 2007; 8: 6-10.

Monoplace hyperbaric chamber use of US Navy Table 6: a 20-year experience [Abstract]

Weaver LK

Abstract

We report a 20-year experience at LDS Hospital, Salt Lake City, UT, using the US Navy Treatment Table 6 (TT6) in an oxygen-filled monoplace hyperbaric chamber (1985–2004). Air breathing was provided via a demand regulator fitted with a SCUBA mouthpiece while the patient wore a nose clip. Intubated patients were mechanically ventilated with a Sechrist 500A ventilator, with a modified circuit providing air, when specified. We treated 90 patients: 72 divers (decompression sickness (DCS) = 67, arterial gas embolism (AGE) = 5), 10 hospital-associated AGE, and 8 miscellaneous conditions. They received a total of 118 TT6 (9 TT6 in intubated patients). Ninety-four per cent of the TT6 schedules were tolerated and completed. The intolerance rate from two surveyed multiplace chambers was zero and 3% of 100 TT6 schedules each. Failure to complete the TT6 was due to oxygen toxicity (4) and claustrophobia (3). The US Navy TT6 was well tolerated by patients with DCS or AGE treated in monoplace hyperbaric chambers, but tolerance may not be as high as when treated in the multiplace chamber.

Medical Director Hyperbaric Medicine, Co-Director Shock-Trauma-Respiratory ICU, LDS Hospital; Professor of Medicine, University of Utah School of Medicine, Salt Lake City, Utah

Reprinted with kind permission from Weaver LK. Monoplace hyperbaric chamber use of US Navy Table 6: a 20-year experience. *Undersea Hyperb Med.* 2006; 33: 85-8.

Key words
Reprinted from, decompression illness, decompression sickness, arterial gas embolism, hyperbaric oxygen therapy, treatment

Age associated risks of recreational scuba diving

Richard W Smerz

Abstract

The effect of ageing on risk for development of decompression illness in divers has often been reported as an incidental finding in epidemiological analyses of diving accidents. No previous publications have specifically attempted to quantify or qualify those risks if present. This study demonstrates that ageing increases risk for injury overall, serious injury in particular, and lessens recovery potential.

Introduction

Ageing has long been believed to increase risk for developing decompression illness in scuba divers. As many as 11 reports have indicated that age represented a risk factor, while three others have suggested otherwise.[1] In a previously published study conducted at the Hyperbaric Treatment Center (HTC) at the University of Hawaii's John A Burns School of Medicine, which had assessed the efficacy and outcomes of the unique treatment tables employed there,[2] it had been noted that older divers who had suffered significant injury seemed to have less favourable outcomes. As a result of this incidental finding, there was an interest to investigate the interaction of ageing and decompression illness to determine whether there was an association between age and susceptibility to decompression illness, as well as recovery from decompression illness. This study attempted to more fully elucidate age-related risks associated with scuba diving accidents and to qualify their nature and quantify their impact.

Methods

A chart review of 889 cases treated for decompression illness (DCI) at the HTC between 1983 and 2003 was undertaken in 2004. During a previous review, all cases had been scored by a single investigator with a pre-treatment as well as a post-treatment functional impairment score based upon physical findings and limitations to the conduct of activities of daily living (ADL) at the time of presentation and at the time of discharge respectively. This allowed for assessment of improvement resulting from recompression therapy. Cases were then grouped according to age decades: 20 years or less, 21–30, 31–40, 41–50, 51–60, and 61 and above. The percentage of the study population represented by each age group was determined. These data were then compared with the same age groupings of injured divers as reported by the Divers Alert Network (DAN) at Duke University for the years 1987–2002 from their database. Both of these data sets were compared to the average percentages in each of the same age groups for all US divers certified by the Professional Association of Diving Instructors (PADI) for the period of time spanning that of the HTC study cohort to determine whether there was any disproportionate representation of injury in any age group. The percentage of HTC cases which failed to achieve complete functional recovery for each group was then determined to assess age-related recovery potential. Finally, the percentage of HTC cases in each age group who presented with serious/severe injury was determined as well as the percentage of those cases that were left with residuals.

Table 1
Percentage HTC cases by age group (n = 889)

Age group (years)	# HTC cases	% HTC cases	% with residuals
20 or less	47	5.2%	6.3%
21–30	293	31.8%	3.8%
31–40	318	35.8%	6.2%
41–50	156	17.5%	8.9%
51–60	60	6.7%	13.3%
61 or more	15	1.6%	33.3%

Results

The number of HTC cases and their percentage of the total population are depicted in Table 1. The percentage of cases in the DAN database for years 1987–2002 for the same age groups as the HTC study population were 3.3%, 21.7%, 35.7%, 23.3%, 8.7%, and 2.0% respectively. Table 2 compares both the HTC and DAN injury percentages to the percentage of PADI-certified US divers in the same age groups.

There were 250 HTC cases (28.1%) that were classified as serious. Table 3 demonstrates the numbers and percentages of serious HTC cases as well as the percentage that had residuals in each age group.

Of the total number of cases classified as mild/moderate severity, only 0.5% failed to achieve full functional recovery.

Discussion

There are few published data in the literature that address the age associated risks of scuba diving aside from those done annually by the Divers Alert Network in the report on recreational diving injuries and fatalities.[3] Several studies

Table 2
Percentage HTC and DAN cases vs. PADI–certified divers by age group

Age group (years)	% HTC cases	% DAN cases	% PADI divers
20 or less	5.2	3.3	17.7
21–30	31.8	21.7	41.0
31–40	35.8	35.7	25.3
41–50	17.5	23.3	12.5
51–60	6.7	8.7	3.0
61 or more	1.6	2.0	0.7

Table 3
HTC serious cases and percentage with residuals by age group

Age group (years)	# HTC serious cases	% serious cases	% with residuals
20 or less	15	31.9%	20%
21–30	62	21.1%	19.3%
31–40	83	26%	21.6%
41–50	49	31.4%	30.6%
51–60	27	45%	29.6%
61 or more	9	60%	44%

have intimated that age is a risk factor, but none have attempted to quantify that risk. This study was undertaken to determine whether such risk exists, attempt to develop a "sense" of the magnitude of any age associated risk as well as to define at what age risks may become significant.

A reasonable place to begin to tackle these questions was in first determining how many divers there are in each of the selected age groupings. PADI has trained about 70% of US recreational divers. Using their statistics,[4] the percentage of the total population of PADI-certified divers in each of this study's age groups could be estimated. It would seem logical that if this is the actual distribution of divers by age group, that accidents randomly occurring to divers might be similarly distributed. This would presume that all certified divers are in fact actively diving consistent with that distribution. That in fact may not and probably is not the case. We need better denominator data such as the total number of dives per diver in each age group before we can more confidently draw any rational conclusions as to the true incidence of injury in any age group. However, it is not unreasonable to conclude that any measure of injury rate which exceeds the known proportion of divers in a particular age group would be unexpected and highly suggestive for significance. Thus beginning at age 31 in both the HTC and DAN data sets there is an injury rate distribution ranging from 1.4 to 2.9 times greater than the age population percentage of certified divers in which the injuries occurred. Between ages 31 and 50 the range is 1.4 to 1.8 times greater and between ages 51 and 61+, the range is 2.2 to 2.9 times greater. This suggests that as divers age, their risk for injury increases and becomes more pronounced after age 40 based on this study (Table 2).

Data from Table 1 based upon the HTC cases shows that with advancing age, an injured diver is at increasingly greater risk for incomplete recovery and again this starts to be more pronounced after age 40 and that risk significantly increases at age 50 and older. Fully one-third of those over age 60 failed to achieve full functional recovery in the HTC population. More ominous was the finding that as one reached the age of 50, there was greater likelihood of sustaining a serious injury along with a markedly increased risk for incomplete recovery (Table 3).

Conclusions

This study clearly demonstrates that as divers age, there is some increased risk for decompression illness. Furthermore, it suggests that they are at greater risk for serious injury. If injured there is greater risk for incomplete recovery, and if seriously injured they are at substantial risk for incomplete recovery. Consequently, older divers should become increasingly conservative with their diving practices, limiting depth and total dive times in order to reduce and limit these risks.

References

1 Bove A. *Diving medicine*, 3rd ed. Philadelphia: Saunders & Co; 1997. p. 156.
2 Smerz R, Overlock, R, Nakayama, H. Hawaiian deep treatments, efficacy and outcomes, 1983–2003. *Undersea Hyperb Med*. 2005; 32: 363-73.
3 Vann RD, Denoble PJ, Uguccioni DM, et al. *Report on decompression illness, diving fatalities, and project dive exploration: DAN's annual review of recreational scuba diving injuries and fatalities based on 2003 data*. Durham: Divers Alert Network; 2004. p. 46-77.
4 PADI Americas. PADI diver statistics. Available at <http://www.padi.com> Accessed 23 December 2005.

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Key words
Decompression sickness, scuba accidents, risk factors, age, DAN – Divers Alert Network, data, treatment sequelae

Reprinted with kind permission from Smerz RW. Age associated risks of recreational scuba diving. *Hawaii Med J*. 2006; 65 :140-1, 153.

Letter to the Editor

Project Stickybeak and DAN AP dive accident reporting project

Dear Sir,

I would like to inform your readers that DAN Asia-Pacific has agreed to incorporate Douglas Walker's Project Stickybeak into a broader data collection and reporting project for dive accidents throughout the Asia-Pacific. We are continuing to work closely with Douglas, who is a major part of our Project Team and his input is invaluable.

I wish to take this opportunity to thank Douglas for the tremendous work he has done with *Project Stickybeak* over more than three decades and for his continued support with this expanded project.

Yours sincerely,

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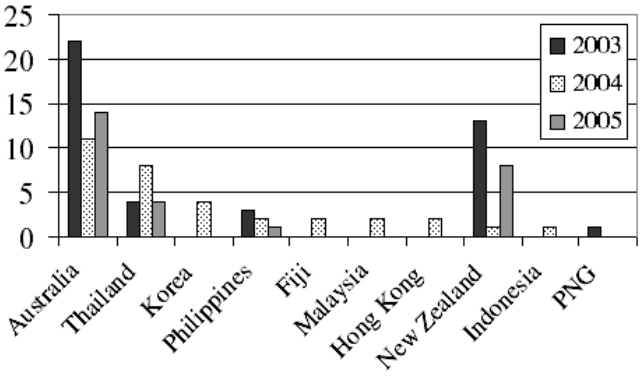
Key words
Letters (to the Editor), accidents, deaths, diving deaths, epidemiology, medical database, DAN - Divers Alert Network

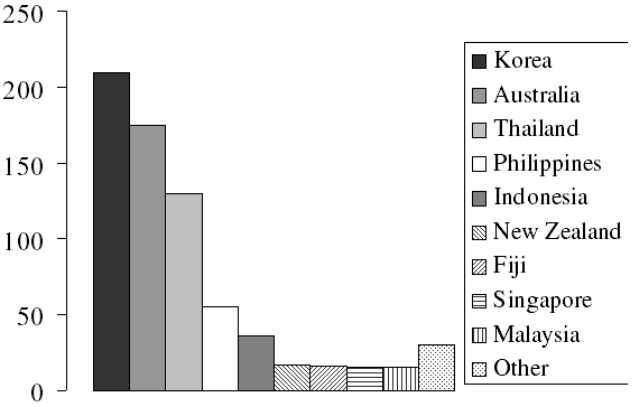
Editor's note: A new notice regarding the expanded DAN AP diving accident and fatality reporting project appears in this issue of the journal on the inside back page to replace the *Project Stickybeak* notice. On behalf of the Society, may I add our enormous appreciation to Dr Walker for his remarkable achievements with *Project Stickybeak* and the invaluable resource that he has provided the diving community.

I would point out that the collation of these tragedies into the database and publications that Dr Walker has created is only one part of the required process. There is a wealth of material here worthy of epidemiological analysis as part of a research degree, and it is the writer's earnest hope that this task will be fulfilled at some time in the future.

Fatalities within the Asia-Pacific region for 2003, 2004 and 2005 documented by DAN AP



Cases of decompression illness within the Asia-Pacific region for 2005 reported to DAN AP by referring hyperbaric units



The database of randomised controlled trials in hyperbaric medicine maintained by Dr Michael Bennett and colleagues at the Prince of Wales Diving and Hyperbaric Medicine Unit is at:

<www.hboevidence.com>

The poetry doctor

The rise and rise of diving computers

It's time to reflect and project again
On those diving computers we use.
Remember first models with inner membrane
That by leaving in the sun we'd abuse?

They held an aura of mistrust and conceit
That threatened the decompression table
And by using such calculating deceit
Suggested that WE were unable!

But now they're intricate in our diving routine
Letting us dive multilevel in style.
Our watches and depth gauges are now "has been"
And we're freed from the square-box profile.

Thermal comfort, fatigue, body weight are requested.
Even plans for flying are sought.
So this technology can adapt and be so adjusted
To bring risk of DCS to nought.

Bottom times too long, ascent rates too steep
Will prompt a warning some way.
Lights flash, buzzers buzz and bleepers will bleep
To warn that all's not OK.

Where will our diving computer take us now?
What new innovations will play?
Smooth-talking voices may tell what and how?
A mask-mounted reading display?

GPS may guide our diving track.
SMS messages may keep us connected.
MP3 music may provide a backing soundtrack.
ECG for our heart inspected.

Our diving computer will soon rule our dive
Controlling all aspects and details.
We won't have to think to now stay alive.
That is ... until it fails.

John Parker
<www.thepoetrydoctor.com>

Nitsch blows No Limits freedive record – safely

Austrian Herbert Nitsch has beaten his own No Limits freediving best by a stunning 29 metres' sea water (msw), to set a new world record of 214 msw depth in a dive off Spetses, Greece, that has been ratified by the sport's governing body AIDA. No Limits is the deepest freediving discipline, in which the diver descends on a weighted sled and returns to the surface aided by a buoyancy device. The desire to smash the 700 ft (213.6 msw) barrier explains why the Austrian chose to try such a deep dive. Freediving world records are usually chipped away a few metres at a time.

Nitsch was underwater for 4 min 24 sec, his maximum depth recorded by three modified dive computers placed on the sled and the diver. The dive was also observed by sonar. The meticulously thought-out sled rig had a number of innovative features. No Limits divers usually ascend aided by an air-bag, but Nitsch employed a solid-state buoyancy unit, eliminating the risk of inflation failure.

Extra weight (total 65 kg) was added to counteract the unit's buoyancy effect on descent. Once the descent weight was dumped, the buoyancy unit could lift Nitsch and his sled rig back up at a rate of 3 $m.sec^{-1}$. The sled rig lanyard attachment systems, together with a personal harness design, ensured that Nitsch would not be trapped if any snagging occurred, and could detach from the whole rig for the final 20 msw or so of his ascent. Rather than burst uncontrollably from the water, he was, therefore, able to hang in the water for a while, including a 30 sec stop at 12 msw, so reducing the risk of DCI. On surfacing, he breathed pure oxygen for 10 minutes as a further precaution.

Other features boosted Nitsch's physiological capacity to descend to great depth. His eye lenses were designed to allow vision when water-filled, saving the need for precious air to equalise pressure in the cavity. Nitsch also carried a drinks bottle with straw into which he exhaled air at 20–30 msw on descent. He was able to suck the air back out later to equalise ears and sinuses, at depths where the diver cannot usually draw air from the lungs for equalisation because they are so compressed. His last draw for equalisation was made a shade deeper than 170 msw. The bottle was filled with water before the dive so that, to meet the regulations, Nitsch did not descend with any air beyond that in his lungs.

Herbert Nitsch's remarkable achievement is reported to have combined clever performance enhancement with advanced safety features – so it is no surprise that he has said he is now seriously considering the great milestone of a 1000 ft (305 msw) No Limits dive!

This edited version is reprinted from a news report from *Diver* magazine, August 2007, p. 16.

SPUMS Annual Scientific Meeting 2008

Dates: May 24 – 31

Venue: Liamo Resort, Kimbe WNB, Papua New Guinea

Guest speakers:

Professor Alf Brubakk
Associate Professor Richard Moon
Dr David Williams (Tropical/Envenomation Medicine)

Themes:

The Treatment Tables
Tropical/Envenomation Medicine Update
Resuscitation Update

Alf Brubakk and Richard Moon are both internationally known diving physiology and medicine experts who have been Guest Speakers at previous SPUMS conferences.

Professor Brubakk is from the Norwegian University of Science and Technology in Trondheim, Norway, and was one of the editors of the 5th Edition of Bennett and Elliott's *Physiology and Medicine of Diving*.

Dr Moon is Associate Professor of Anesthesiology at Duke University Medical Center, USA, and is the Medical Director of DAN International.

David Williams is a research scientist attached to the Australian Venom Research Unit at the University of Melbourne, Australia. His primary interest is in the management of the envenomed victim in tropical countries and he is a knowledgeable and entertaining speaker.

This will be an outstanding meeting in one of the best diving locations in the world.
Details of venue and conference may be found on the SPUMS website: <www.spums.org.au>
For early registration and booking click on 'Diving and U/W Medicine Conferences' and then download the registration and booking forms by clicking on 'Conference Registration'. For details of the venue click on 'Conference Information'.
Early registration/booking is recommended.

Full registration details and a call for papers will accompany the September issue of the Journal.

If you wish to present a paper please contact the Convenor.
Abstracts for presentation should be submitted before 30 April 2008 as a Word file of up to 250 words (excluding references – 4 only) and with only one figure.
Intending speakers are reminded that it is SPUMS policy that their presentation is published in *Diving and Hyperbaric Medicine*. The Editor will contact speakers prior to the meeting.

Conference attendees will be able to receive CME points from relevant medical bodies (RACGP, ANZCA, NZCGP, etc).

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DMAC statement on commercial diving and health 10 July 2007

The health risks associated with work in the commercial diving industry are the subject of a single-page statement issued by the Diving Medical Advisory Committee (DMAC). DMAC is the independent body of diving medical specialists from across Europe that seeks to provide advice about medical and certain safety aspects of commercial diving. The Committee has members from Italy, The Netherlands, Norway and the UK.

"*Divers intending a career in the industry, as well as those responsible for their management, safety and welfare, need to be aware of the health risks associated with work in the industry*," explains the Committee's chairman, **Dr Alf O Brubakk** of the University of Trondheim, Norway. "*Our statement is intended to provide a summary of the current state of knowledge. We strongly advise that individuals wishing to have further, or fuller, information should contact their diving medical adviser.*"

The one-page statement summarises, in five bullet points, the current state of knowledge with respect to pressure effects and other risks arising from the environment, equipment and activity and gives a brief overview on the debate concerning possible long-term health effects for divers.

"*As we say in a sixth and final bullet point, the findings indicate that although long-term health effects are mostly minor in nature, there is a continuing need to improve all aspects of safety within the diving industry*," says Dr Brubakk. "*The improvements in diving procedures which have resulted in significant reductions in incidence of decompression illness may contribute to a reduction in long-term health effects in the future. However, attention should also be paid to other aspects of commercial diver safety, including the risks of trauma and the control of toxic exposures. Measures to control these risks would be expected to reduce any long-term health impact in the future.*"

Further information on DMAC's work is available at <www.dmac-diving.org> and the statement (IMCA D 09/06) can be downloaded from this website.

Issued on behalf of DMAC by:
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Marine Sciences News

Warming reducing ocean life, increasing carbon dioxide

New satellite data is showing that the warming of the world's oceans is reducing ocean life while contributing to increased global warming. The ocean's food chain is based upon the growth of billions of microscopic plants. New data show that ocean warming is reducing these plants, thus imperilling ocean fisheries and marine life. "*We show on a global scale that the growth of…phytoplankton, is strongly tied to changes in the warming of the ocean*," said David Siegel, professor of marine science in the Department of Geography at the University of California, Santa Barbara. "*Phytoplankton grow faster in a cool ocean and slower in a warm one. The scary part is that the oceans are warming now – probably caused by our emissions of greenhouse gases, like carbon dioxide.*"

These microscopic plants are predicted to grow even slower in the warmer oceans of the future. This, in turn, will reduce the food available to fish and other organisms, including marine birds and mammals. Phytoplankton are responsible for about the same amount of photosynthesis each year as all the plants on land combined. Another result of reduced phytoplankton is that the atmosphere depends on the consumption of atmospheric carbon dioxide (CO_2) by these plants. Reduced phytoplankton means less CO_2 is taken up by the ocean, which could speed global warming. These findings are from a National Aeronautics and Space Administration-funded analysis of data from the Sea-Viewing Wide Field-of-View Sensor (Sea WiFS) instrument on the OrbView-2 spacecraft.

The uninterrupted nine-year record shows in great detail the ups and downs of marine biological activity or productivity from month to month and year to year. Captured at the start of this data record was a major, rapid rebound in ocean biological activity after a major El Niño event. The rise and fall of global ocean plant life was compared with various measures of recent global climate change, such as changes in sea surface temperature and surface winds. When the climate warms, the temperature of the upper ocean also increases, making it lighter than the denser cold water beneath it. This results in a layering of ocean waters that creates an effective barrier between the surface layer and the nutrients below, cutting off phytoplankton's food supply. This effect was confirmed by comparing records of ocean surface water density with the Sea WiFS biological data.

Reprinted with minor editing from a news item in *Sea Technology*. January 2007. p. 63.

Scientists prove Nemo does come home

A team of Australian, American and French coral reef scientists have established that Nemo – the lovable orange, black-and-white clownfish of movie fame – really does come home. Around 60 per cent of clownfish return to their home reef after being swept into the open ocean as babies. Working on pristine coral reefs in a marine protected area in Papua New Guinea, an international team, led by Dr Geoff Jones and Dr Glenn Almany of the Australian Research Council Centre of Excellence for Coral Reef Studies at James Cook University, has pioneered a new way to study fish populations by tagging adult fish with a minute trace of a harmless isotope, which they then pass on to their offspring.

The tag is enabling the researchers to understand the degree to which young fish return to their home area or go off to interbreed with more distant populations. This helps to build a picture of the extent to which fish populations are connected or isolated from one another, currently a vital missing link in the sustainable management of fish stocks. "*If we can understand how fish larvae disperse, it will enable better design of marine protected areas and this will help in the rebuilding of threatened fish populations*," Almany explained.

In trials at Kimbe Bay, Papua New Guinea, researchers tagged over 300 female clownfish and vagabond butterflyfish with a barium isotope. Females pass the isotope to their offspring and it lodges in their offspring's ear-bones. "*The isotope is stable, non-radioactive and quite harmless to the fish in these minute amounts, or to humans if it were to be used to tag a table fish*," Almany explained. "*It's simply a way of telling one group of fish of the same species from another*." The team later returned to confirm the tags had worked and studied how many of the offspring went back to their home reef or had dispersed to other reefs. They found around 60 per cent of the juvenile fish returned to the home reef – a tiny dot in the ocean only 300 metres across – after being carried out to sea as babies.

Reprinted with minor editing from a news item in *Sea Technology*. June 2007. p. 51-2.

Vitamin B_{12} an essential vitamin for marine life

Vitamin B_{12}, an essential vitamin for land-dwelling animals including humans, also turns out to be an essential ingredient for growing marine plants that are critical to the ocean food web and Earth's climate. The presence or absence of B_{12} in the ocean plays a vital and previously overlooked role in determining where, how much and what kinds of microscopic algae, or phytoplankton, will bloom in the sea. These photosynthesizing plants, in turn, have a critical impact on Earth's climate as they draw huge amounts of carbon dioxide from the air, incorporating carbon into their bodies. When they die or are eaten, carbon is transferred to the ocean depths, where it cannot re-enter the atmosphere.

B_{12} contains cobalt and can be synthesized only by certain singled-celled bacteria and Archaea. Humans, animals and many algae require B_{12} to manufacture essential proteins, but they cannot make it themselves and must either acquire it from the environment or eat food that contains B_{12}, said the study's researchers, Erin Bertrand and Mak Saito, of the Woods Hole Oceanographic Institution. They wondered whether the vitamin was also important in the ocean, where B_{12} and cobalt are both found in exceedingly low concentrations.

Water samples were collected from three locales in the highly fertile Ross Sea off Antarctica during an expedition in 2005. To one set of samples, they added B_{12} and iron (another essential nutrient for plant growth); to a second set, they added just iron; and to a third, neither was added. Samples stimulated with both iron and B_{12} showed significantly higher concentrations of plant life in general and greater concentrations of a particular type of marine algae called diatoms.

"*The possibility that a vitamin could substantially influence phytoplankton growth and community composition in the marine environment is a novel and exciting finding*," the study states. The finding underscores the complexities of the marine food web and raises questions about the delicately balanced ecosystem's vulnerabilities to changing climate. It also sheds light on the sources and cycling of vitamin B_{12} and cobalt in the ocean, especially in the Southern Ocean around Antarctica, where the only nearby continent – a standard source of metal particles blown into the sea – is largely ice-covered.

Reprinted with minor editing from a news item in *Sea Technology*. July 2007. p. 62.

Scientists discover new life in the Antarctic deep sea

Scientists have found hundreds of new marine creatures in the vast, dark deep sea surrounding Antarctica. Carnivorous sponges, free-swimming worms, crustaceans and mollusks living in the Weddell Sea in the Southern Ocean provide new insights into the evolution of ocean life. Scientists described how creatures in the deeper parts of the Southern Ocean – the source for much of the deep water in the world ocean – are likely to be related to animals living in both the adjacent shallower waters and in other parts of the deep ocean.

A key question for scientists is whether shallow-water species colonized the deep ocean or vice versa. Recent research findings suggest that the glacial cycle of the advance and retreat of ice led to an intermingling of species that originated in shallow- and deep-water habitats. "*The Antarctic deep sea is potentially the cradle of life of the global marine species*," said Professor Angelika Brandt from the Zoological Institute and Zoological Museum, at the University of Hamburg. "*Our research results challenge suggestions that the deep sea diversity in the Southern Ocean is poor. We now have a better understanding in the evolution of the marine species and how they can adapt to changes in climate and environments*."

"*What was once thought to be a featureless abyss is in fact a dynamic, variable and biologically rich environment*," said Dr Katrin Linse, a marine biologist from the British Antarctic Survey. "*Finding this extraordinary treasure trove of marine life is our first step to understanding the complex relationships between the deep ocean and distribution of marine life*."

Three research expeditions, as part of the Antarctic Benthic Deep-Sea Biodiversity project, took place between 2002 and 2005. An international team from 14 research organisations investigated the seafloor landscape, its continental slope rise and changing water depths to build a picture of this little-known ocean region. They found more than 700 new species.

Reprinted with minor editing from a news item in *Sea Technology*. July 2007. p. 49.

BRITISH HYPERBARIC ASSOCIATION ANNUAL MEETING 2007

Dates: 01 to 04 November 2007
Venue: Oban, Scotland

For information contact: BHA 2007, Dunstaffnage Hyperbaric Unit, Scottish Association for Marine Science, Oban, Argyll, Scotland PA37 1QA
E-mail: <info@bha2007.org>
Website: <www.bha2007.org>

UNDERSEA and HYPERBARIC MEDICAL SOCIETY
Annual Scientific Meeting 2008 - preliminary notice

Dates: June 2008
Venue: Salt Lake City

General information and online registration can be found at <http://www.uhms.org/Meetings/AMMeetingsMain.htm>
For additional information contact: Lisa Wasdin
E-mail: <lisa@uhms.org>

ASIAN HYPERBARIC & DIVING MEDICAL ASSOCIATION
Annual Scientific Meeting 2008 – preliminary notice

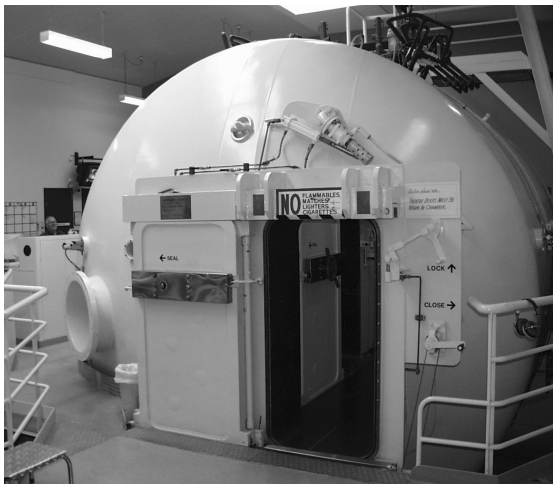
Dates: 15 to 17 May 2008
Venue: Cat Ba Island, Vietnam
For additional information contact:
Dr ('Tony') Lee Chin Thang
Medical Director, Hyperbaric Health Asia
E-mail: <hyperbarichealth@gmail.com>

The Australia and New Zealand Hyperbaric Medicine Group

Introductory Course in Diving and Hyperbaric Medicine
31 March to 11 April 2008
Prince of Wales Hospital, Sydney, Australia

Course content includes:

- History of hyperbaric oxygen
- Physics and physiology of compression
- Accepted indications of hyperbaric oxygen
- Management of decompression illness
- 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-(02)-9382-3880
Fax: +61-(02)-9382-3882
E-mail: <Gabrielle.Janik@sesiahs.health.nsw.gov.au>

The Hyperbaric Research Prize

Guidelines and criteria for nomination and award

The Hyperbaric Research Prize has been introduced to further encourage the scientific advancement of hyperbaric medicine. The Hyperbaric Research Prize 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 Foundation does not preclude the nomination of individuals who have been awarded other prizes and honours for this same research. The Hyperbaric Research Prize will be awarded annually whenever a suitable nominee is identified.

The Hyperbaric Research Prize is international in scope. The awardee may reside or work in any country in the world. However, the research must be available in English to the Foundation's Nominations Review and Awards Committees.

The Hyperbaric Research Prize takes the form of commissioned art piece and US$ 10,000.00 honorarium. Candidates will have produced, within three years of their nomination, research that:

- Identifies important basic mechanisms supporting the existing uses or potentially new uses of hyperbaric oxygen therapy
- Results in the elevation of hyperbaric oxygen therapy to Level 1 evidence of efficacy for a given condition
- Represents ground-breaking clinical findings related to new applications of hyperbaric oxygen therapy

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

Hyperbaric Medicine 2008 Course

Dates: 3 to 5 April 2008
Venue: Columbia, South Carolina

As in the past, the Hyperbaric Medicine 2008 programme will feature an internationally respected faculty and will draw an equally broad audience. Topics will range from emerging new indications for hyperbaric oxygen therapy, case management updates on existing uses and areas of controversy.

If you would like to hear from a particular speaker or on a particular topic we would welcome your suggestions.

Go to: <www.baromedical.com/hbo2008>

Shannon Blanton,
Human Resources Assistant, National Baromedical Services Inc
E-mail: <shannon.blanton@palmettohealth.org>

Instructions to authors

(revised December 2006)

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 3,000 words or fewer. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 15 references. Abstracts are also required for all case reports and review papers. 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 should be as tab-separated text rather than using the columns/tables options or other software and each submitted double-spaced as a separate file. No vertical or horizontal borders are to be used.
Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPG or BMP format. Our firewall has a maximum size of 5 Mb for incoming files or messages with attachments. Large files should be submitted on disc.
Photographs should be glossy, black-and-white or colour. Posting high-quality hard copies of all illustrations is a sensible back-up for electronic files. 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:

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

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.

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PURPOSES OF THE SOCIETY

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
To convene members of the Society annually at a scientific conference

OFFICE HOLDERS

ADMINISTRATION

MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
Membership is also available for retired medical practitioners and medical students.

Membership applications are best completed online at the Society's website <www.SPUMS.org.au>

Further information on the Society may be obtained by going to:
<www.SPUMS.org.au>
or e-mailing <spumsadm@bigpond.net.au>
or writing to: SPUMS Membership,
C/o Australian and New Zealand College of Anaesthetists,
630 St Kilda Road, Melbourne, Victoria 3004, Australia

The Society's financial year is January to December, the same as the Journal year
For current subscription rates please go to <www.SPUMS.org.au>

DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA
1-800-088-200 (in Australia)
+61-8-8212-9242 (International)
The toll-free number 1-800-088-200 can only be used in Australia

NEW ZEALAND
0800-4-DES111 or 09-445-8454 (in New Zealand)
+64-9-445-8454 (International)
The toll-free number 0800-4-DES111 can only be used in New Zealand

SOUTH-EAST ASIA
+65-750-5546 (Singapore Navy)
+63-2-815-9911 (Philippines)
+605-681-9485 (Malaysia)

The DES numbers are generously supported by DAN-AP

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-SEAP website: <www.danseap.org>
They should be returned to:

DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

DIVING-RELATED FATALITIES RESOURCE

The coronial documents relating to diving fatalities in Australian waters up to and including 1998 have been deposited by Dr Douglas Walker for safe keeping in the National Library of Australia, Canberra.

Accession number for the collection is: MS ACC 03/38.

These documents have been the basis for the reports in this Journal as *Project Stickybeak*. They are available free of charge to *bona fide* researchers attending the library in person, subject to an agreement regarding anonymity.

It is hoped that other researchers will similarly securely deposit documents relating to diving incidents when they have no further immediate need of them. Such documents can contain data of great value for subsequent research.