# Diving and Hyperbaric Medicine

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

SPUMS

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EUBS

## First issue joint SPUMS–EUBS journal

**Blinding the blinded**

**CAGE, computers and curiosities**

**Project Stickybeak: 2002 Australian fatalities**

**Endothelial function in diving rats**

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## SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY

## EUROPEAN UNDERWATER AND BAROMEDICAL SOCIETY

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

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

# The Editor's offering

The South Pacific Underwater Medicine Society (SPUMS) was formed in 1971 by a small group of Australian doctors with Dr Carl Edmonds as the first President. A newsletter commenced the same year, and this evolved quickly into an educational quarterly journal, now in its 38th year of publication, funded through members' subscriptions. At present SPUMS has about 850 members. Previously known as the *SPUMS Journal*, the name of the Society's publication was changed in 2006 to *Diving and Hyperbaric Medicine* (DHM) to reflect its increasingly international nature. The Journal is indexed on EMBASE and we are waiting to see whether the application for ISI indexing, and thus an 'Impact Factor' score, has been successful. The present standard owes a huge debt to its previous editor, Dr John Knight, who oversaw its publication, as Assistant Editor (with Dr Douglas Walker) and then sole Editor, for 23 years. The writer has been Editor for five years.

The European Underwater and Baromedical Society (EUBS) was also formed in 1971 at a meeting at the Royal Society of Medicine in London, a meeting which, as a young anaesthetic resident, I was privileged to attend. Its first scientific meeting was held in Stockholm in 1973. For many years a newsletter was circulated to members, and then under Dr Peter Mueller's care this evolved in 1999–2000 into the *European Journal of Underwater and Hyperbaric Medicine* (EJUHM). However, EJUHM always struggled to be a true vehicle for EUBS and to consistently attract articles, and it has had to operate on a very limited budget.

In 2005, EUBS approached SPUMS with a proposal that the two societies join forces to produce a joint journal. The SPUMS Executive was sympathetic to this idea and this first joint issue of DHM is the culmination of these cooperative efforts. This arrangement will initially run for a trial period of two years. The success of this venture, as both Society presidents make absolutely clear in their welcome messages, depends on you, the members of our two societies. There are potential advantages and pitfalls in such a joint venture. Although DHM is an established journal in its own right, the active involvement of Europe can only enhance this, thus benefiting both societies.

Current editorial policy is epitomised by Richard Smith, former Editor of the *British Medical Journal*, who said that journals were for readers first and foremost and what they do best "*is what the rest of the media do best: stir up, prompt debate, upset, probe, legitimise and set agendas. They are good at telling readers what to think about but not what to think…*".[1] SPUMS has always had an emphasis on diving physiology and medicine. With the increasing acceptance of hyperbaric medicine in the Western world, it is now important that DHM also becomes a vehicle for basic and applied research and clinical reports in this field. We have always aimed to publish a diversity of material:

- Original and review articles
- Case series and reports
- Educational and general interest material in the form of
  - The diving doctor's diary
  - The world as it is
  - Critical appraisals (CATs) and Cochrane reviews
  - Opinion papers and Commentaries
- Reprints of full Articles and Abstracts from the literature
- Letters to the Editor
- Book reviews
- Society News & Notices

SPUMS members include physicians from all hospital specialties and primary health care (the latter probably the largest cohort), as well as nurses, physiotherapists, laboratory scientists, ambulance officers, diver medical technicians, hyperbaric technicians and diving instructors to list but some. I suspect the same is true of EUBS, judging from the people whom I had the pleasure of meeting at Sharm El Sheikh last year. To meet the research and educational needs of such a diverse readership is a continuing challenge. Thus, DHM, as long as I am Editor, will remain not simply a vehicle for original research but continue to provide reviews, educational material and to publish some lesser articles that might not always, under the strictest precepts of peer review, otherwise pass muster, but nevertheless have a point of interest.

League tables of one sort or another never tell the full story. However, it is worthwhile commenting on the productivity and support of DHM by Australian and New Zealand hyperbaric units. In the past six years, the Prince of Wales Hospital, Sydney, and the Royal Adelaide Hospital, Adelaide, between them have contributed 30 publications to DHM, including 11 original articles. This is more than the remaining 11 units put together (24 publications, 6 original articles). At the same time, at least six original articles that would have been suitable for DHM have been published in the American journal. Had these also been published here, they would have enhanced the reputation of your own publication considerably. I hope that in the years to come, we will see an improvement in the volume of submissions from both Australasian and European hyperbaric centres, as well as the growing Asian hyperbaric community. Best wishes to you all in your endeavours.

*Mike Davis*

**Reference**

1 Smith R. Travelling but never arriving: reflections of a retiring editor. *BMJ*. 2004; 329(7460): 242–4.

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**Front cover photo of a crested blenny, *Parablennius laticlavius*, hidden amongst colonial anemones, was taken at the Poor Knights Islands by Dr Martin Sayer.**

# Messages from the Presidents of SPUMS and EUBS

## Dr Christopher Acott, President of SPUMS

This is a special issue of the Journal. It is the first following the combining of the EUBS and the SPUMS publications. I warmly welcome EUBS members and hope they will enjoy and contribute actively to its pages.

The SPUMS Journal started as a newsletter with the formation of the Society in the early 1970s. It was published infrequently and was available only to members. It was eagerly read by those in Australia and New Zealand who had an interest in diving medicine. However, it represented the views of a few; there was no peer review and little debate. Over the following years, under the editorial guidance of Douglas Walker and John Knight, the newsletter became a journal. All articles published are now peer reviewed, there is invited editorial comment and the Journal is freely available in various libraries. It became the 'flagship' of the Society and still is. In 2006 it underwent a name change to become *Diving and Hyperbaric Medicine*, in recognition of its increasingly international distribution.

In the past, clinicians and researchers in the field of diving and hyperbaric medicine have tended to submit their work to other journals for publication. Thus, much of the Journal's content has relied, and still does, on the speakers invited to the SPUMS Annual Scientific Meeting for original and review papers. This has been somewhat demoralising for the editors. It has also been disappointing to the President and Committee because some of those papers published elsewhere were submitted to obtain the SPUMS Diploma of Diving and Hyperbaric Medicine. It seemed that the Society was 'good enough' for a Diploma but not 'good enough' for the research to be published in its journal.

Medline recognition for the Journal has been sought twice – in the mid-1980s and the mid-1990s – but was not successful on either occasion. However, the Journal has been indexed on EMBASE for some years. With the future collaborative efforts of EUBS and SPUMS in *Diving and Hyperbaric Medicine* it is hoped that it will be seen by members of both societies as a truly international alternative publication avenue. The Chief Editor will be Michael Davis (Editor for the past five years), who will have the assistance of an Editorial Board. This will consist of Peter Mueller as European Editor and five others. We hope this amalgamation of the two societies will lead to an enhanced exchange of ideas and clinicians internationally, and that we will become more aware of each other's clinical practice and have an opportunity to exchange views in a way that has been somewhat limited in the past. The Journal's expansion and recognition rests with the societies' members – the old newspaper adage "publish or perish" is relevant to us.

## Professor Alf O Brubakk, President of EUBS

This is the first issue of the combined journal and in many ways a new start. For many years we have talked about having closer ties between SPUMS and EUBS, but apart from participation by some of us in the yearly meetings, there has been little cooperation. We hope that this will change and that the combined journal will be an important avenue for joint projects. In general, we Europeans probably know more of what goes on in the USA than we know about activities in Australia, New Zealand and the Asia-Pacific region, and I guess that SPUMS members have a similar problem in reverse.

Diving and hyperbaric medicine is a small field and cannot function in isolation. I am convinced that the field has a lot to offer in other fields of physiology and medicine and that the effect of pressure and different gas mixes can provide many important insights. This potential has by no means been utilised adequately.

Joining the two journals will require additional funding. Shortly EUBS members will receive a ballot for added dues, as we have previously announced. We have received no negative comments on this, and there was general acclamation to proceed from members at the Assembly in Sharm El Sheikh last summer. So we trust that you all see the need for this to happen to take our Society forward.

The establishment of this joint journal effort is in many ways a gamble and the success of this experiment will be dependent on you, the members. You need to provide the material to fill the pages and to contribute your knowledge, insights and effort. If you have a paper to contribute, send it here (spumsj@cdhb.govt.nz) before you send it to another journal. Only in this way will we be able to gain full Medline recognition. We fully understand that at this stage you might not send your best efforts to our journal, but our goal must be to make this journal one of the best. This is, in particular, a request for support from the more experienced and recognised scientists among us; if each of you would send just one paper a year for the next couple of years to this journal, we will in a short time establish it as one of the leading in our field.

# Original articles

## Blinding the blinded – assessing the effectiveness of a sham treatment in a multiplace hyperbaric chamber trial

Dan Rainolds and Robert Long

**Key words**
Hyperbaric research, hyperbaric oxygen, profile, hyperbaric facilities

**Abstract**

(Rainolds D, Long R. Blinding the blinded – assessing the effectiveness of a sham treatment in a multiplace hyperbaric chamber trial. *Diving and Hyperbaric Medicine*. 2008; 38: 3-7.)
Hyperbaric oxygen therapy (HBOT) is used for a variety of problem wounds as an adjunctive treatment. The therapeutic impact of adding HBOT to a wound-healing regimen in many cases remains unclear and an ongoing need exists for additional randomised controlled trials. Many of these clinical studies require a sham group of study participants. To date, there has not been any published research on the concealment of sham treatments in multiplace hyperbaric chambers. The aim of this pilot project was to validate the existing blinding procedures used at one hyperbaric facility. Sixty-six volunteer recreational scuba divers, who had not previously been exposed to compression in a hyperbaric chamber, were recruited through local dive shops. One group was pressurised to 203 kPa and the other was minimally pressurised to 121 kPa, the minimum pressure required to cause middle ear pressure changes. Both protocols implemented continuous, though subtle, pressure variations toward the attainment of the final target pressure. A nearly identical number of subjects in both the 203 kPa (n = 32) and 121 kPa (n = 34) groups believed they had undergone a treatment pressure to 203 kPa (72% versus 71%) indicating a similarity of perception between the two groups.

### Introduction

Hyperbaric oxygen therapy (HBOT) consists of the administration of 100% oxygen ($O_2$) at pressures above 101.3 kPa (1.0 ATA). The Undersea and Hyperbaric Medical Society, an important source of information for diving and hyperbaric medicine physiology worldwide, lists 13 indications for HBOT. Eleven of these are non-diving related, e.g., carbon monoxide poisoning, clostridial infections, acute traumatic ischaemias, and enhancement of healing in selected problem wounds. A common use for HBOT is as an adjunctive modality in the healing of chronic or hypoxic wounds. A typical session for wound healing requires the patient to breathe the increased partial pressure of oxygen ($PPO_2$) for periods of 90–120 minutes. Therapy can be delivered in either a monoplace or multiplace chamber and is typically given once a day for several weeks until the wound is either healed or healing.

To scientifically validate the purported benefits of adjunctive HBOT against existing wound-healing regimens, additional randomised controlled trials (RCTs) need to be undertaken. Exaggerated claims of the benefits of HBOT by some have increased scepticism towards hyperbaric medicine among many in the general medical community. This has further highlighted the need for carefully designed and conducted trials.

Historically, blinded hyperbaric trials have utilised one of two techniques for creating a sham treatment – either the breathing gas mixture is altered or the treatment pressure is maintained at or near 101.3 kPa. As the former technique has certain disadvantages related to cost, complexity and patient risk, it is more common for the pressure to be varied between the sham and treatment groups. The aim of this project was an attempt to validate the existing blinding procedure used at our facility, the Wesley Centre for Hyperbaric Medicine (WCHM). Since this is the first published trial of its kind, we believe it may offer other multiplace hyperbaric facilities a protocol for blinding subjects for their own research.

### Techniques of blinding in a multiplace hyperbaric chamber

OPTION ONE

Both the treatment and sham groups are compressed to identical pressures but the sham group breathes a reduced $PPO_2$ thus breathing a normoxic mixture at pressure. An example of this technique would be to have the treatment group breathe 100% $O_2$ at 203 kPa (2.0 ATA) and the sham group breathe a 10% $O_2$ mix also at 203 kPa. This method has been effectively used in the past and has the advantage of ensuring both groups actually undergo identical pressurisations.[1] Some potential disadvantages with this method include the costs associated with providing the reduced-oxygen gas mixtures and the possibility of inadvertently supplying a hypoxic mixture when gas switches are made during treatment. A further concern is that as the percentage of $O_2$ is decreased there is an increasing

risk of decompression sickness (DCS) for patients in the sham group. In the above example the use of a 10% $O_2$ mix at 203 kPa gives an equivalent air depth (EAD) of 12.8 metres' sea water (msw). This allows for a no-decompression treatment of only 75 minutes. If the study were done at 243 kPa (a pressure commonly used in HBOT) then the EAD jumps to 17 msw and the risk of DCS rises accordingly.

OPTION TWO

In many hyperbaric studies the sham group is compressed to only 111–121 kPa.[2–4] The slight pressurisation of the chamber ensures the chamber door stays sealed. If the chamber door were to inadvertently open midway through the treatment it would reveal that the chamber had not actually been pressurised. Using a minimal change in pressure also reduces the risks of DCS and barotrauma to the sham group. A further benefit is realised in cost savings as this technique does not require expensive gas mixes described above in Option 1. This method does give the patient a feeling of pressurisation as even at 111 kPa the pressure change is felt on the middle ear by most people, requiring a Valsalva or similar ear-clearing technique.

The main disadvantage of this form of patient blinding is that the compression time, as compared with the typical compression rate of 5–10 minutes to achieve a pressure of 203–243 kPa, is quite quick, often just a few seconds. At first this may not seem to be a matter of great concern but it may not always be possible to ensure that the sham and the treatment group subjects do not come into contact with each other in a busy hyperbaric facility. If the two groups were to compare treatment times, they could reveal significant differences in compression times, an issue best avoided if possible.

OPTION THREE

A final option and the one we feel is most likely to truly blind the two groups, is to use multiple, small changes of pressurisation during compression/decompression. This is done for both the sham and the treatment groups. The advantages to this technique are that:

- overall, each group undergoes the experience of pressurisation for a similar duration;
- the compression/decompression profiles are fairly simple to achieve for trained chamber technicians;
- both sham and treatment groups feel the need to clear their ears during the compression phase; and
- costs are kept to a minimum.

The protocol had previously been tested at our facility in a very limited fashion but with the current study we hoped to validate its effectiveness.

**Table 1**
**Demographics of the volunteer divers in the 'treatment' (T) group (pressurised to 203 kPa) and the 'sham' (S) group (pressurised to 121 kPa). Two divers in the sham group had over 500 dives each**

| Group | Subjects | Male | Female | Mean age (yrs) | Mean dives |
|---|---|---|---|---|---|
| T | 30 | 19 | 11 | 32 | 65 |
| S | 34 | 22 | 12 | 33.4 | 125 |

## Methods

The study was approved by the Uniting Healthcare Human Research Ethics Committee, Brisbane. Sixty-six volunteer, certified, recreational scuba divers from the local area, who had not previously been exposed to compression in a hyperbaric chamber, were recruited through local dive shops. The divers were not paid for their participation. The risks of participation, which were principally associated with barotrauma, were explained. Divers were chosen as test subjects as it was thought that they would be more likely than typical non-diver hyperbaric patients to assess accurately the pressure to which they are exposed.

After having the study explained to them and completing a signed consent form, the first group of divers to arrive on a study day was randomised (3–6 subjects per chamber run) to undergo a compression to either 203 kPa, the 'treatment' group, or to 121 kPa, the 'sham' group. The number of divers in each run was considered not to be relevant. The compression profile for each subsequent group on that day was then assigned in alternating fashion. All participants were instructed to avoid communication with each other during the study and this was strictly enforced by staff members.

Once all participants within a group were seated in the chamber and the inside attendant was satisfied everyone was ready, the pressurisation commenced. Initially all groups were pressurised to 111 kPa to seal the chamber door. After a brief stop, all groups did a 10 minute 'descent' procedure that used an up/down pressure profile to mask pressure changes. The ideal pressure–time profiles are shown in Figures 1 and 2 for both the sham and treatment groups.

Immediately after completing the assigned profile all test subjects were asked to fill out a form indicating whether they believed they had undergone pressurisation to a maximum of 121 kPa or 203 kPa, or they were unsure. We used a standard 'off the shelf' dive computer to capture the actual compression profiles. The computer used was found to have an error of over-calculation by 10 kPa (1 msw equivalent) at gauge pressures less than 40 kPa, when calibrated against our chamber gauge. Therefore, the real-time pressure recordings shown in Figures 3 and 4 exceed the actual pressure by 1 msw. Due to this error, the pressure tracings were viewed as representative only and not seen as exact mirrors of the pressures achieved.

**Figure 1**
**Ideal sham profile for a dry-chamber pressurisation to 121 kPa**

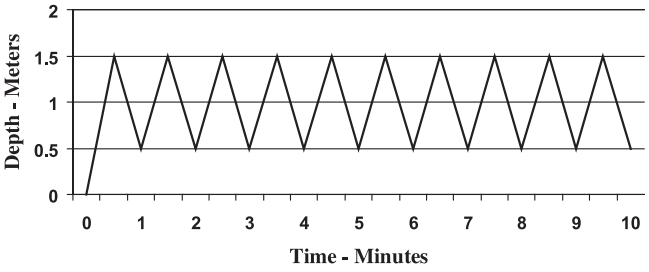
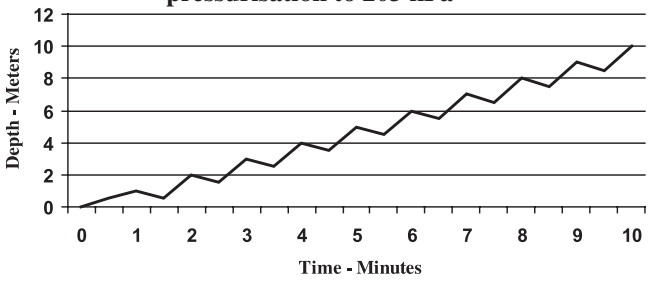



**Figure 2**
**Ideal treatment profile for a dry-chamber pressurisation to 203 kPa**




The study was an equivalence study with an aim to test whether two different treatments were equivalent.[5,6] As it is nearly impossible to blind people from the perception of pressurisation, our goal was to achieve a sham protocol that would be perceived as identical to the actual treatment protocol. The tolerance for testing in this study was set at ±15%, a figure the investigators felt was both reasonable to detect a variance between the two protocols and deemed to be clinically important. The Newcombe method was employed to calculate the confidence interval for the difference between two proportions from independent samples.[7]

For purposes of analysis the 'Not sure' group was collapsed within the group that had a perception of not being pressurised to 203 kPa. This was done in order to increase the expected value of each cell to above 5, which was required for a valid Chi-squared test. Thus, the proportion of patients who perceived they had definitely been pressurised in each group was compared.

## Results

There were no significant differences in age or sex between the two groups. Divers were all between the ages of 21 and 44, having between 8 and 598 dives (Table 1). The mean number of dives in the sham group was nearly twice that of the treatment group, as two divers in the sham group had over 500 dives each. Correcting for these outliers, the average number of dives in the sham group was very similar to that of the treatment group (60 versus 65; rounded).

The perceptions of pressurisation of the two groups are

**Figure 3**
**Tracing of an actual sham pressurisation to 121 kPa. There is an offset of 1 msw at low pressures in the gauge used, which thus over reads the actual pressure by this value**

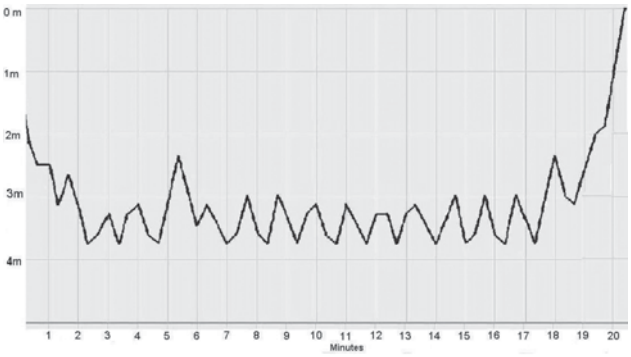



**Figure 4**
**Tracing of an actual treatment pressurisation to 203 kPa. Despite the initial offset of 1 msw in the gauge used, it did not over read at depth**

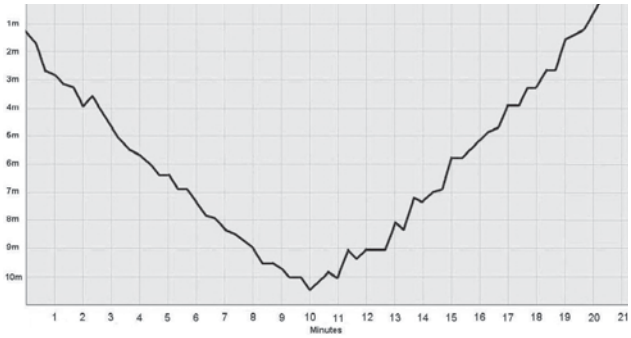
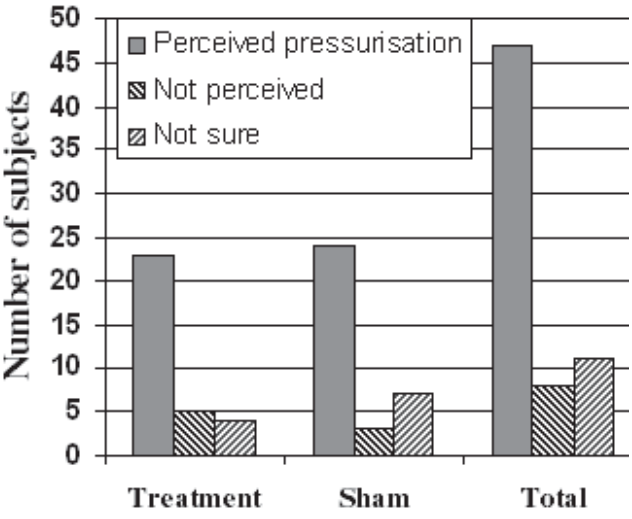



**Figure 5**
**Perception of pressurisation for subjects undergoing 203 kPa of pressure or a sham group undergoing 121 kPa of pressure**



presented in Figure 5. Interestingly the two divers in the sham group with over 500 dives each believed that they had been pressurised to 203 kPa.

Approximately 70% of subjects in each group reported that they had been pressurised to 203 kPa (71.9% and 70.6% for the treatment and sham groups respectively). The difference between these two groups is 1.3% (95% confidence interval of the difference in proportions -20.1% to +22.2%).

## Discussion

When designing a randomised clinical trial it is important to consider how the control group will be handled. Although there may be debate as to the optimal method for blinding participants in a trial, the principle of blinding itself is a cornerstone of modern medicine, with its foundations laid over 250 years ago by British physician James Lind.[8–11] In modern medical trials, the goal is to reduce bias between groups to such a level that any differences observed between them can be said confidently to be related to the intervention itself and not to a placebo effect. In order to effectively assess the results of a particular intervention, it is important to utilise validated blinding techniques. For pharmaceutical studies this may involve a relatively simple and inexpensive process of using visually identical modes of drug delivery for both arms of the trial, but for other types of clinical trials blinding may be much harder to achieve.

Driven by both science and economics, there is an ever-increasing emphasis on the need for evidence-based medicine. It is rather difficult to blind participants to an intervention which itself cannot be concealed. An example might be a trial investigating the use of therapeutic massage for back strain. Blinding participants from knowing whether they were randomised to the massage group or the alternative is difficult, if not impossible. Further, the principles of informed consent may preclude concealment of the intended goals of the trial. Good-quality, blinded, hyperbaric trials have been hampered by these same difficulties. Optimally many clinical studies in hyperbaric medicine require a sham group of study participants and, unlike a trial of a new drug in which the costs of the placebo group are minimal, a single placebo hyperbaric treatment may cost hundreds of dollars. Partly as a result of these high costs, there have been relatively few double-blinded HBOT trials in the past.

Our interest in providing reliable blinding procedures in a multiplace hyperbaric facility began with our recent involvement in a large, multi-centre, blinded HBOT trial. The trial protocol specified that the sham group should be maintained as close to ambient pressure as possible. In order to keep the chamber at or near 101.3 kPa and maintain similar run times between the sham and treatment groups, we have used the option which utilises small yet perceptible pressure changes for both groups. These small pressure variations mean both groups of study subjects experience similar middle ear pressure changes. Further, both groups will have similar overall compression times. We believe this technique to be superior to protocols utilising a standard straight compression profile. Typically a straight compression to achieve 243 kPa requires 8–10 minutes in our eight-patient multiplace chamber compared with the 10–15 seconds to achieve the chamber-door seal of 121 kPa.

The confidence intervals in this study (95% CI -20.1% to +22.2%) are larger than the 15% tolerance limit determined by the investigators to test for equivalence. Since the lower and upper confidence limits exceeded these tolerances, the study does not provide unambiguous evidence that the treatment and the sham are equivalent. However, in an equivalence trial, unlike a typical study testing difference, a conventional significance test has little relevance and absolute equivalence can never be fully achieved.[6] Since true equivalence was not achieved, we cannot say categorically that the two arms of the trial provide identical perceptions of pressurisation, despite the difference between the two groups being only 1.3%.

Limitations of our study were the fact that the actual pressurisations deviated slightly from the ideal pressurisation protocol and also that we had a relatively small number of subjects for a study of equivalence. To definitively assess equivalence a much larger sample size, of over 350 per group, would be required. It should be noted that the results from our trial (performed at 203 kPa) may not be directly extrapolated for a pressurisation to 243 kPa (a treatment pressure used commonly in HBOT).

Before allowing any of our technicians to pressurise the test subjects, the ideal pressurisation pattern was described to them and they were subsequently observed to be following this ideal protocol. Actual pressure tracings were recorded for each technician but not for every compression. Although target depth was never exceeded, we did find some variability between the actual compression profiles and the ideal as described above; however, there was no evidence that this had an important influence on the results. It seems that the presence of frequent variations in pressure is more important than strict adherence to the actual pressures of the protocol.

## Conclusions

The blinding pressure-variation protocol described here is cheaper, simpler, and safer than other multiplace sham options. The majority of subjects in both the treatment and sham groups believed they had undergone a therapeutic treatment pressure. Although the confidence intervals exceeded the tolerance limits set beforehand, the study supports the contention that the technique described here is likely to blind ‘sham treatment’ patients in equal proportion to ‘treatment’ patients when assessing their ultimate treatment pressure, and that, therefore, it can be used with reasonable confidence in hyperbaric RCTs to a treatment pressure of 203 kPa.

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**Conflicts of interest**

No conflict of interest was present.

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# Provisional report on diving-related fatalities in Australian waters 2002

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**Key words**
Diving deaths, scuba, breath-hold diving, surface-supply breathing apparatus (SSBA), diving accidents, case reports

**Abstract**

(Walker D. Provisional report on diving-related fatalities in Australian waters 2002. *Diving and Hyperbaric Medicine*. 2008; 38: 8-28.)
This report covers a total of 33 deaths, of which 17 were in people using a snorkel (three breath-hold diving), 13 in those using scuba, and three in those using surface-supply (hookah) breathing apparatus. There was a wide range of causes of death among the snorkel users, the most unusual being two cases of 'stroke' due to acute, severe hypertensive responses to box jellyfish envenomation. Cardiovascular factors were implicated in nine cases, including a significant history of cardiac disease in three, and single cases of 'silent' cardiomyopathy, 'stroke' in association with hypertension, and obesity. Two cases occurred during the victims' probable first use of a snorkel. In the scuba group, cardiac factors were implicated in 10 of the 13 fatalities, though other factors were also present. Inexperience was critical in at least two cases, and one died when an unsuspected congenital bronchogenic cyst ruptured during an apparently normal ascent. These reports are based on presently available coronial or published reports.

## Introduction

Project Stickybeak has identified and reported on deaths associated with diving in Australia on an annual basis since 1972.[1–5] The deaths identified from official sources in 2002 are reported here. Summaries of the data for each case were made, and these form the basis of this review. Ethical approval was given to the author by the State Coroners for Queensland and New South Wales, and through the Divers Alert Network Asia-Pacific Research Group by the coroners of the remaining Australian states. The general methodology for searching for and reviewing the documentary evidence associated with fatalities has been described previously.[4] These case reports are based largely on the information obtained by the police and others on behalf of the coroner. As there is inevitably variance in the way those present recall events, on occasion a choice has to be made as to which details most probably reflect the actual facts. Where such differences appear to be important, this is noted. The details frequently involved opinions on the experience of the deceased, the water conditions, depths, distances, and passage of time. The purpose of these reports is to assist the identification, and therefore avoidance, of factors predisposing to a serious or fatal outcome to a potentially safe activity.

## Snorkelling and breath-hold diving fatalities

Seventeen snorkelling and breath-hold diving deaths were identified in Australian waters during 2002. The basic data are summarised in Table 1.

CASE BH 02/01

Four friends decided to go spear fishing, two of whom had frequently dived in the same area. The victim, a fit 24-year-old man, was reported to be an experienced diver before he came to Australia from his homeland. The chosen locality was too rough, but a calmer area was found on the other side of a rocky headland. Access was from a rock platform and the two most experienced divers entered the water first to check the conditions. The sea was choppy but the visibility was good, and the remaining two entered the water. The victim speared an octopus, then handed the spear to one of the others and swam back towards the rock wall behind a protruding rock. One of his friends swam after him and saw him as he stood on a rock ledge with his upper body out of the water. He was seen to signal he was 'OK' and then indicated his friend should continue diving on the reef, which he did. When the victim failed to rejoin him, his friend swam back to where he had last seen him but found no sign of him. He swam out to the other two, which took about 10 minutes, to ask whether they had seen him. Becoming worried by his absence, they came ashore, but the check from land was unsuccessful so they all re-entered the water to check among the rocks. Finally accepting that he was missing they notified the Park Rangers' office and a police search was organised. The body was not found until it washed ashore three days later.

*Autopsy:* The autopsy report is limited, stating that "there was green/black discolouration of the skin, mild atherosclerosis of the right coronary artery, and decomposition", and made no report of evidence of trauma. The cause of death was given as drowning.

*Comment:* No information is available concerning his experience in rough water, but even an experienced swimmer could be temporarily impaired if rough water caused contact with a rock.

*Summary:* BREATH-HOLD DIVING; REPUTEDLY EXPERIENCED; BUDDY SEPARATION IN ROUGH WATER AMONG ROCKS; BODY NOT RECOVERED FOR 3 DAYS; DROWNING.

CASE BH 02/02

This fatality was the first recorded in Australia as following a sting from a small carybdeid (box jellyfish) thought to be *Carukia barnesi*, causing the Irukandji syndrome, and has been reported in detail previously.[6] The victim was a 58-year-old male overseas visitor who was stung on his chest and face while in shallow water close to the beach on a resort island. Although he was aware of something stinging him, he did not see the creature. Within about 20 minutes he became distressed, a delay typical of the toxins from box jellyfish, with generalised muscular cramping pains, sweating, anxiety and nausea. His blood pressure was 260/160 and heart rate 142 beats per minute. He was given pethidine, morphine, metoclopramide, promethazine, and diazepam, but 10 minutes later his condition suddenly deteriorated and he became unresponsive, with stertorous breathing. A provisional diagnosis of cerebrovascular accident was made. He was air evacuated to a mainland hospital, where brain CT showed an extensive intracerebral haemorrhage effacing the right ventricle. Death occurred the next day, about 18 hours from the time he left the water.

*Autopsy:* No autopsy was done as the CT had demonstrated the presence of the large intracerebral haemorrhage.

*Comment:* Until this fatality, the danger from these small box jellyfish had been little recognised, though they were first identified in 1963. The dramatic response to the toxin may have been increased by the fact that he was on warfarin prophylaxis as he had a prosthetic aortic valve replacement.

*Summary:* SNORKELLING; CLOSE TO BEACH; MILD STING FELT; ACUTE COLLAPSE WITHIN 30 MINUTES; AORTIC VALVE REPLACEMENT; ON WARFARIN; IRUKANDJI SYNDROME; CEREBRAL HAEMORRHAGE.

CASE BH 02/03

This 44-year-old man was the second victim of the Irukandji syndrome on the Great Barrier Reef (GBR). Details of the incident are sketchy, but it is known that he died in hospital from a cardiac event, a consequence of the severe hypertension caused by the toxin. It was reported he had a medical history of a previous 'stroke'. No autopsy results are available. Scrapings from his clothing showed the culprit was not one of the usual *Irukandji* species but one of the *Malo* genus.

*Comment:* Only limited information is available about this incident, the medical history of the victim, or the medical management.

*Summary:* SNORKELLING; STING FROM BOX JELLFISH OF *MALO* GENUS; ACUTE HYPERTENSION; POSSIBLE HISTORY PREVIOUS CEREBROVASCULAR EPISODE; CEREBROVASCULAR ACCIDENT.

CASE BH 02/04

This 82-year-old man, on a round-the-world holiday with his wife, visited one of the resort islands on the GBR. He had undergone a right hip replacement in March 2000 and had been given a clean bill of health a couple of months before starting the trip. He was booked on a guided snorkel tour while his wife had joined a hike on the island. The boat carried a skipper, a crewman who was to be the lookout, one instructor with a resort diver, and an instructor to lead the eight making the snorkel tour. They were given a safety briefing and asked whether they had any health problems or were using medications, and about their competence in the water. He said he had not been in the water "for a while" but was fine in the water. He declined the offered wetsuit and fins, saying he felt the latter weighed him down. The details are unclear but it appears that some accompanied the instructor while others were swimming independently but under the supervision of the safety lookout. There was minimal current and the island sheltered this area from the wind. The vessel was a 'live boat' and acting as the safety boat, rather than mooring, so not needing to put its rescue tender in the water. One of the group noticed the victim floating face up, his snorkel pointing underwater, so made a 'help me' signal. The instructor was about five metres away and rapidly swam to him. This was also seen by the lookout, the boat reaching the victim shortly after the instructor. The victim was unconscious and not breathing, so resuscitation was started as soon as he was pulled into the boat. The other passengers were recalled and a rapid trip was made back to the island's medical centre. There, the nurse hooked up a defibrillator while awaiting the arrival of paramedics. Resuscitation efforts were ceased after 50 minutes with no response.

*Autopsy:* The autopsy revealed no evidence of drowning. The heart appeared normal, but a few minute areas of subendocardial fibrosis were visible in the antero-septal wall of the left ventricle. The descending branch of the left coronary artery near its origin was almost completely occluded, but its more distal branches appeared widely patent as was the circumflex artery; the right coronary artery had segmental narrowing of about 20 to 30%. Within the aorta there was significant atherosclerosis, particularly in the abdomen, with a number of ulcerating plaques. Histology of the myocardium was essentially normal. It was thought that the most likely cause of death was an acute arrhythmia.

**Table 1. Summary of diving-related fatalities (BNS – buddy not separated, BSB – buddy separation before incident,**

| Case | Age | Sex | Training | Experience | Dive group | Dive purpose |
|---|---|---|---|---|---|---|
| 02/01 | 24 | Male | Nil | Experienced | GSB | Spear fishing |
| 02/02 | 58 | Male | Nil | Not stated | BNS | Recreation |
| 02/03 | 44 | Male | Nil | Not stated | BNS | Recreation |
| 02/04 | 82 | Male | Not stated | Not stated | GNS | Recreation |
| 02/05 | 68 | Female | Nil | Nil | BSB | Recreation |
| 02/06 | 66 | Female | Nil | Nil | GNS | Recreation |
| 02/07 | 66 | Male | Nil | Nil | BSB | Recreation |
| 02/08 | 53 | Female | Nil | Nil | Solo | Recreation |
| 02/09 | 61 | Male | Nil | Nil | Solo | Recreation |
| 02/10 | 19 | Male | Nil | Some | GSB | Recreation |
| 02/11 | 28 | Male | Some | Experienced | BSB | Recreation |
| 02/12 | 72 | Male | Nil | Nil | GSB | Recreation |
| 02/13 | 61 | Male | Not stated | Not stated | Solo | Recreation |
| 02/14 | 56 | Male | Not stated | Not stated | BSB | Recreation |
| 02/15 | 46 | Male | Not stated | Some | BSB | Recreation |
| 02/16 | 48 | Male | Trained | Not stated | BSB | Spear fishing |
| 02/17 | 52 | Male | Not stated | Some | BSB | Abalone fishing |

*Comment:* This incident occurred among a group swimming freely under the supervision of two safety watchers on the boat and a crew member in the water. None of these people or any of the snorkellers noticed anything untoward to alert them that the victim was in trouble.

*Summary:* SNORKELLING; SILENT DEATH CLOSE TO OTHERS; CORONARY ATHEROMA; PRESUMED CARDIAC DEATH FROM ACUTE ARRHYTHMIA.

CASE BH 02/05

This 68-year-old lady was on holiday from overseas with her daughter and taking a day trip to the GBR. They were issued with snorkelling equipment and, though this is not recorded, probably heard a safety talk on snorkelling. On the island, they hired a buoyancy vest for the mother, and booked a supervised snorkel tour for later that morning. To pass the time they decided to practise snorkelling around the jetty area. The daughter entered the water first; her mother followed shortly after and was seen to start swimming. A few minutes later, the daughter looked round and could not see her mother. Although the life jacket could be seen floating at the surface about five metres away, she could not see her mother's snorkel and there was no movement apparent. She started to swim to her mother, but before she got there a stranger swam out and started towing her mother to the beach. There, he started CPR, soon joined by others, two of whom were doctors. They successfully maintained the victim's circulation until the emergency helicopter arrived and transferred her to hospital. She died later that afternoon.

**in Australian waters in 2002, snorkel and breath-hold incidents**
**BSD – buddy separation during incident, GSB – group separation before incident)**

| Depth (msw) | | Weight belt | Comment |
|---|---|---|---|
| Dive | Incident | | |
| Not stated | Surface | No | Buddy separation, ? trauma/heart |
| Not stated | Surface | No | Jellyfish sting/CVA |
| Not stated | Surface | No | Jellyfish sting/cardiac |
| Not stated | Surface | No | Cardiac |
| 0.5 | Surface | No | Drowned in knee-deep water, wearing buoyancy vest |
| Not stated | Surface | No | Cardiac, wearing buoyancy vest |
| Not stated | Surface | No | Cardiac |
| Not stated | Surface | No | Cardiac |
| Not stated | Surface | No | Cardiac |
| Not stated | Surface | No | Drowned |
| 30 | 2.7 | No | Hyperventilation, ascent hypoxia, rescued from 47.2 msw |
| 1 | Surface | No | Cardiac, wearing buoyancy vest |
| Not stated | Surface | No | Cardiac |
| Not stated | Surface | No | Cardiac |
| Not stated | Surface | No | CVA |
| Not stated | Not stated | Yes | Trauma? |
| Not stated | Not stated | Yes | Drowned? GIT history |

*Autopsy:* At autopsy her coronary arteries were widely patent. There was focal myocardial fibrosis noted on histology. No other diseases were identified. The cause of death was given as drowning.

*Comment:* Although not specifically noted, she had almost certainly never used snorkel equipment previously, and the hiring of a buoyancy vest suggests a great lack of confidence in her swimming ability. Her daughter said the water was only knee-deep where the incident occurred, so this appears to be another example of the inescapable tunneling of thought induced by panic. In this case, simply standing up in the shallow water at any time would have led to a favourable outcome.

*Summary:* SNORKELLING; PROBABLE FIRST USE; WEARING LIFE JACKET; BUDDY ONLY 5 METRES AWAY IN KNEE-DEEP WATER; SILENT DROWNING.

CASE BH 02/06

This healthy, 66-year-old woman and her husband, who was a doctor, took a trip to a GBR island with their overseas group. Her only medication was 'Lipitor' for cholesterol control. They had viewed a video on safe snorkelling on the trip out and in the afternoon were provided with mask, snorkel, fins and buoyancy vests before they were taken in a tender to a larger, moored boat for a guided snorkel. There were eight in the group, plus an instructor and a divemaster. The water was choppy, and she, her husband, and a friend were to hold on to a rubber ring and be towed by the instructor, the divemaster also towing a ring. She appeared to have no problems, hooking her arm over the ring to hold on. While they were being towed back to the

boat her mask was seen to be dislodged and the instructor replaced it, and replaced the snorkel in her mouth. Soon after this she was seen to be face up, though she turned back to face down when given a small push by her husband. It is possible the end of her snorkel had been underwater. The snorkel was still in her mouth at this time. A short time later the instructor noticed she was in trouble, took hold of her, and removed her mask and snorkel. She was foaming at the mouth. He rapidly brought her back to the moored boat and resuscitation was started as she was brought back to shore, where oxygen and a defibrillator were employed. Spontaneous breathing returned briefly and her eyes opened but she became unresponsive again and resuscitation was reinstituted. She was evacuated by helicopter to hospital, but was declared dead on arrival.

*Autopsy:* The autopsy showed there were only mild coronary changes, maximal narrowing of 40%, and no evidence of myocardial ischaemia. The cause of death was given as drowning, which may have followed salt-water aspiration causing a cardiac inhibition response.

*Comment:* This incident occurred in between the frequent occasions when the instructor stopped and raised his head to check on those he was towing. It underlines the rapidity with which a critical situation can occur in the water.

*Summary:* SNORKELLING; POSSIBLE FIRST USE; WEARING BUOYANCY VEST; BEING TOWED ON A BUOYANCY RING BY AN INSTRUCTOR; SILENT LOSS OF CONSCIOUSNESS; DROWNING.

CASE BH 02/07

During their holiday to Australia, two brothers took a day trip to a pontoon on the GBR. A snorkelling safety talk was given on the way out. After they arrived at the pontoon they were given mask, fins and snorkel. Passengers with medical or other problems were asked to tell the staff, but neither brother declared any illness. However, the victim, aged 66 years, had a past history of deep vein thrombosis and was on warfarin whilst his elder brother had asthma, although he had apparently not brought his 'ventolin' inhaler on the trip.

The designated swimming area was monitored by a lookout with a rescuer ready to enter the water if needed, and a crew change every half hour to avoid any loss of vigilance. The lookout, who wore an orange safety vest, had a list of those who had notified a medical problem so as to watch them particularly; these individuals were identifiable by a pink ribbon tied to their snorkel. There were about 25 to 40 others in the water when the brothers entered the water together. They found there was a strong current they had not been warned to expect, and after a short time the elder brother became anxious he would have an asthma attack. He waved for assistance as they had been instructed to do, but there was no response. With some difficulty, he managed to get himself back to the pontoon, exhausted. He had not told his brother he was making a return to the pontoon, being too interested in his personal survival. He went and lay down to recover and slept for about 45 minutes. When he awoke, he was unable to find his brother upon searching the pontoon. He became increasingly worried and reported this to the captain. All snorkellers were recalled and a count confirmed that one person was missing. A boat search found the victim's body near one of the adjacent reefs. Despite the presence of rigor mortis, one crewman attempted resuscitation. It was now about three hours since his brother had last seen him. The safety lookouts reported having seen none of those in the water make a signal for assistance in contrast to the elder brother's account.

*Autopsy:* At autopsy, the heart appeared healthy, with no significant coronary disease. Histology showed no evidence of past or recent infarcts, but had occasional areas of relatively diffuse fibrosis or scarring. The pathologist suggested that myocarditis, sarcoid, vascular spasm, or undetected significant atherosclerotic narrowing was the probable cause of death. He noted that he had discussed the findings with other forensic pathologists and their view was that the changes were essentially those of a cardiomyopathy and the actual cause of death was probably a cardiac arrhythmia. The cause of death was given as drowning.

*Comment:* The victim was reportedly an average swimmer but fully capable of getting himself out of trouble, which makes a sudden cardiac factor a reasonable explanation for his drowning. The failure of the lookout to see either his brother's signal or the floating body indicates the difficulty in watching such a large group of snorkellers.

*Summary:* SNORKELLING; EXPERIENCE UNKNOWN; HISTORY OF DEEP VEIN THROMBOSIS ON WARFARIN; SUPERVISED AREA WITH LARGE NUMBER OF SNORKELLERS; BUDDY SEPARATION; UNEXPECTED CURRENT; SILENT DEATH; POSSIBLE CARDIOMYOPATHY; DROWNING.

CASE BH 02/08

A couple joined a day trip to the GBR. A snorkelling safety talk was given on the outward trip. The wife, aged 53 years, suffered from foot problems, requiring 'Panadeine forte', though she had taken none that day. She also revealed that she suffered some type of cardiac arrhythmia especially if she was stressed, and this required her to use 'Anginine'. The crew member who took her medical form noticed there was evidence of a past thyroid operation she had failed to mention. On being told she would not be allowed to undertake a 'resort dive experience' she retrieved her form, tore it up, and said she would go to another operator next day and not be so honest in giving her health history! This crew member thought the woman looked much older than her stated age. It was suggested that the couple snorkel, but first they were asked how they were feeling as it had been a rough trip out and both had been seasick. She swam and

they took a glass-bottom boat trip before lunch. After a heavy lunch, she decided to go for another snorkel whilst her husband rested. After a while, he thought his wife was likely to be tired of snorkelling and went to see if she had returned but was unable to find her anywhere, then met a crew member who told him his wife was probably dead. The designated safety lookout had been alerted to an incident by a call from a crewman in a nearby glass-bottomed boat concerning a person in the water who was not moving. He took a dinghy and found the person unconscious with an arm over the snorkel line of one of the 'rope trails' (these were buoyed at intervals and were all connected to the pontoon). The victim was quickly dragged from the water and brought back to the pontoon where resuscitation efforts were unavailing and a formal declaration of death was made by the Medivac team after it arrived.

*Autopsy:* At autopsy, mild hypertrophy of the left ventricle was noted, the histology showing areas of fibrosis but no evidence of recent infarction or myocarditis. The left coronary artery had about 60% narrowing from an atheromatous plaque about 20 mm from its origin, the right vessel being about 50% narrowed 30 mm from its origin by atheroma. The official cause of death was given as myocardial ischaemia resulting from atherosclerotic coronary artery disease.

*Comment:* The victim had been correctly refused a 'resort dive' but was permitted to snorkel without a buddy in a supervised area. Supervision of individual snorkellers in a crowd can never be perfect, particularly when no indication is given by a swimmer that they are experiencing a problem. No practical and acceptable solution appears likely to be found for the sudden death risk that is a feature of many of the fatalities in this age group of visitors to the GBR.

*Summary:* SNORKELLING; SOLO BUT IN SUPERVISED AREA; HISTORY OF PAROXYSMAL ARRHYTHMIA WITH ANGINA; PAINFUL FEET SO UNABLE TO WEAR FINS; SEASICKNESS EN ROUTE; SILENT DEATH; CORONARY ATHEROMA; CARDIAC DEATH.

CASE BH 02/09

Although travelling together, this couple had met whilst overseas solely to have a companion during their holidays. Neither understood English beyond that essential for basic needs, which limited their understanding of the safety talk given in English over the public address system during the boat trip out to the reef. It is not known whether the 61-year-old victim had ever snorkelled previously. After the incident, his companion stated through an interpreter her ignorance of what medications the victim took, but knew he had had a heart operation 12 to 13 years ago and possibly a heart attack seven years earlier. The boat was anchored in the shelter close to a cay but not on the more commonly utilised permanent mooring as there was a 1.4 metre swell there. Masks, snorkels and fins were supplied and the victim hired a 'shorty' wetsuit. While his travelling companion was still getting ready to snorkel, he jumped into the water without putting his mask on. She heard him say "*I am having difficulties putting the mask on and I believe I had enough already*" then saw him swim towards the platform at the stern of the boat. She found a piece of rope for him to hang onto before herself jumping into the water. About 10 to 15 minutes later, a tender took her back to the main boat, where she found her travelling companion on the platform receiving resuscitation.

In the interim a lookout was watching the swimmers from the boat, changing his position as the boat swung at its mooring. However, the alarm was raised by the boat's hostess who happened to look down and saw a man sitting on the stern duck board. He seemed to be having trouble breathing and did not answer when she asked if he was all right. She told the skipper, who immediately went down to him. The victim was struggling to breathe and not responding to questions. About 30 seconds after being reached, he fell backwards into the skipper's arms. He was lowered onto the deck and resuscitation commenced as he had stopped breathing. Oxygen was soon provided and an airway inserted. Resuscitation efforts were unavailing and a formal declaration of death was made by the Medivac team after it arrived.

*Autopsy:* There was a vertical central chest scar from his cardiac surgery, cardiac tamponade from blood in the pericardial sack due to a ruptured right ventricle (2.5 x 2.5 cm hole), thin-walled right and left ventricles, and a moderately enlarged heart, with severe proximal atherosclerosis in the major coronary arteries. Cause of death was cardiac rupture.

*Comment:* The victim had appeared to be in good health and been swimming on a number of previous occasions with his travelling companion without apparent symptoms.

*Summary:* SNORKELLING; HISTORY OF OPEN HEART SURGERY AND MYOCARDIAL INFARCT; LANGUAGE PROBLEM; SUDDEN ONSET OF BREATHLESSNESS AFTER WATER ENTRY; CARDIAC TAMPONADE FROM RIGHT VENTRICULAR RUPTURE.

CASE BH 02/10

A family and friends drove to a headland for lunch, intending to swim afterwards. An elder brother warned the 19-year-old victim not to go too far out because he was not a good swimmer. The three, identical triplets, and a friend entered the sea from the beach wearing fins, mask and snorkel. They were in and out of the water repeatedly, and then two of the triplets left the water without noticing that the third was not following them. A witness sitting on the headland described hearing a faint cry for help and then saw the victim about 15 metres from the rocks panicking in a rip and being pounded by breaking waves. He courageously

jumped into the sea after judging the correct moment to do so, his intention being to pull the person away from the rough water and avoid endangering himself in the process. When reached, the victim was unconscious and had vomit in his mask, which the rescuer removed before attempting to use the rip to keep away from the rocks. They were both repeatedly submerged by the turbulent water. After about 12 minutes, two more people reached them with a body board and pulled the victim onto it just before two lifesavers arrived in their inflatable rescue boat. He died in hospital two days later having failed to regain consciousness.

*Autopsy:* No adverse health factors were noted. The cause of death was given as drowning.

*Comment:* The opinion of the rescuer was that if the victim had not panicked and tried to fight the rip, but let it take him away from the rocks, he would probably have survived. A young boy in the family group told one of the brothers afterwards that he thought the victim had not been wearing fins when found but this was never formally recorded.

*Summary:* SNORKELLING; NOT STRONG SWIMMER; BUDDY SEPARATION; CAUGHT IN RIP CLOSE TO ROCKS; PANIC; BRAVE RESCUE ATTEMPT BY WITNESS; DROWNING.

CASE BH 02/11

The 28-year-old victim was described by his sister and co-workers as being extremely fit and healthy; a diving instructor able to breath-hold dive to 40 metres' sea water (msw) and stay underwater for one and a half minutes. He was employed on a live-aboard dive boat on a six-day GBR trip. On the fourth day, he was without dive-guide duties for the day. The dive site was a coral bommie rising to about 14 metres below the surface, famous for its schools of pelagic fish. There was a sheer drop to approximately 40 metres at its edge. The sea was described as calm with a minimal current, good visibility and water temperature 28°C. Once all the passengers had entered the water, the victim did a breath-hold dive to about 10 msw near the stern of the boat. Then he made a second dive, to 30 msw (recorded on a dive computer set in 'free dive' mode). There were two crew members in addition to the designated lookout watching as he started his dive and the time was noted. Alarm was voiced when two minutes had passed without him resurfacing. One of the observers entered the water and saw a lot of bubbles about 30 metres below, and a diver rapidly ascending holding a body. He freedived to assist in bringing the victim to the surface, aware that the scuba diver would need to make a decompression safety stop. At about 6 msw depth he ran out of air and had to let the body go. When he surfaced he called for assistance and oxygen. The victim was taken onto the boat and an emergency radio call made.

An instructor was taking a group of divers for a nitrox dive on the bommie and one of this class, who was an underwater photographer, took a photo of the freediver at about 30 msw. He appeared calm and smiled as his photo was taken. Another pupil also saw him as he descended towards the school of barracuda they were watching at 27 msw and watched him look at the dive computer on his wrist, turn and wave and start to ascend. A witness saw him make a short stop before starting his return to the surface and confirmed that this had been his usual freediving practice. Another witness, scuba diving with a buddy at 10–15 msw, saw him as he was ascending, slowly kicking with his fins, but then lost sight of him to watch some fish. The instructor was unaware there was anything wrong until one of the divers pointed animatedly towards the bottom where he saw the victim about 15 metres below him, so immediately dived to reach him. There was blood coming from his mouth, his eyes were closed, there was blood and water in his mask, and he was unconscious. His hands were locked with his fists twisted in towards his chest. The instructor immediately started to bring him rapidly to the surface, in his account making no mention of the intervention of the breath-hold diver from the boat above. At the surface, resuscitation with supplemental oxygen was initiated and this continued as the victim was airlifted to hospital, where death was formally declared. His computer showed he had dived to 30 metres, waited a few seconds, then ascended to 2.7 msw (now 2 minutes from leaving the surface) before sinking to a maximum depth of 47.2 msw. His dive computer showed that he was submerged for a total of 7 minutes and 40 seconds.

*Autopsy:* The autopsy confirmed he had been in excellent health, although mild fatty changes were noted on liver histology. Pulmonary oedema was the only pathology. Blood alcohol nil, urine alcohol 16 mg.100 $ml^{-1}$. The cause of death was drowning following a post-hyperventilation anoxic blackout.

*Comment:* This appears to have been a drowning following a post-hyperventilation blackout (ascent hypoxia). Unfortunately it appears to be an innate characteristic in those who attempt to push their underwater breath-hold ability to believe they are immune from the risks of hypoxia.

*Summary:* BREATH-HOLD DIVING; HEALTHY DIVING INSTRUCTOR; EXPERIENCED BREATH-HOLD DIVER; REACHED 3 MSW ON ASCENT FROM 30 MSW THEN SANK TO 47.2 MSW; POST-HYPERVENTILATION ASCENT HYPOXIA; DROWNING.

CASE BH 02/12

This 72-year-old, heavy-smoking male attended his doctor for two-monthly health checks, but was taking no medication, and had not reported any symptoms. He and his two daughters were overseas visitors taking a trip to an island. They went to the main snorkelling area of the beach and hired masks, fins, and snorkels but declined the offer of instruction on the grounds that they had all snorkelled

before, the father at least three times, though he had not been in the water for a while. Because he had trouble the last time he had snorkelled he had hired a life vest as well. The duty lifesaver "*observed the elderly man in front of the beach hire hut. He was moving his arms and swimming about slowly*" and thought he needed watching as he did not appear to be a competent swimmer. He was swimming in waist-deep water. One daughter saw her father nearby, face down and snorkel sticking up like it should, and thought he was watching the fish. She put her head underwater and saw his arms were hanging down and not moving. She called to him, then pulled on his life jacket, but obtained no response, so she rolled him over and saw his eyes were closed and bubbles were coming from his mouth. She turned him on his side and started to tow him to shore, helped by another person who was swimming nearby. A lifeguard was quickly in attendance and started resuscitation. The victim was intubated by a lifeguard, but the tube was removed when he started breathing and bringing up a lot of water. Whilst being moved on a trolley, he again ceased breathing. An oxygen unit and defibrillator were obtained from the nearby dive shop and his heart and breathing were restarted, but he remained unconscious. He required continued rescue breathing as he was not breathing adequately for himself. On their arrival, paramedics found him in cardiac arrest and worked on him for about 10 minutes before pronouncing him dead.

*Autopsy:* There was an 85–90% stenosis of the proximal right coronary artery. Histology showed moderate interstitial fibrosis in the myocardium and the epicardial arteries showed severe calcific atherosclerosis. Moderate to severe atherosclerosis was present in the aorta with ulceration in the abdominal portion. There was a midline upper abdominal scar from a past gastrectomy with gastro-jejunal anastomosis. The cause of death was given as drowning.

*Comment:* The history of a silent death in close proximity to others makes it probable that a sudden cardiac event led to his inhaling water. It is possible that he panicked and drowned despite the life jacket and shallowness of the water. The life jacket failed to keep his face out of the water.

*Summary:* SNORKELLING; REGULAR HEALTH CHECKS; HEAVY SMOKER; SHALLOW, CALM WATER; SILENT DEATH CLOSE TO OTHERS; CORONARY ATHEROMA; CRITICAL FACTOR POSSIBLY CARDIAC AND/OR INEXPERIENCE; DROWNING.

CASE BH 02/13

A 61-year-old man from overseas and his companion visited the GBR on a day trip. While the boat was anchored at the reef he went for a snorkel, but returned after 10 minutes and went to rest on a sun bed. Although he claimed he was feeling fine, he looked pale and was breathless. During the short time taken for his companion to fetch his heart tablets he became pulseless and apnoeic. Resuscitation efforts including oxygen were continued until paramedics arrived by helicopter at which time he was pronounced dead. He had a history of myocardial infarcts in 1982 and 1991, and triple bypass surgery in 1992. A cardiac catheterisation in 2001 showed that two grafts were occluded and the third partially obstructed. He was taking digoxin, sertaline hydrochloride, enalapril maleate, atorvastatin, aspirin, and a diuretic.

*Autopsy:* There was cardiomegaly (weight 650 g, twice normal), evidence of old posteroseptal and inferior infarcts with borderline left ventricular aneurism formation at the apex, and the aortic valve cusps were sclerotic, but without significant stenosis. There was severe atherosclerosis of the coronary arteries, the ascending and transverse aorta and its major vessels. A graft leading from the aorta appeared occluded. There was fibrosis of the left ventricle wall, in some areas full thickness. Cause of death was given as arrhythmia and asystole due to coronary vascular insufficiency and myocardial ischaemic damage.

*Comment:* There is no record of the victim's apparent health prior to his death but this degree of coronary arterial disease would be likely to produce symptoms. His decision to swim was the trigger for a fatal cardiac episode which was likely to occur whatever life decisions he made.

*Summary:* SNORKELLING; HISTORY TWO MYOCARDIAL INFARCTS; TRIPLE BYPASS WHICH LATER STENOSED; ON MEDICATION; CALM WATER; FELT ILL; RETURNED TO BOAT; CARDIAC ARREST; ACUTE CARDIAC FAILURE.

CASE BH 02/14

This incident occurred during a four-day cruise by a commercial charter fishing boat which carried a crew of two and six passengers. Four of the passengers were related, a man with his twin sons and brother (the victim). The victim, aged 56 years, was described as appearing to be in good health, a non-smoker and non-drinker. On a calm, sunny day while the boat was anchored, the passengers had a swim before lunch. There was a coral bommie about 10 metres from the boat and the victim decided to snorkel to it accompanied by one of the twins. One of the crew was keeping a watch while filleting fish on the aft deck. After a short time the twin returned leaving the victim on the far side of the bommie. As this broke the 'stay in pairs' safety rule, the skipper ordered him to return, though in fact it was the other twin who swam back to the bommie. He found his uncle floating face down as if he were looking at the corals below and then noticed that his legs were dangling under him, so yelled out and immediately turned him face up. He pulled the mask off and noticed froth coming from the victim's mouth; then he started towing him towards the boat. The crewman soon reached him with a life ring on a line and the skipper also entered the water to assist the recovery. Resuscitation continued for about 25 minutes, at which point an emergency doctor contacted by phone advised them to cease their efforts.

*Autopsy:* The autopsy revealed extensive coronary atherosclerosis, with 90% obstruction of the anterior descending artery 3 cm from its origin, and a 70% stenosis of the right coronary artery 5 cm from its origin. The heart showed no gross evidence of scarring or previous myocardial infarction. Histology of the myocardium showed numerous small areas of scarring typical of ischaemic damage but there was no evidence of recent infarction or myocarditis. The cause of death was given as myocardial ischaemia due to coronary stenosis due to atherosclerosis.

*Comment:* The victim's brother later recalled his mentioning some recent pain in his left arm, but as his work involved installation of suspended ceilings this was believed, possibly correctly, to be a musculo-skeletal injury. It is unlikely the outcome would have been different even had there been no buddy separation, but the skipper should be commended for his insistence on the buddy protocol. This is a further illustration of the fact that, in this age group, silent death can occur in the apparently healthy.

*Summary:* SNORKELLING; APPARENTLY HEALTHY; CALM WATER; BUDDY SEPARATION; ATHEROSCLEROTIC STENOSIS, TWO MAJOR CORONARIES; CARDIAC ISCHAEMIA.

CASE BH 02/15

An eleven-strong group from overseas had joined other holidaymakers to make the trip to a popular island and view the coral reef. The victim, a 46-year-old man, was described as an overweight, pack-a-day smoker who was also a heavy drinker. He had hypertension and was taking unspecified medication for this. On the trip to the island he mentioned that he felt unwell in the chest and nauseated. The group chose to swim or snorkel off a beach using hired equipment. The victim explained to the others how to snorkel as they entered the water. There is no mention of conditions but it is probable the sea was calm. After about 45 minutes of snorkelling they all left the water. The victim had a cigarette and a can of beer before returning to the water. About 30 minutes later, he was noticed floating face down and two lifeguards were alerted. They quickly brought him ashore and started resuscitation. Paramedics arrived about 50 minutes later; there was no response to resuscitation.

*Autopsy:* The autopsy revealed an obese man with a well-defined area of haemorrhagic disruption within the mid-right cerebral hemisphere extending into the thalamic region. The major vessels of the Circle of Willis were patent although mildly atherosclerotic. The coronary arteries were widely patent, but there was thickening of the walls of the more distal branches. The left ventricle myocardium was thickened (up to 33 mm) and the heart was enlarged. The liver appeared significantly enlarged, and histology confirmed the presence of severe fatty degeneration. Serum alcohol level of 50 $mmol.L^{-1}$ was from blood taken at the autopsy two days after death and of uncertain significance. There was no evidence of drowning. The cause of death was given as a right-sided cerebral haemorrhage.

*Comment:* He was unfit, but not to a degree that either he or his friends thought it unwise for him to snorkel. There is nothing to suggest that he unduly exerted himself. It is likely this death was not preventable and not a consequence of his in-water activity.

*Summary:* SNORKELLING; HISTORY HYPERTENSION, HEAVY ALCOHOL AND SMOKING; OVERWEIGHT; IN GROUP; SILENT DEATH; RIGHT CEREBRAL HAEMORRHAGE.

CASE BH 02/16

Two married couples, one from overseas, were friends of long standing. The victim, a 48-year-old man, was described by his wife as a very competent diver, such that she never had any fears for his safety; however, no details of his experience are recorded and he had only a basic scuba diving certificate. No details are supplied concerning his friend's training, but it is apparent the friend believed himself to be the more experienced spear fisherman. The two men entered the sea off rocks, the buddy leading. Each wore a wetsuit, weight belt, fins, mask, and snorkel, and both carried spear guns. The buddy saw a fish and dived, surfacing occasionally for air as he pursued it for about five minutes. When he gave up the chase and looked around he could not see his friend so thought he must have returned to the beach. Water depth was about 3 msw in an area close to a rocky reef. When the buddy reached an area where he could stand up, he indicated to those on the shore that his friend was missing, then started to swim back to where he had last seen him. He saw him floating face down and as soon as he reached him started to pull him towards the beach. Soon, others entered the water and helped bring him ashore. There is no mention concerning the management of his weight belt. Resuscitation was commenced while help was summoned. The rescue helicopter and an ambulance came but the victim showed no response to resuscitation efforts.

*Autopsy:* The autopsy showed only minimal coronary atheroma and no medical factors to explain the victim's death. A 60 mm diameter bruise was noted on the inner surface of his scalp in the central forehead area but did not appear to be regarded as significant by the pathologist. The cause of death was given as drowning.

*Comment:* The investigation of this death was sufficient to exclude suspicious circumstances but omitted serious consideration of why it occurred. No reason can be suggested for his death except the possibility that his swimming ability and water confidence level were overstated; the fact that the buddy had decided to lead may indicate that he regarded his friend as lacking experience, though this is not stated. There is no description of the sea conditions so it is probable they were not adverse. The suggestion was made that he may

have hit his head on a rock because of the forehead bruise and proximity to the rocky reef. Whether the equipment was borrowed, his own or a combination was not noted.

*Summary:* SPEARFISHING; APPARENTLY COMPETENT, SCUBA TRAINED; BUDDY SEPARATION; SILENT SURFACE DEATH; NO DISEASE FACTORS; POSSIBILITY OF HEAD TRAUMA; DROWNING.

CASE BH 02/17

The 52-year-old male victim was with others hunting for abalone both on a reef and in the water close to the reef over which waves were breaking. The top of the reef was jagged and slippery. He was described as a good swimmer, and his only known medical history was medication for a gastric ulcer. His son saw him from time to time walking on the reef or swimming close to it, but was concentrating on his own hunting and only became anxious about his father some 90 minutes later. The body was found three weeks later.

*Autopsy:* The body was too decomposed for more than a finding that the victim's death was 'consistent with immersion'.

*Comment:* It is not known whether this fatality occurred in the water or on the reef, but as it was a location containing a potentially dangerous mix of insecure, sharp surfaces and wave action, a fall could easily have occurred and drowning happened before the victim could regain command of his situation.

*Summary:* SNORKELLING; CLOSE TO/ON REEF HUNTING ABALONE; HISTORY OF GASTRIC ULCER; ROUGH SEA; REEF JAGGED AND SLIPPERY; BUDDY SEPARATION; BODY NOT FOUND FOR 3 WEEKS; DROWNED.

**Scuba and surface-supply (hookah) fatalities**

Table 2 provides additional data not included in the case summaries below but derived from the same sources.

CASE SC 02/01

Four friends were diving on a wreck, depth about 37 msw, intending to salvage an anchor and a dinghy they had found previously close to the wreck. The victim, a 55-year-old man, was regarded as a very experienced diver, but he had stated his intention to limit his dive to seven to nine minutes "*because his doctor had advised him not to dive, as he had a viral heart condition*". This case has been reported previously by Acott,[7] but some additional details are provided here.

He and his buddy were the first to enter the water and performed their task of attaching the boat's anchor line to the anchor to be salvaged. While doing this the sand was stirred up and visibility lost, a 'silt out' situation resulting in separation. The buddy, knowing they had completed their job, decided to ascend. After boarding the boat, he decided to re-enter the water to make a decompression stop. As he was descending the line, he saw the victim, motionless, eyes closed, unresponsive but with the regulator in his mouth, so he grabbed him and brought him to the surface. The two other divers described later how they had entered the water 10 minutes or so after the first couple. They were met by the victim when they reached the anchor chain, about 1.5 metres above the sea bed. He made a quick 'out of air' signal and snatched the regulator from the mouth of one of the other divers, who in turn grabbed the regulator from his buddy's mouth. Fortunately the buddy was able to quickly reach her 'octopus' secondary regulator. They then commenced a rapid ascent up the anchor line, connected in a 'daisy chain' with the victim leading. At about 30 msw, the diver whose regulator the victim had been using noticed that the victim's regulator was now hanging by his side, minus its mouthpiece. They continued their ascent, omitting planned decompression. Once at the surface, they required ropes to get him into the boat. His BCD was noted to be inflated. There was no report of any resuscitation efforts. Examination of his equipment showed that the wetsuit jacket was too small, there was a small leak from the BCD supply, and the mouthpiece of the regulator was missing, but there was no functional fault. A dive computer showed his last dive as 22 minutes, maximum depth 38 msw.

*Autopsy:* A pre-autopsy X-ray showed the presence of air within the heart, confirmed at autopsy. There was marked cardiomegaly, patchy interstitial fibrosis, but no significant atherosclerosis. This supported the diagnosis of viral myocarditis. There was no other underlying organic disease. The tongue had been bitten. Medical history included hypertensive cardiomyopathy with previous heart failure, described by the victim's cardiologist as stable. Cause of death was given as cerebral arterial gas embolism (CAGE) and drowning.

*Comment:* The victim had told his buddies about medical advice not to dive, then failed to follow his dive plan. His failure to ascend when he became separated from his buddy was the first step in a fatal cascade of mistakes. It is possible that nitrogen narcosis affected his behaviour.

*Summary:* EXPERIENCED; 37 MSW DIVE; BUDDY SEPARATION AT DEPTH IN SILT OUT; OUT OF AIR; RAPID ASCENT WITH INFLATED BCD; TIGHT WETSUIT JACKET; OBESITY; HISTORY OF HYPERTENSIVE CARDIOMYOPATHY; NITROGEN NARCOSIS POSSIBLE FACTOR; CAGE.

CASE SC 02/02

This case of a 42-year-old woman was also described previously by Acott.[7] In summary, she was obese, with limited mobility from a previous back injury. She was diving

**Table 2. Summary of diving-related fatalities in Australian waters in 2002, scuba and surface-supply incidents**
**BSD – buddy separation during incident, CAGE – cerebral arterial gas embolism, GSB – group**

| Case | Age | Sex | Training | Experience | Dive group | Dive purpose | Depth (msw)* Dive |
|---|---|---|---|---|---|---|---|
| **Scuba** | | | | | | | |
| 02/01 | 55 | Male | Trained | Experienced | BSB | Wreck | 38 |
| 02/02 | 42 | Female | Trained | Some | BNS | Recreation | 3 |
| 02/03 | 52 | Female | Trained | Nil | BSD | Recreation | 11 |
| 02/04 | 56 | Male | Trained | Not stated | BSD | Recreation | 20 |
| 02/05 | 37 | Female | Trained | Nil | BSB | Recreation | 3 |
| 02/06 | 51 | Female | Trained | Some | BNS | Recreation | 11 |
| 02/07 | 44 | Male | Trained | Experienced | GSB | Recreation | 3 |
| 02/08 | 44 | Male | Trained | Experienced | BSB | Crayfishing | 21 |
| 02/09 | 17 | Male | Trained | Some | BSB | Crayfishing | 7† |
| 02/10 | 56 | Male | Trained | Experienced | GSB | Recreation | 18 |
| 02/11 | 63 | Male | Trained | Some | GSB | Recreation | 13 |
| 02/12 | 64 | Male | Trained | Experienced | BNS | Recreation | 13 |
| 02/13 | 20 | Female | Trained | Some | BSD | Recreation | 10 |
| **Hookah** | | | | | | | |
| 02/01 | 52 | Male | Trained | Experienced | BSB | Crayfishing | 8 |
| 02/02 | 23 | Male | Not stated | Experienced | Solo | Scallops | 10 |
| 02/03 | 43 | Male | Not stated | Experienced | Solo | Crayfishing | Not stated |

with an inexperienced buddy in a relatively sheltered bay in calm sea with a slight swell. They snorkelled out to a rock about 250 metres off shore, making a couple of rest stops on the way, and there began diving. The victim found she was too buoyant and so her buddy put another weight on her belt, giving her a total of about 18 kg. After about 10 minutes at 3–4 msw, she indicated she wished to rest, so they surfaced and swam to some rocks intending to climb out. Wave action pushed them into the rocks, repeatedly submerging the victim. It is uncertain whether she retained the regulator in her mouth properly, and she seemed to be having difficulty with her buoyancy. Despite her buddy's attempts at rescue and the arrival of assistance, resuscitation was unsuccessful. Examination of her equipment showed there was adequate remaining air and the regulator and BCD functioned correctly.

*Autopsy:* The autopsy showed marked pulmonary oedema and bilateral pleural effusions. No underlying disease was found and a drug screen was negative. The cause of death was given as salt-water drowning.

*Comment:* This lady appeared to be obese and unfit. The outcome was due to fatigue, and panic when repeated waves submerged her on the rocks. Whether her back pain was a factor is not clear, but she was definitely diving over-weighted and never released her weight belt.

*Summary:* TRAINED BUT INEXPERIENCED; PHYSICALLY UNFIT; OBESE; HISTORY OF BACK INJURY; FATIGUED BY SURFACE SWIM; WAVE ACTION ON SURFACE; FAILED TO INFLATE BCD OR DITCH WEIGHT BELT; EXCESS WEIGHTS;

**(BCD – buoyancy compensation device, BNS – buddy not separated, BSB – buddy separation before incident, separation before incident; *depths and weights rounded; †witness statements range from 5–10 msw)**

| Depth (msw)* Incident | Weight belt | Weight belt kg* | BCD | Air left | Equip test | Comment |
|---|---|---|---|---|---|---|
| 38 | On | Not stated | Infl | Nil | Slight fault | CAGE |
| Surface | Buddy | 18 | Not infl | ++ | NAD | Drowned |
| 11 | On | 10 | Infl | Low | NAD | CAGE? |
| Surface | On | 9 | Not stated | +++ | NAD | Cardiac |
| Surface | On | 12 | Infl | +++ | Some adverse | Drowned |
| 11 | On | Not stated | Buddy | ++ | NAD | Pulmonary oedema |
| Not stated | On | Not stated | Not infl | ++ | NAD | Drowned |
| 8 | On | 9 | Infl | +++ | Some adverse | Cardiac |
| 7† | On | Not stated | Not infl | ++ | NAD | Drowned/Cardiac? |
| 18 | Not stated | Not stated | Not infl | ++ | NAD | Cardiac |
| 13 | On | Not stated | Infl | ++ | NAD | CAGE/Cardiac |
| 13 | On | Not stated | Not infl | Not stated | Not stated | Cardiac |
| 3 | On | Not stated | Not infl | Not stated | NAD | CAGE; ruptured bronchogenic cyst |
| Surface | On | 22 | Not infl | n/a | Some adverse | Cardiac |
| Surface | On | Not stated | Not infl | n/a | Some adverse | Shark attack |
| Surface | On | Not stated | Not stated | n/a | Not checked | Motor boat propeller; major head injury |

INEXPERIENCED BUDDY; DROWNING.

CASE SC 02/03

The victim was a 52-year-old woman from overseas, with a history of hypertension and asthma, but who undertook regular daily exercise including cycling up to 50 km. According to her friend she had not taken any asthma medication for many months, but had been medically retired from her teaching position some unknown time previously. She joined a trip to the GBR, her first open-water dive since her basic training 13 months previously. The boat moored sheltered from the waves by an islet. She had appeared anxious during the outward trip and had asked the instructor to check the assembly of her equipment on the boat, and admitted to feeling quite nervous as it was so long since she had last dived. All her equipment was hired and she was wearing a 5 mm wetsuit, over which she had a thermal short-sleeve vest with a hood. After a buoyancy check, she dived with 10.5 kg of weights, one being placed in her BCD pocket.

The incident dive was her second dive of the trip; no details of the first are recorded. Water temperature was 22°C. It was planned that they would make a 40 minute dive to a maximum depth of 12 metres. She was to dive with two others (one of whom was a 'resort diver') in a group led by an instructor. During the descent she left the group and headed back to the surface. This was noticed by the instructor, who indicated to the others to remain where they were whilst he ascended, meeting her descending again. She indicated that she was okay. After an uncertain time, she indicated she

was low on air, but the instructor assured her she still had plenty. There was a strong current into which the instructor indicated they should swim following him. Because of the strong current, the group became separated and the victim was seen swimming away from the other three. She made a solo ascent. When he surfaced, the instructor saw her floating upright with her BCD fully inflated, about 15 metres away, mask in position and regulator out of her mouth. He swam back to the boat and asked for it to be taken to pick up her and the other divers. As the boat was approaching her he saw that she was now lying face up and not reacting as water washed over her face. He jumped into the water and swam to her to start rescue breathing. Her lips were cyanosed and she was unresponsive. On board, resuscitation was commenced, supplemental oxygen given, and the Coast Guard advised. On shore, advanced life support was unsuccessful. Examination of her equipment showed that it was functioning correctly, and the contents gauge showed 20 bar remaining air.

*Autopsy:* At autopsy, the pleural cavities were opened under water; there was no evidence of pneumothorax. The left lung had a 4 cm lobulated tumour at its hilum, a small cell carcinoma. There was concentric left ventricular hypertrophy consistent with the history of hypertension; petechial haemorrhages were present over the great cardiac vein and on the posterior wall of the heart. The coronary arteries were widely patent. Histology of the lungs showed a slight increase in mucus glands in some bronchi 'consistent with the history of asthma'. In view of the incident history and absence of barotraumas, the pathologist's opinion was that death resulted from a sudden cardiac episode.

*Comment:* Despite her exercise history, she may not have been particularly fit, and she was clearly anxious because this was her first dive since her course 13 months before. The dive leader was aware of her anxiety but had responsibility for a 'resort diver'. The strong current was a factor in her separation from the group during ascent. An alternative cause of her sudden death was CAGE on clinical grounds despite absence of direct evidence. The medical reason for her retirement is unknown.

*Summary:* TRAINED; INEXPERIENCED; HYPERTENSION ON MEDICATION; ASTHMA HISTORY; ANXIETY; STRONG CURRENT; BUDDY SEPARATION; SOLO ASCENT; LOSS OF CONSCIOUSNESS AT SURFACE WITH FULLY INFLATED BCD; NO EVIDENCE PULMONARY BAROTRAUMA; SMALL LUNG CANCER TUMOUR; CAUSE OF DEATH UNCERTAIN; POSSIBLE CARDIAC EVENT OR CAGE.

CASE SC 02/04

This 56-year-old man was on a wreck dive trip through a dive shop with four other customers. He was described as short to medium height and slightly overweight. His diving experience and medical history were not recorded. The dive boat was moored to the buoy line on the wreck and a full dive briefing was given. The water conditions were described as a bit choppy, with a one metre swell, sea breeze of 13–15 knots and an incoming tide of about one knot. The victim was the first to enter the water, from the bow. He was seen to give the 'OK' signal then swim to the line, holding on to it with both hands, regulator in his mouth and mask in position.

A short time later the boatman heard a commotion "like anxious voices" at the bow of the boat and saw the victim gripping the line, with the regulator out of his mouth, rising out of the water when the bow rose with the waves. He did not respond when the boatman yelled to him to put the regulator back in his mouth, and when his buddy replaced it he soon spat it out again yelling out in a panicked voice. The boatman shouted several times telling him to make his way to the stern of the boat where there was a mermaid line. He was seen to swim past the starboard side of the boat, about two metres from it, and showed no response when the mermaid line, which had been pulled in, was thrown to him. He drifted beyond the end of the rope so a longer one was let out, but he made no effort to take hold of it even when it touched him. Whether he dropped his weights is not stated. Two of the other divers reached him about 40 metres beyond the boat and they stayed, one on each side of him, while the boat was brought to pick them up, helping to pull him aboard. There was some delay in recalling the other two as they had dived, unaware of the developing drama, and one of those on board had to dive to bring them back. Resuscitation was commenced, and this was continued by two ambulance officers who came out on a surf life-saving boat, but without response. Examination of the equipment found no faults.

*Autopsy:* Autopsy was reported as showing a right coronary artery occlusion; no additional details are available.

*Comment:* It is possible that panic was a critical factor in triggering the cardiac event. There were no signs of distress or ill health before the victim entered the water, but it is apparent that he found his situation holding on to the line between the dive boat and the buoy stressful as the line lifted and dipped in response to the boat's rise and fall.

*Summary:* EXPERIENCED; APPARENTLY FIT, MILDLY OVERWEIGHT; PROBABLE PANIC RESPONSE AT SURFACE AFTER ENTRY INTO ROUGH WATER; RIGHT CORONARY ARTERY OCCLUSION; SUDDEN CARDIAC DEATH.

CASE SC 02/05

For this 37-year-old overseas woman and her husband this was their last opportunity for a scuba dive before returning home. They had taken their basic course about 6 years previously, but she had probably made only seven dives since then. Her husband said she was very fit because she

was an aerobics instructor, but was liable to panic if she got into difficulty, and apparently she was not a good swimmer. Her husband claimed he had dived in all types of water conditions. They hired tanks, weight belts and hoods, the remaining equipment being their own including the semi-drysuit the victim was wearing. Her experience of diving wearing this type of suit is unknown. She was carrying 12 kg of weight, and using a 55 cu ft cylinder and a regulator with no contents gauge as it was at a dive shop for repair.

Her husband experienced problems in assembling the equipment on the beach, so he asked for advice from both a diving instructor and a newly trained diver. When the instructor returned from taking his group for a dive, 45 minutes later, they were still there on the beach. Both these witnesses concluded that he was seriously inexperienced and in need of revision of the basic skills. However, the instructor thought this area was so safe that he was not overly concerned about them. He later saw them snorkelling in shallow water about five metres from the shore. They probably entered the water from a boat ramp, though this is not clearly stated, and swam out with their BCDs inflated. Their dive plan was to watch sharks. About 35 m off the boat ramp, she said "something doesn't feel right". They agreed she should return to shore and he would continue with his dive. When he surfaced from his dive 15–20 minutes later he called out to ask where his wife was. Learning she was not ashore he descended and started a search for her but the current, surge, poor visibility, and a looming low-air situation limited his search and he contacted the police after coming ashore. He then learned from his children that she had earlier experienced a problem with her mask leaking, that "*something had broken off but she had been able to put it back together*". Her body was found that evening by police divers 40 m offshore, her fins, mask, weight belt and tank still in their correct positions.

Examination of the equipment revealed tears in the silicon mouthpieces of both the main and secondary regulators, which allowed a spray of water when the user inhaled. There was no contents gauge but the tank was full. The semi-drysuit had a tight neck seal. However, her husband reported that she was floating high in the water while swimming when they separated and there is no evidence that she ever changed from snorkel to scuba use.

*Autopsy:* A pre-autopsy X-ray was negative. There was no evidence of coronary atheroma, but perivascular and subendocardial fibrosis and thickened myocardial vessels, and the cellular changes of hypertrophy were seen on histology. There were other findings not relevant to this accident. The cause of death was given as drowning.

*Comment:* Gross inexperience and a liability to panic, compounded by a lack of awareness of the significance of these factors for safe diving, were the critical elements in this tragedy, combined with a poor decision to do a solo return swim to the beach. It is difficult to reconcile the husband's description of his wife floating high in the water as she began her surface swim back to the beach, her BCD inflated, with the experience of a police diver using the same equipment during a test dive. He stated he felt too heavy when he entered the water and had to fin hard to remain at the surface, this requiring so much effort he became out of breath. It is not stated whether his build was similar to the victim's and there is no statement that he wore a drysuit for the test. The tight neck seal and the water spray from the split in the regulator mouthpiece may have contributed to panic.

*Summary:* TRAINED BUT INEXPERIENCED; LIABLE TO PANIC; NEW SEMI-DRYSUIT WITH TIGHT NECK SEAL; BUDDY SEPARATION ON SURFACE BEFORE DIVE; MASK LEAK; BCD INFLATED; FAILED TO DROP WEIGHTS; DROWNED.

CASE SC 02/06

This 51-year-old woman was described as a bright person and extremely health conscious. She had made 20 dives since her basic course 18 months previously. One of her buddies (Buddy 1) on this dive, who had trained with her, reported a previous incident where she appeared to have an episode of impaired consciousness while trying to exit onto rocks. Another friend reported occasions where the victim had complained of trouble breathing, blaming her regulator for 'playing up', and aborting one dive even before descending, citing this as the reason. This dive was arranged as a threesome, a commercial diver friend (Buddy 2) of Buddy 1 joining them, at a sheltered harbour with a reef in 10–15 metres' water. There were several fishermen on the breakwater and the divers were aware of the need to keep well clear of their lines. They entered the water from a sloping flat rock close to the breakwater, Buddy 1 towing an inflated inner tube with a dive flag attached. He soon became separated from the others as he became tangled in one of the fishing lines and had to surface to get free. After regrouping on the surface, the three of them descended again.

The victim was noted by Buddy 1 to make a rapid, vertical, head-down descent. He then noticed the weight on the line from the inner tube was suspended above the sea bed so returned to the surface where he let down more line before rejoining the other two. It was at this time that the victim signaled to Buddy 2 that she wished to surface and they made a slow ascent at a controlled rate facing each other. Buddy 1 described following them, but stopping at 4–5 msw as he expected them to return after correcting some equipment problem. When they remained absent, he surfaced and saw them swimming towards a moored boat about 20 metres away, Buddy 2 appearing to assist the victim. While resting against a large tractor tyre attached to the mooring, she said "*I'm feeling dizzy and going downhill*". Her voice sounded weak, and she appeared to be short of breath and tired, and Buddy 2 had inflated her BCD. At this time the regulator was in her mouth, which was well above the water. As there was nobody nearby to provide help, and the moored boat was too

high to climb into, they decided to swim her to shore.

Buddy 2 described how the victim ascended far faster and descended more rapidly than he did on each occasion and that he had held her arm to control her final ascent while maintaining eye contact to keep her calm. She was breathing quickly as they ascended. At the surface she started using her snorkel but soon panicked so he helped her reach her regulator whilst supporting her with his other arm. She was finning weakly and unable to grasp the boat's anchor chain when they reached it so he put her arms over the tractor tyre and supported her till Buddy 1 reached them. Initially they finned each side of her holding her under the armpits with her on her back. Within two minutes of leaving the mooring the regulator fell from her mouth and she was unable to retain it when replaced. They maintained her face above the water during the swim back to the breakwater area against a current. She lost consciousness before reaching the shore. The swell breaking on the rocks created some difficulty getting her on shore. Assistance arrived but resuscitation was unsuccessful.

Examination of the equipment showed about 85 bar of air; unexpectedly low as they had been diving for only 10 minutes and she started with 210 bar. The BCD functioned correctly and the equipment was in good condition except for incorrect seating of the second (octopus) regulator's diaphragm; this regulator was not used during the incident. There was an excessive water content in her cylinder ( >160 $mg.m^{-3}$; the recommended upper limit <100 $mg.m^{-3}$) of uncertain significance.

*Autopsy:* The autopsy report noted cerebral oedema, frothy fluid in the lower airways, degeneration of the mitral and tricuspid valves, with early ballooning of the tricuspid, sarcoidosis in the hilar lymph nodes, microcalcification of the bundle of His, and fractured ribs consistent with vigorous resuscitation efforts. The Coroner gave the cause of death as 'more consistent with pulmonary oedema than drowning', but the pathologist gave drowning as cause of death, with sarcoidosis, tricuspid valve degeneration, and unacceptable water in the air tank as contributing factors, in summarising his autopsy findings. However, a year later in a written presentation to the Coroner he gave his opinion that the diver's pulmonary oedema syndrome might be the reason for this fatality.[9] He also suggested there be early involvement of medical specialists in diving and hyperbaric medicine in the investigation of diving deaths in this State.

*Comment:* There were several strange incidents in the victim's diving history as mentioned above, including an episode of cyanosis and semiconsciousness after a dive, exhaustion, disorientation and breathlessness. It is unfortunate these previous episodes were not investigated medically. No definite conclusion was reached as to why she drowned, possibilities including a cardiac conduction defect causing arrhythmia. The cardiac valve degeneration may also have been clinically significant.

*Summary:* TRAINED; LIMITED EXPERIENCE; HISTORY OF BREATHLESS EPISODES AND EXCESSIVE FATIGUE; FAILURE TO INVESTIGATE PREVIOUS DIVING INCIDENTS; OVERCONFIDENT; EXCESSIVE USE OF AIR; MITRAL AND TRICUSPID VALVE DISEASE; DROWNED.

CASE SC 02/07

A 44-year-old scientist, who was an experienced diver, was visiting from overseas. He was tired after the flight, and had slept poorly the first night after he arrived. The opportunity to dive arose on the second evening. Wetsuits, weight belts and tanks were hired, but he and a colleague had brought the rest of their equipment with them. They were guided by two experienced local divers. The victim's experience had been in tropical waters but his colleague was a qualified diving instructor with cold-water dive experience. They entered the water off a boat ramp close to a shallow rock wall where sea dragons are commonly found. Conditions were described as being calm, but cold.

The dive leader had a video camera and asked another colleague who was to remain ashore to record their water entry. When this was viewed later, it showed the victim's gear was not properly adjusted – the tank strap could be seen hanging down in a loop so the tank was able to slip out as the BCD was lifted off the ground. Also the 'octopus' regulator was not secured so that it was readily available and the BCD inflator hose was also not secured. Just before the victim entered the water, his BCD feed and depth gauge could be seen almost off his left shoulder, which would result in them floating behind him and being extremely difficult to reach. His buddy reported checking him before the victim entered the water. They each had a torch, and a 'cyalume' stick attached to their tank.

The group submerged when in chest-deep water and swam to the rock wall, about nine metres away. One of them found a sea dragon in about 3 msw depth. It was then noticed that one diver was missing so they immediately surfaced. The victim's light was seen on the seabed almost at the end of the launching ramp in a depth of about 2 msw. His colleague reached him, inflated his BCD and easily brought him to the surface and towed him to shore where the other two divers helped to pull him out of the water and start resuscitation, continuing till the ambulance arrived: there was no response.

Examination of the equipment showed that the venturi of the second-stage regulator was set in pre-dive rather than dive mode, which would have restricted air flow. Otherwise the equipment functioned satisfactorily. Excessive water and $CO_2$ content was noted in the air in the cylinder.

*Autopsy:* Pre-autopsy CT showed a small amount of gas in the liver, a large air bubble in the stomach, probably due to the resuscitation efforts, and a small amount of air in the

heart. The left coronary arteries showed up to 30% narrowing by atherosclerosis with a 60% narrowing of the distal right coronary artery. These stenoses were not considered clinically significant by the pathologist. The cause of death was given as drowning.

*Comment:* The critical factors were thought to have been the cold water, to which he was unused, fatigue and anxiety as he was inexperienced in night diving and became separated from the others. His snorkel was missing but the significance of this is unknown. His death occurred very shortly after entering relatively shallow water. He was apparently healthy, though with a history of asthma, and had passed a pre-employment medical check some six months before this dive.

*Summary:* EXPERIENCED BUT POSSIBLY FIRST COLD-WATER NIGHT DIVE; ASTHMA HISTORY; BUDDY SEPARATION SOON AFTER WATER ENTRY IN SHALLOW WATER; FAILED TO INFLATE BCD OR DROP WEIGHT BELT; CORONARY ARTERY ATHEROMA; DROWNING.

CASE SC 02/08

This 44-year-old experienced male diver was on medication for IgA nephritis. A dive for crayfish was planned with two friends, involving a climb down a rocky cliff to reach a suitable water entry site. The water was described as calm with no swell. The victim had been diving regularly for years; the experience of his buddy reportedly was less than his. They separated after entering the water and it was only when the other two surfaced after 45 minutes that they saw the victim floating face upwards at the surface. At first they thought he was resting, then they saw a wave wash over his face without him showing any reaction, so realised something was wrong. His weight belt was on, BCD inflated. They towed him to a rock platform and commenced resuscitation, this being continued till paramedics arrived: there was no response.

Examination of the equipment showed 150 bar in the cylinder, indicating death had occurred soon after entry into the water. The equipment was described as being quite old, but well maintained. The tank contained a small quantity of water and the second stage had a slight problem with the diaphragm that made it difficult to breathe under stressful conditions.

*Autopsy:* A pre-autopsy X-ray of the chest revealed no gas in the mediastinum or pleural cavities, and the heart contained no air when opened underwater. The proximal segment of the left anterior descending coronary artery had an 80–90% occlusion and the right coronary artery showed approximately 70% stenosis by eccentric atheroma. No thrombi were present in the coronary arteries. Histology of the kidney showed scattered hyalinised glomeruli. Cause of death was given as spontaneous lethal arrhythmia in a man with significant double-vessel coronary artery disease.

*Comment:* The inflated BCD indicates that the victim had enough time to react to the onset of a problem. He had reportedly aborted his previous dive because of chest pain but had not sought medical advice. It is apparent that he managed the difficult access to the water down the cliff path without obvious symptoms, but this may have expended his cardiac reserve and left no margin to meet the demands of the dive itself.

*Summary:* EXPERIENCED; BUDDY SEPARATION; HISTORY OF ABORTING PREVIOUS DIVE DUE TO CHEST PAIN; NEPHRITIS; INFLATED BCD; WEIGHT BELT NOT RELEASED; CORONARY ARTERY DISEASE; SUDDEN CARDIAC DEATH.

CASE SC 02/09

A family group and friends were visiting a relatively sheltered, shallow, sandy bay to catch crayfish. Approximately 100 metres offshore there is a rocky outcrop, and there are numerous reefs that conceal crayfish. The sea was calm with only a slight swell, excellent visibility, and a slight surge around the reef opening. The victim, a 17-year-old male, had trained several years before but had dived infrequently since. He was apparently fit and well, apart from a history of a 'drop attack' some two years previously. He and his father undertook a 45 minute boat dive, then returned to shore for a rest. As the victim and his father had about 100 bar of air remaining, they decided to make a second dive in the same area and were taken out in one of the boats. After about 10–15 minutes the father caught a large crayfish under a ledge while the victim held the torch, but it was soft-shelled and he let it go. When he looked around again he did not immediately see his son, but then saw him lying motionless under a ledge, apparently trapped. He attempted to pull him free but quickly abandoned this when, he said, his mouth suddenly filled with water and he made a rapid ascent to the surface, there yelling out to the man in the boat. He was pulled into the boat and this man, realising the situation was urgent, put on scuba gear and jumped into the water. He saw the fins sticking out under a ledge in about 5 msw. Swimming round to the other side, he saw the victim was unconscious, the regulator out of his mouth, and his arms in front of him. After unbuckling the victim's backpack unit, he pulled him free and brought him to the surface. Resuscitation was initially successful but he died later in hospital without regaining consciousness. The electrocardiogram showed a long QT interval. Inspection of the equipment showed it to be in good condition, and the tank still contained 90 bar.

*Autopsy:* No autopsy was performed. The cause of death was certified by the hospital doctor as cerebral anoxic damage, a 'delayed-drowning' death, with the victim's long QT syndrome a possible critical factor.

*Comment:* This case has been reported previously by Acott in a review of the long QT syndrome.[8] The fatal progression of events was initiated by the victim's decision to leave his buddy, despite their agreement to stay together, then he became wedged under a ledge. The two divers were separated for possibly a minute or so. Whether the victim was actually trapped is uncertain, it being possible he was merely difficult to extricate after losing consciousness, or he may have felt trapped and lost the regulator from his mouth as the critical sequence of events.

*Summary:* TRAINED BUT INEXPERIENCED; CRAYFISHING; BRIEF BUDDY SEPARATION; FOUND CAUGHT UNDER A LEDGE; PREVIOUS BLACKOUT; LONG QT SYNDROME; ACUTE CARDIAC DEATH.

CASE SC 02/10

Prior to a family trip to Australia this 56-year-old man underwent a 'full medical check' at his own request, including a cardiac stress test. This indicated 'a slight abnormality' so he had a cardiac catheterisation and blood tests. An elevated blood sugar was noted and he received dietary advice. He was assured he was fit to continue the type of diving to which he was accustomed. He was accompanied by his wife and their two sons, one of whom had been diving for 10 years and was now a divemaster. The victim had trained in 1997 and made about 80 dives around the world with his wife as his usual buddy. They walked in the Blue Mountains and climbed the Sydney Harbour Bridge before staying at a GBR island resort, and booking a diving trip. On the dive charter boat, a routine safety talk was given to the mixture of scuba divers and snorkellers, and he was cleared to dive after showing the recent medical certificate from his physician.

The family members were using their own equipment except for hired tanks. Another safety talk was given to the scuba group, all equipment was checked, and they were divided into two groups of three buddy pairs with an instructor as guide. The victim was buddied with his wife and their two sons were paired together, all being in the same group. During the descent, his wife experienced some ear equalisation problems and they ended up on the seabed close to the other group of six divers. Meanwhile their two sons had returned to the surface along with their dive guide because the less experienced one was unable to effectively clear his mask. He decided he would rather join the snorkellers than dive again, while his brother chose to buddy with their guide. There was a slight current but the sea was calm and visibility about 9 metres. Meanwhile, the victim and his wife saw the other group move off and followed them, holding hands, but its guide noticed and signaled to them to go with their own guide, who was now close behind them.

They followed her till the victim's wife pointed out some coral for the victim to photograph. While he was trying to focus his camera, his wife saw that their guide was swimming away and noticed that there was a slight current against them. She signalled to the victim what she intended, then followed their guide. They had swum about 3–5 metres before the guide turned round and queried where her buddy was. It was only then she realised her husband was not close behind her. She could see the coral he had been photographing but he was no longer there. At this time they heard a dinghy moving above them. The group now ascended, making a safety stop together, and surfaced close to the dive boat. It was only then that she learned her husband was ill and receiving emergency resuscitation. Prolonged resuscitation efforts were unsuccessful. The safety lookouts had seen the victim surface about 10–15 metres from the boat. He was waving his arms and definitely conscious at this time, but was not heard to shout or make any noise. He was reached by the dive tender within a minute or two, by which time he was unconscious, floating on his back, mask on his face and BCD not inflated. The regulator was held tightly by his clenched teeth.

Examination of the equipment showed no faults and ample remaining air, but there is no description of whether the integrated weights were still present in the BCD pouches or had been dropped. His gauge showed maximum depth to be 18.5 msw.

*Autopsy:* The autopsy revealed evidence of an old anterior myocardial infarct near the coronary ostia. The remaining cardiac tissue appeared normal macroscopically, but histology showed extensive areas of interstitial fibrosis in the anterior left ventricle. The coronary arteries were patent and showed only moderate atheroma, while the abdominal aorta also showed moderate atheroma. There was no indication of arterial gas embolism; nothing in the reported findings suggests this diagnosis. The cause of death was given as probable ventricular arrhythmia due to myocardial ischaemia due to coronary atheroma.

*Comment:* As there was no reason to suspect dive stress, there is no suggestion that the fatal event could or should have been predicted. This case is an unfortunate demonstration of the limitations in predicting life expectation even after thorough medical investigations.

*Summary:* TRAINED; EXPERIENCED; RECENT CARDIOLOGICAL ASSESSMENT WITH MINOR PROBLEM NOTED AND ELEVATED BLOOD SUGAR; BUDDY SEPARATION; SOLO ASCENT; LOSS OF CONSCIOUSNESS ON SURFACE; EVIDENCE OLD MYOCARDIAL SCAR; ACUTE CARDIAC DEATH.

CASE SC 02/11

Travelling alone from overseas, this 63-year-old man was trained but 'a bit rusty' having not dived for two years. However, he made two dives without incident following his arrival and he reported this when he signed up for a live-aboard dive cruise. It was noted he had some difficulty

controlling his buoyancy, this tending to cause separation from his buddy. At the dive briefing, it is thought he indicated he had made a night dive the previous night, but this is uncertain. There was a slight current and clear visibility when he and his two buddies descended to 13 msw. He wore a lycra suit under a wetsuit and hired scuba equipment. After about 25 minutes, they passed another group and soon after this his buddies noticed his absence. They assumed he had joined this other group so were not worried.

The surface safety watch saw a light come to the surface about 70 metres away and called out, receiving a reply from the diver indicating he was okay but wished for assistance back to the dive boat; he gave no appearance of any distress. The water was too shallow for the tender to reach him but he said he was not able to swim to it, so a rope was thrown to him and he held onto it while he was dragged clear of the coral. As he raised his hand to be helped into the tender he went slack so the crewman removed his weight belt and other equipment and tried to pull him into the tender, but when he found he could not do so he entered the water to keep the diver's head above the surface and to give in-water rescue breathing. With others to help he was brought to the dive vessel where resuscitation was started, but to no avail. Examination of the equipment found it to be in good condition and there was about 90 bar in the cylinder.

*Autopsy:* A pre-autopsy X-ray showed no evidence of abnormal tissue air, and no evidence was found during physical examination of the body. The myocardium of the left ventricle appeared slightly thickened (28–33 mm). The left anterior descending (LAD) coronary artery had an area of over 80% stenosis 8 mm from its origin and 9 mm in length, but was patent further on. The right coronary artery was widely patent throughout its length. There was no evidence of past or recent myocardial infarction. Lung histology showed possible signs of an intercurrent infection. The cause of death was given as cardiac failure.

*Comment:* The reason for the victim's separation and solo unobserved ascent is unknown as he had adequate remaining air. It is possible he experienced some panic when he lost contact with his buddies. Whether this resulted in a cardiac arrhythmia or an air embolism cannot be known. Although a diagnosis of CAGE may be sustainable on clinical grounds, the severity of the LAD stenosis makes a cardiac cause of death a more probable diagnosis. Pharmacological evidence of the use of nasal decongestants supports the possibility of a respiratory tract infection, though this had no apparent part in the course of events.

*Summary:* TRAINED; NIGHT DIVE; BUDDY SEPARATION; LOSS OF CONSCIOUSNESS AT SURFACE; POSSIBLE RESPIRATORY TRACT INFECTION; CORONARY ARTERY DISEASE; NO EVIDENCE OF CAGE; SUDDEN CARDIAC DEATH.

CASE SC 02/12

The victim was a 64-year-old, apparently fit man with an active lifestyle, whose occasional symptoms from a hiatus hernia were his only medical problem. He had mentioned taking an antacid "for an upset stomach" on the morning of this dive. He was a very experienced and active diver, and had dived on the two days prior to this boat dive. His first dive was incident free and, after an appropriate surface interval, he made a second dive with his buddy. The water conditions were good, no significant current or wave action. After about 20 minutes his buddy looked back, as he was following close behind, and they exchanged 'OK' signals, but when the buddy turned round five minutes later he saw the victim stationary in the water with the regulator out of his mouth. When he tried to replace it he was unsuccessful as the victim was unresponsive. Rescue breathing was started as soon as he was brought to the surface, and resuscitation in the dive boat, but without success.

*Autopsy:* At autopsy he had an enlarged heart and significant coronary artery disease. Acute cardiac death.

*Comment:* This death appears to have been completely unpredictable. It is possible that the victim's 'indigestion' had, in part, been anginal pains.

*Summary:* EXPERIENCED; APPARENTLY FIT MAN; SUDDEN LOSS OF CONSCIOUSNESS UNDERWATER; CORONARY DISEASE; ENLARGED HEART; SUDDEN CARDIAC DEATH.

CASE SC 02/13

The victim was a 20-year-old, apparently fit woman, trained but inexperienced. At the end of an uneventful dive lasting 40 minutes to a maximum depth of 10 msw she and her buddy had ascended to 3 msw. There, without warning, she began to descend head first, apparently unconscious. Her buddy brought her to the surface but she showed no response to resuscitation efforts.

*Autopsy:* The autopsy revealed the presence of a congenital bronchogenic cyst in the right upper lobe, which had partially ruptured, probably due to the air expanding during ascent, resulting in an arterial gas embolus. There was haemorrhage within the centre of the cyst.

*Comment:* No other details are available about the victim's medical history, the diving incident and the autopsy findings. It is possible that a chest X-ray as part of a diving medical assessment might have demonstrated the cyst's presence.

*Summary:* TRAINED BUT INEXPERIENCED; NORMAL ASCENT; RUPTURE OF UNDIAGNOSED CONGENITAL BRONCHOGENIC CYST; CAGE.

CASE HH 02/01

A 52-year-old man, who had been diving for 18 years, was described by his son as "*fit and healthy, been diving most of his life and never had any problems*". He took out two friends for a day's diving. This case has been reported previously by Acott.[7]

It was an excellent day for diving, a calm sea with visibility of 6–9 metres, water temperature 22°C and a slight breeze. The boat and hookah equipment, which was owned by the victim, was thoroughly checked by him before the dives. His wetsuit was very tight, and he required considerable assistance to put it on. While one of his friends remained in the boat as an observer, the other two dived twice to hunt crayfish. After about 15 minutes into the second dive, the buddy experienced some pain in his ears and temple, and felt it was time to end the dive. They ascended slowly and at the surface swam side by side towards the boat, three to four metres away. When the buddy reached the boat he looked round but could not see his friend, then looked down and saw him lying on the sea floor with no bubbles coming from his regulator. The buddy, who had experienced no problem with his air supply, immediately swam down and with difficulty removed and ditched the victim's harness and vest before bringing him to the surface. He was pulled into the boat and resuscitation started but there was no response. They returned to land, which took about half an hour. There, the non-diver of the trio landed and went for assistance, but after walking a short distance he began to have breathing difficulty, collapsed and also died despite resuscitation attempts by off-duty ambulance officers.

*Autopsy:* The victim was obese (132 kg; BMI 38 $kg.m^{-2}$) and had emphysema with at least one large bulla and others at the apex of his left lung, but there was no evidence of pulmonary barotrauma. A carboxyhaemoglobin level was 2%. There were severe fatty changes in his liver, moderate coronary atheroma, and minor ischaemic cardiac fibrosis. The cause of death was given as drowning.

*Comment:* The tight wetsuit may have increased the effort expended during the dives and with his other medical problems have contributed to impaired pulmonary gas exchange. His buddy cannot have been unaware of the victim's lack of fitness but obviously was not concerned by this. It is possible that he suffered a fatal cardiac arrhythmia as he swam towards the boat, becoming rapidly incapacitated and incapable of calling for help before drowning. There is no evidence of any equipment factor, and the water conditions did not cause any problems for either diver.

*Summary:* SURFACE-SUPPLY BREATHING APPARATUS; EXPERIENCED; OBESITY; EMPHYSEMA WITH BULLAE AT APEX LEFT LUNG; MINOR MYOCARDIAL ISCHAEMIC CHANGES; TIGHT WETSUIT; POSSIBLE ACUTE CARDIAC DEATH; COLLATERAL DEATH NON-DIVER CREWMAN; DROWNING.

CASE HH 02/02

This 23-year-old professional scallop fisherman was diving turn-and-turn-about with his assistant, in about 10 msw. About five to ten minutes into the dive, whilst he was sorting the catch on board the boat, the assistant heard the victim yelling. He had not seen him surface, but "*knew something was wrong so kicked the motors over and put it in gear*" and started to motor to him, 50–100 metres away. He then saw a white pointer shark, "*it was enormous, the size of the boat* [6.5 metres]", and witnessed it attack almost immediately, taking the unfortunate diver in its jaws, shaking its head and thrashing him around. He hit the shark with the side of the boat and managed to pull his friend into the boat, receiving electric shocks from a 'shark pod' carried by the diver as he did so. The victim died soon after. The boat's VHF radio was defective and their mobile phone had a flat battery, so the assistant had to ask some fishermen he met as he was returning to port to radio ahead to warn others of the danger.

*Autopsy:* The autopsy detailed the amputation of the victim's right lower limb at the hip joint and the multiple irregular incised bite marks on the buttocks and upper left leg.

*Comment:* Blood loss and shock from massive trauma made this an inevitable fatality. Examination of the victim's 'shark pod' showed that it had been incorrectly applied, though in a manner probably common amongst users. The apparatus produces an electrical field that impacts on the shark's receptors, the Ampullae of Lorenzi, causing it discomfort, then muscle spasm if it comes too close. The pod has three components – a battery with an electrode worn on the air cylinder or back of the buoyancy vest, a second electrode worn on one fin, and a hand switch which comes over the diver's shoulder and is attached to the front of the buoyancy vest. For maximum protection it should be switched on whilst in the water, but as the perceived risk is minimal while the diver is working close to the sea bed, only arising as the diver is ascending and while at the surface, it is common practice for divers to switch it off while on the sea bed, avoiding electrical shocks to the wearer. However, on this occasion the fin electrode had been placed on the air hose attached by a float and as the hose is flexible the two electrodes can become too close to exert an effective deterrent field when the diver is at the surface – the time this shark attacked. Police examination showed the 'pod' to work well if correctly worn. It is probable the shark became interested in the diver while he worked with the pod turned off. This may have resulted in him switching it back on and then starting to ascend, the field deterring the shark till he reached the surface and one electrode rose out of the water. Electrodes should be at least 1.5 metres apart.

*Summary:* SURFACE SUPPLY BREATHING APPARATUS; PROFESSIONAL DIVER; 'SHARK POD' INCORRECTLY POSITIONED; WHITE POINTER SHARK ATTACK; MASSIVE TRAUMA.

CASE HH 02/03

This 43-year-old diver was surfacing from a crayfishing dive with his buddy when he was hit by the propeller of a boat and suffered severe head trauma. The injury left him in a vegetative state from which he never recovered, and he died 11 months later. A 'diver down' flag was displayed on the dive boat, but it is unclear whether the hookah hose extended beyond the 50 m (in Western Australia) 'no go' zone. Despite an intensive search, the boat involved was not located. However, it is unlikely the boat's occupants remained unaware of the event as details were widely publicised. Due to the long interval between the incident and the diver's death, the normal coronial investigation for a diving-related fatality was not instituted. Details of the incident are still lacking but continue to be sought.

*Summary:* SURFACE-SUPPLY BREATHING APPARATUS; MAJOR HEAD TRAUMA FROM BOAT PROPELLER; BOAT NOT IDENTIFIED; COMA FOR ELEVEN MONTHS BEFORE DEATH.

**Discussion**

The safest way to learn about dangerous situations and their avoidance is through an examination and understanding of what has befallen others, death being the worst endpoint of any misadventure. Despite the unsuitability of the underwater environment for any air-breathing creature, it can be successfully entered if the necessary conditions are met. It is the purpose of training, the development and correct use of appropriate, well-designed equipment, and an adequate understanding of potential medical problems and their management to reduce the risks of diving to acceptable levels. It is only through constant measurement of reality against theory and assumptions that we can improve safety.

Understanding of complex problems is a dynamic process that challenges accepted beliefs with the evidence of new data. However, there are particular problems in the matter of diver safety in accessing the validity and weight of data offered by persons involved in a serious incident, such as a fatality. There will inevitably be a tendency on the part of witnesses to present a simple and 'clean' report on the events, one limited to satisfying the investigator's area of interest. This may leave unexplored matters that appear peripheral to the main focus of the coronial investigation – for example, the medical history of the victim – often as a consequence of the difficulty in obtaining this information from the family. The clinical significance of a medical condition may be overlooked, or the absence of a 'medical history' may not be an accurate representation of the facts.

SNORKELLERS

Though buddy separation frequently occurred before the fatal cascade of events, this may not have been the critical element in most of the snorkelling incidents. Water power was a probable factor in three cases, either close to rocks or, as in the case of BH 02/10, in the form of a rip current. Only one case of ascent hypoxia from a deep breath-hold dive was reported (BH 02/11). In the two snorkellers who died from box jellyfish envenomation, it is possible that there might have been a better outcome but for their underlying medical conditions. As a result of these and other such incidents, the Queensland Irukandji Taskforce has recently developed guidelines for the emergency management of the Irukandji syndrome.[10]

As in previous years, cardiovascular factors were common, though sometimes these were accepted as the likely cause of death rather than being proven to be so. This may be a reflection of the age range of the majority of those who died. Of the 17 cases, 14 were over 40 years of age, the eldest being 82 years old. Of special interest to those running commercial trips to the Great Barrier Reef is that 12 of the 17 snorkellers who died were middle-aged or elderly overseas visitors (average age 69 years), and unsuspected cardiac factors are believed to have been critical in seven of these, with a CVA consequent to hypertension in another. It is difficult to see how anyone could give an accurate prediction of the safety of swimming to someone with a cardiac problem but who has no known symptoms, except by unjustifiably restricting the activities of the many who are now correctly advised to undertake exercise. The continued occurrence of unobserved deaths in supervised swimming areas appears to confirm the silent nature of many such deaths but remains a potential concern regarding the techniques of supervision adopted in this industry. Unfortunately the wearing of a life vest does not prevent drowning, as cases BH 02/05, BH 02/06 and BH 02/12 demonstrate.

SCUBA DIVERS

One unexpected finding has been the frequency of health problems (10 out of 13 divers), often unsuspected, in this group of apparently healthy people undertaking scuba diving, an activity with energy demands that may be severe in an emergency situation. This is consistent with the recent report on New Zealand diving fatalities.[11] However, in only one instance (SC 02/1) had the person disregarded a specific warning from a cardiologist not to dive. There were three instances (SC 02/2, SC 02/06, SC 02/09) in which symptoms worth further investigation were, unfortunately, not reported to a doctor, but in the remainder the health problems appear to have remained occult. In one case (SC 02/10) a specialist had legitimately made the decision that diving was allowable, illustrating the problem of predicting the future. Whether a person liable to panic (SC 02/05) should dive is another area problematic to enforcement.

Although two had a history of asthma there is nothing to suggest that this played any part in their deaths. It is difficult to see how fatalities can be reduced other than by a strict adherence to generally promoted safe diving protocols

and an increased awareness among the diving community of the value in seeking informed medical advice on any health problems they may experience. A bronchogenic cyst constitutes a 'classic' but a rare risk factor – one insufficiently common to justify a return to routine chest X-ray as a requisite to medical clearance for dive training.

HOOKAH DIVERS

These three cases illustrate different, potentially dangerous situations. In two, the critical factors implicated were known and accepted by the victims, though they both failed to recognise their significance. In the first case it is likely that the victim was undoubtedly aware of his lack of physical fitness, but both he and his buddy failed to recognise the serious health risk this factor constituted. In the second case the shark attack might not have occurred had the deterrent equipment been worn as advised by the manufacturer. This diver was unfortunate that an apparently common practice in how the 'shark pod' was worn and used left him exposed to risk at the surface. The manufacturers of such equipment should consider how to address the problem wearers experience from shocks at the surface. The third death indicated that it is not always possible to escape the actions of others who ignore safety rules.

## Conclusions

It is difficult to see how the occurrence of deaths among visitors swimming with snorkels on the GBR could be reduced. There is now a real awareness among the commercial firms providing this service of the need to provide safety advice, a watch over those in the water, and offer buoyancy aids. However, a significant proportion of their clients are carrying cardiovascular risk factors and it would appear unacceptable to have a compulsory age cut-off for allowing them to snorkel swim. Water power is more likely to be a factor in non-commercial diving situations. Reiteration of the potential dangers of breath-hold diving can be difficult in the special breed of divers seeking to set greater depth records.

Although only one scuba diver received, and ignored, medical advice not to dive, two others unfortunately failed to attend a doctor to discuss their symptoms. The incidence of cardiovascular factors is likely to remain a problem as they are often unrecognised by the victim, and the value of regular age-related medical fitness checks is debatable. A continued emphasis on training, experience, and awareness of the need to dive within one's ability, remains a necessity.

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# The effect of two consecutive dives on bubble formation and endothelial function in rats

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**Key words**
Decompression sickness, endothelium, bubbles, hyperbaric research

**Abstract**
(Havnes MB, Møllerløkken A, Brubakk AO. The effect of two consecutive dives on bubble formation and endothelial function in rats. *Diving and Hyperbaric Medicine*. 2008; 38: 29-32.)
**Introduction:** Gas bubble formation during and after decompression is considered to be the main initiator of decompression sickness (DCS). Compressed-air workers have been reported to acclimatise to the working environment and hence have a reduced risk of DCS, but the exact nature of the adaptation is not known. In the present study, we investigated the effect of two consecutive dives, separated by a 24-hour surface interval, on bubble formation and endothelial damage in rats.
**Methods:** A total of 30 rats were divided into four groups, one control group and three dive groups with different dive profiles, of which two of the groups had two dives. The amount of bubbles in the pulmonary artery was estimated by ultrasound for one hour after surfacing, and tension measurements were performed *in vitro* on segments of the abdominal aorta following sacrifice of the animals.
**Results:** No significant differences between the groups were found in endothelial function or bubble grade. However, animals that died immediately after the dive, irrespective of grouping order, had lower acetylcholine-induced dilatory responses in the aorta than surviving rats.
**Conclusion:** Bubble formation and endothelial function among rats were not significantly affected by exposure to consecutive dives 24 hours apart. An adaptive, protective effect of repeated dives was hence not seen in this animal model.

## Introduction

Injuries to the organism related to decompression sickness (DCS) are caused by gas bubbles, which are believed to originate from pre-formed bubble-nuclei during decompression.[1] Gas bubbles formed during decompression can lead to mechanical damage of the endothelium or even stripping of endothelial cells.[2]

Repeated dives have generally been associated with increased risk of DCS, due to cumulative upload of nitrogen ($N_2$).[3,4] There are different opinions on the effects of repetitive diving and how to minimize the risk of DCS in such dives. Divers and caisson workers have reported increased tolerance to DCS with daily pressure exposures, which decreases after a few weeks' layoff. This observation has been confirmed in controlled studies.[5, 6]

Different explanations for adaptation or acclimatization to hyperbaric conditions range from increased resistance to DCS through repeated exposures, to different populations of gas nuclei being eliminated at specific pressures and loss of adaptation when nuclei re-accumulate.[4–7] The exact nature of adaptation or acclimatization is not yet known. Hence, the present study was initiated to determine in rats the effect of two consecutive dives, separated by a 24-hour surface interval, on bubble formation and on endothelial function.

## Methods

A total of 32 female Sprague-Dawley albino rats (Kirkeby, Sweden) were selected, but two were excluded for technical reasons; the remaining 30 animals, weighing 321.1 ± 21.8 g, were used in the experiment. All experimental procedures and the care of the experimental animals conformed to the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes, and the protocol was approved by the Norwegian Council for Animal Research.

### PRESSURE PROFILE

Following one week of acclimatization, the rats were randomly assigned to one of four groups (A, B, C or D), where three of the groups (B, C & D) were exposed to different compression profiles (Table 1). The compressions were performed in a 20 L hyperbaric chamber with continuous air supply. Group A was a non-diving control group. Group B was observed for one hour with ultrasound after surfacing to detect bubbles and then sacrificed. Groups C and D underwent the first of two compressions, were observed for one hour and then rested for 24 hours before undergoing a second compression. After the second dive, the rats in groups C and D that did not die were again observed for one hour with ultrasound, before they were sacrificed.

### BUBBLE DETECTION

Immediately after surfacing, the rats were anaesthetised with a 2 ml per kg bodyweight injection of haloperidol (0.7 $mg.ml^{-1}$), fentanyl (0.01 $mg.ml^{-1}$) and midazolam (1.07 $mg.ml^{-1}$) s.c. The pulmonary artery was monitored for gas bubbles using a 10 MHz transducer connected to a GE Vingmed System Five ultrasound scanner (GE Vingmed,

**Table 1**
**The compression and decompression rates and depth of the dive profiles together with the observation period. The two dives in groups C and D were separated by a 24 hr surface interval. *The rats that died after the dive did not have an hour observation period**

| Group (n) | Compression rate kPa.min$^{-1}$ | Decompression rate kPa.min$^{-1}$ | Dive depth kPa | Observation period hr* |
|---|---|---|---|---|
| **Group A** (8) | 0 | 0 | 0 | 0 |
| **Group B** (7) | 200 | 700 | 50 | 1 |
| **Group C** (8) | 200 | 400 + 700 | 50 | 1 |
| **Group D** (7) | 200 | 550 + 700 | 50 | 1 |

Horten, Norway). Bubbles were seen as bright spots, and verified with Doppler. The amount of bubbles was graded using a six-level grading scale described previously.[8]

TENSION MEASUREMENTS

Following the observation period, each live rat was sacrificed, or if the rat died early post-dive, the abdominal aorta was carefully dissected out and placed in an aerated (5% $CO_2$, 95% $O_2$) sodium-potassium buffer (139 mM) of the following composition: $Na^+$ 139 mM, $K^+$ 4.6 mM, $Mg^{2+}$ 1.2 mM, $Cl^-$ 134 mM, $HCO_3^-$ 15 mM, $H_2PO_4^-$ 1.2 mM, $Ca^{2+}$ 1.5 mM and glucose 11 mM (Sigma-Aldrich).

The tension in the abdominal aorta was measured using a myograph and PowerLab™ data system (Danish Myo Technology, Chart™ software, ADInstruments, Oxfordshire, England) as described previously.[9] The myograph measures the force (in milliNewton, mN) generated in the muscles during contraction caused by exposure to agonists. The incoming signals were digitised and displayed real-time on computer. After calibration of the myograph, three cylindrical segments (1.5–2.5 mm) of the abdominal aorta were mounted on two parallel L-shaped metal prongs in organ baths filled with the sodium-potassium buffer. A tension of 0.7–0.8 gram was gradually applied to the segments before they were allowed to stabilise for half an hour.

The contractile capacity of each vessel was examined by alternate exposure to a potassium-rich (60 mM) buffer solution ($Na^+$ 84 mM, $K^+$ 60 mM, $Mg^{2+}$ 1.2 mM, $Cl^-$ 133 mM, $HCO_3^-$ 15 mM, $H_2PO_4^-$ 1.2 mM, $Ca^{2+}$ 1.5 mM and glucose 11 mM) and to a sodium-potassium buffer. The vessels were precontracted in the bath with cumulative doses of nor-adrenaline (NA) until a stable level (70–100% of the response to potassium) was reached. After 30 minutes, cumulative doses of acetylcholine (ACh) were added in increments ($10^{-8}$–$10^{-3}$ M, Sigma-Aldrich). The relaxation response that followed was assumed to depend on how much the endothelium function was affected by the bubbles. The relaxation response was also examined with cumulative doses of substance P (SP) ($10^{-11}$–$10^{-6}$ M; Sigma-Aldrich) after a new precontraction of NA. The performance of the smooth muscle layer was examined with cumulative doses of sodium nitroprusside (SNP, $10^{-8}$–$10^{-4}$ M). Dose-response curves with all agonists were obtained. The resultant values are relative relaxation percentages, calculated using the baseline tension and the precontraction values as reference points and are presented with the maximal relaxation response to the agonists (Imax). In addition, $EC_{50}$ was calculated, which is defined as the concentration of the agonist that leads to 50% of the total relaxation.

STATISTICAL ANALYSIS

The results are presented as means ± SD. Non-parametric statistical methods were used due to the small number of animals in each group. Kruskall-Wallis test was performed to assess differences between all the groups. Further investigation of differences between the groups was achieved using Mann-Whitney and the Wilcoxon signed-rank tests for unpaired data. The relationship between death/survival and Imax of ACh was also calculated using Mann-Whitney and the Wilcoxon signed-rank tests. The level of statistical significance was set at $P < 0.05$. All statistical analyses were performed using SPSS 13.0.

## Results

The survival rate of the rats varied between the groups (Table 2), but the differences were not statistically significant. No significant correlation was found between survival and body weight.

**Table 2**
**The numbers of animals that died or survived; all animals in Groups C and D survived the first dive. Imax of acetylcholine (Ach) presented as mean ± SD; *one animal in each of groups B and D excluded for technical reasons; † P = 0.048**

| Group (n) | Dive outcome | |
|---|---|---|
| | Died | Survived |
| **Group A** (8) | n/a | n/a |
| **Group B** (7)* | 4 | 3 |
| **Group C** (8) | 5 | 3 |
| **Group D** (7)* | 2 | 5 |
| **Imax (ACh)**† | 43.33 ± 21.86 | 65.52 ± 23.11 |

**Table 3**
**Dilatation response in abdominal aorta presented as relative percentages as defined by precontraction triggered by NA and baseline tension (Imax). In addition $EC_{50}$ values are presented. Agonists used to trigger relaxation were acetylcholine (ACh), substance P (SP) and sodium nitroprusside (SNP). Data presented as mean ± SD. $EC_{50}$ values are the concentrations ($^{-log}$M) of the agonists that lead to 50% of the total relaxation**

| Group (n) | ACh | | SP | | SNP | |
|---|---|---|---|---|---|---|
| | Imax (%) | $EC_{50}$ ($^{-log}$M) | Imax (%) | $EC_{50}$ ($^{-log}$M) | Imax (%) | $EC_{50}$($^{-log}$M) |
| **Group A** (8) | 47.15 ± 18.66 | 6.08 ± 0.61 | 9.70 ± 13.17 | 7.88 ± 1.20 | 80.50 ± 21.77 | 5.17± 0.23 |
| **Group B** (7) | 57.61 ± 22.26 | 5.73 ± 0.42 | 22.72 ± 21.10 | 8.28 ± 1.86 | 73.48 ± 41.63 | 4.84 ± 0.20 |
| **Group C** (8) | 54.08 ± 28.53 | 6.23 ± 0.41 | 13.61 ± 9.34 | 9.38 ± 0.82 | 100.39 ± 67.22 | 4.92 ± 0.19 |
| **Group D** (7) | 51.63 ± 25.91 | 5.99 ± 0.42 | 14.15 ± 22.40 | 9.54 ± 0.76 | 84.44 ± 22.31 | 5.04 ± 0.28 |

BUBBLE DETECTION

The bubble grade varied from 0 to 5 within all the dive groups. In group D, two out of seven animals had bubble grade 5, while in group B four out of seven had grade 5 and in group C, five out of eight. This difference was not significant at the 5% level. All the rats that died immediately after the dive had bubble grade 5. In the present study there were no significant differences in bubble formation related to weight (P = 0.207).

TENSION MEASUREMENTS

There were no significant differences in the *in vitro* relaxation response of the abdominal aorta between the four groups (Table 3). However, animals in groups B, C and D that survived the observation period had a significantly higher maximal dilatory response in the abdominal aorta induced by ACh (Imax (ACh)) compared with animals that died immediately after the dive (P = 0.04, Table 2). Sensitivity to the agonists was tested by calculation of $EC_{50}$ values for the agonists. There were no significant changes in sensitivity to the agonists in any of the groups.

## Discussion

An impaired endothelial response (Imax) to ACh was found in animals that died immediately following a dive compared with animals that survived, but no differences were found between the four groups in bubble formation and endothelial function. Thus, two consecutive dives separated by 24 hours did not lead to any adaptation regarding tolerance to decompression stress. However, a higher bubble grade was observed in non-survivors compared with those who survived the entire observation period. Nishi found an increased risk of developing serious DCS when a large number of bubbles were detected in the vascular system of humans.[10] Previous research at our laboratory has shown a relationship between gas bubbles and mechanical endothelial damage and that the damage seems to be related to the amount of bubbles and not to the duration of exposure.[11] This is in accordance with the results of the present study.

The degree of relaxation varied within the groups, but the response to the endothelium-independent agonist SNP seemed unaffected by the dive and the vascular bubbles. Thus, this result confirms that the change in vasoactive response is related only to endothelial function and not to the function in the vascular smooth muscle layer.

It might be that the difference found in endothelial function is somehow influenced by the survival rate itself, due to severe hypoxia. However, from the experimental design of this study, this remains as speculation. In all of the animals that did not survive the entire observation period, the abdominal aorta was dissected out within 10 minutes. The endothelial measurements were also performed in isolated organ baths allowing for the exclusion of any influence from higher regulatory systems. Although possible, we consider death in itself as unlikely to be the cause of the difference in endothelial measurements.

The survival rate of the rats varied between the groups, but the differences were not statistically significant. It is well known that there is a significant variability in bubble formation among individuals. While the mechanisms that cause individual differences in susceptibility to DCS are unknown, body weight has been regarded as a predisposing factor for bubble formation.[12] Broome et al, however, state that weight is not a risk factor for DCS, but rather a supplementary factor in sedentary animals.[13] They found no reduction in DCS incidence in lighter animals compared with heavier animals. A study by Carturan supports these findings.[14] In the present study weight was significantly related neither to survival (P = 0.14) nor bubble formation (P = 0.207).

Although not significantly different, there was a trend towards reduced bubble production in group D compared with the other dive groups. Group D had two rats with bubble grade 5, while group B had four and group C five rats with the same bubble grade. Group B had only a single dive, unlike groups C and D. The pressure difference in the first dive between group C and D was 150 kPa with the same bottom time. Although too early to draw any conclusions from

this observation, it is tempting to speculate that if previous exposure to pressure does have any 'protective' effect, the pressure has to be above a certain level.

The present study examined if a prior dive had any effect on bubble formation and endothelial function in a second dive performed 24 hours later. There were no significant differences between the groups with regard to either of the two parameters, but an impaired endothelial response to ACh was found in the animals that died compared with the ones that survived.

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# The consequences of misinterpreting dive computers: three case studies

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**Key words**
Case studies, dive computers, models, decompression sickness, recreational diving, diving research

**Abstract**

(Sayer MDJ, Wilson CM, Laden G, Lonsdale P. The consequences of misinterpreting dive computers: three case studies. *Diving and Hyperbaric Medicine*. 2008; 38: 33-9.)
Three cases are presented where there is a direct link between how the divers used their dive computers and the eventual requirement for their therapeutic recompression. The first case involves a diver with a previous history of decompression incidents making adjustments to their dive computer without understanding the outcomes of those alterations. The second case involves two divers running out of air and surfacing having missed significant amounts of decompression, caused by the dive computer not reducing their decompression obligation in actual time. This effect and performance differences between three models of computers were demonstrated in subsequent compression chamber trials reported here. The final case involves a diver who completed their dive within the indicated limits of their dive computer but subsequently developed serious neurological decompression sickness that left severe permanent residua. Compression chamber trials suggested that a combination of poor measurement accuracy and outdated decompression management in the computer used could have contributed to the diver's eventual poor outcome.

## Introduction

For a number of decades, dive computers that calculate and display decompression information for divers have been evolving in their accuracy, complexity and the range of information being manipulated. External features such as gas use, heart rate and gas-mixture changes can now be monitored remotely, and the use of closed- or open-circuit breathing systems included in their calculations.[1–4] These technological advances mean that dive computers are now capable of delivering high-quality information for a diversity of uses.[5–7] In recreational diving, their use is widespread and in this and other diving sectors that predominantly use dive computers, such as scientific diving, decompression illness rates are among the lowest that have been published.[8–13] However, dive computer information may sometimes be open to misinterpretation. Three cases are presented where the misuse of dive computers may have directly contributed to the onset and severity of decompression sickness. Dive computer manufacturers rarely publish technical information sufficient to understand fully how some computers function, and so, in two of the reported cases, compression chamber trials were employed to study their performances.

## Case studies

### CASE 1

A 33-year-old, female, advanced open-water diver was diving using a *Suunto Vytec*™ dive computer. She completed six dives, two dives a day, maximum depths ranging from 16–27 metres' sea water (msw) and total dive times from 50–55 minutes with surface intervals approximating 2.25 hr. Dive three of the series was to a depth of 24.4 msw for a total time of 53 minutes (Figure 1). The download of that dive showed that the computer went into decompression mode after 21 minutes; the divers ascended from 17 msw 42 minutes into the dive. The ascent was notable for two registered ascent rate warnings, a violation of depth ceiling and a recording of the computer being switched into compass mode. On surfacing, incomplete decompression had been undertaken and the computer locked out into gauge mode (i.e., displayed depth/time information only). An error message was displayed in the form of the letters "Er" on the screen. The divers were confused about this because her buddy's computer had cleared of any decompression obligation on surfacing. None of the dive party understood the relevance of the "Er" display. An attempt was made to unlock the computer by hanging it on a shotline during the surface interval. However, the computer remained in gauge mode and so for her subsequent three dives she used a *Suunto Gecko* computer which had not been dived that week. The *Suunto Gecko* does not have a PC download facility.

On the third diving day, about two hours after her sixth dive, she reported symptoms of probable decompression sickness (DCS). She was transferred to the Dunstaffnage Hyperbaric Unit (DHU) where initial examination showed weakness in the left elbow, poor heel-toe walking and a pronounced unsteady tandem Romberg test. Recompression on an extended Royal Navy Treatment Table 62 (RN 62) started within seven hours of surfacing, and at the end of the treatment she appeared well. She was transferred to Oban Hospital for observation. Later the following day she deteriorated, with pain and weakness in the left arm and shoulder, and her walking and balance were unsteady. Despite three further treatments, complicated by symptoms of pulmonary oxygen toxicity, she became more ataxic

**Figure 1**
**The depth/time profile of the third dive of Case 1. The download shows the computer in decompression after approximately 21 minutes, rapid ascent warnings at 43–44 and 53 minutes, a depth ceiling violation at 47 minutes, a switch to compass mode at 49 minutes and surfacing at 53–54 minutes**

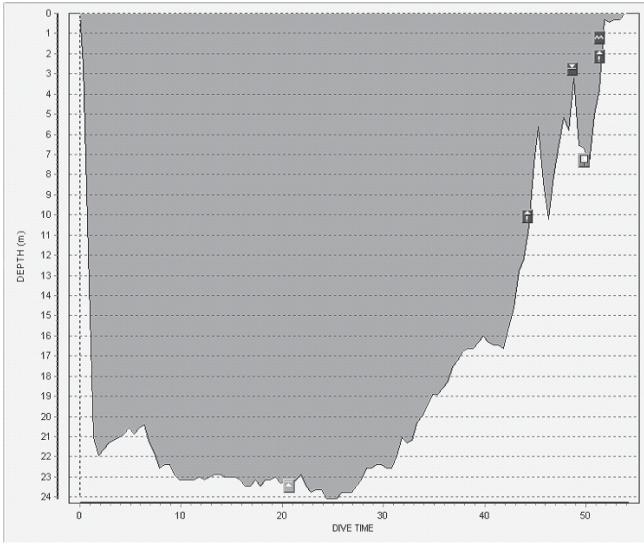
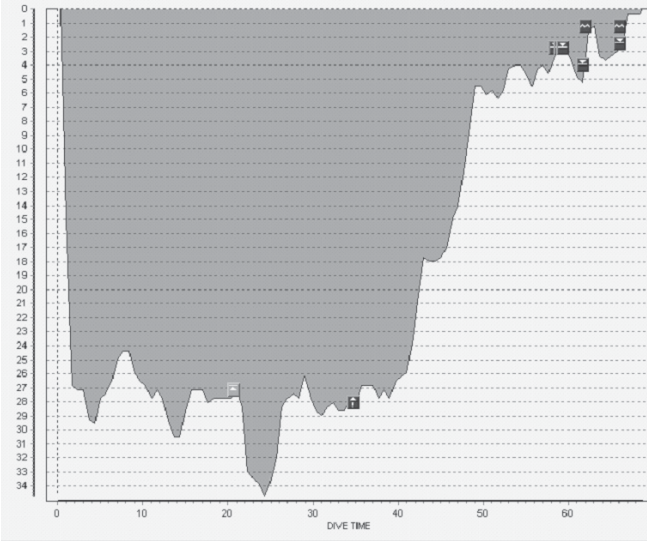



**Figure 2**
**The depth/time profile of the sixth dive of Case 2. The download shows the computer in decompression after approximately 21 minutes, rapid ascent warnings at 33–34 minutes, numerous depth ceiling violations between 57 and 65 minutes, and surfacing at 61 and 66 minutes**




and was transferred to the Aberdeen Hyperbaric Unit for further specialist care. In Aberdeen, she received three daily Comex 12 (222 kPa) treatments with some improvement after the first treatment but little after that. An MRI of the brain was normal and, 12 days after the dive incident, she was referred for further rehabilitation to the Glasgow Brain Injury Centre.

During her treatment she reported that two years previously she had been treated for DCS at another Scottish chamber (Orkney). In the month following that treatment, she had an episode of suspected cutaneous DCS that went untreated. Subsequent investigation showed a patent foramen ovale (PFO), which was closed successfully and she returned to diving 15 months later. She purchased the *Suunto Vytec* computer with a view to adjusting its settings to make her decompression management more conservative.

The *Suunto Vytec* is a relatively advanced, new-generation dive computer that can be operated in air, nitrox or gauge modes. There is a facility to switch gases (up to 3 mixes, any one ranging from 21–99% oxygen content), optional wireless pressure transmission, extensive memory functions and a built-in dive simulator. The *Vytec* employs an adjustable *Suunto*-modified reduced gradient bubble model (RGBM) and is PC interface compatible. It is programmed with eight diver-adjustable settings that can be altered singly or in combination to produce many levels of added conservatism, three for altitude, three for personal conservatism and either the full capacity of the RGBM (RGB100) or reduced power (RGB50). In this case, the download indicated that her computer had been set to altitude setting A2 (1,500–3,000 m) and RGB50, but remained at the default personal setting of P0. It was, therefore, unsurprising that on the third dive the altered *Suunto Vytec* had indicated a higher decompression requirement than the dive leader's unmodified computer. The computer had done what it had been programmed to do and produced a more conservative dive profile. However, this was either ignored or not understood, or the consequences of alterations had been forgotten. Further, this was only part of the problem. The decision to use another, undived computer resulted in her basing subsequent decompression management on a computer with no existing nitrogen loading and, possibly, in an unmodified mode.

CASE 2

A 16-year-old, male, advanced open-water diver was using a *Suunto Vyper*™ dive computer. The *Suunto Vyper* carries fewer functions than the *Vytec* but still has air/nitrox/gauge modes and a PC interface, and is controlled using the *Suunto* RGBM decompression model. The diver undertook six dives in three days, with maximum depths ranging from 20–35 msw, total times ranging from 24–68 minutes and with surface intervals of 2–5 hr. The sixth dive was to a maximum depth of 35 msw (Figure 2). For the first 21 minutes of the dive, the maximum depth ranged from 25–30 msw. The computer went into decompression mode after 21 minutes. However, the divers remained at depth and, in fact, reached their maximum depth of 35 msw after 24 minutes. They initiated their final ascent after 40 minutes. At that stage, the download indicated a total ascent time of 29 minutes

**Figure 3**
**Download of the incident dive from the *Buddy Nexus* computer used in Case 3**


with a first decompression stop at 5.3 msw. Their ascent was slow and complicated by undertaking a stop for one minute at 18 msw; it took them just over eight minutes to ascend to 5 msw. This slow ascent meant that, on reaching 5 msw, a remaining surfacing time of 29 minutes was still being indicated. Whereas a total dive time of 69 minutes was indicated when the ascent was initiated, on reaching 5 msw, the total dive time was now 78 minutes. The divers attempted to complete the decompression indicated, but had trouble maintaining a constant depth. As a result, by 61 minutes into the dive they still required 21 minutes of decompression. However, they were both running out of air and decided to surface. As they both had 30 bar air left, their group advised them to return to their decompression depth until they had completely run out of air. This they did, but ran out after only six minutes and re-surfaced; the computer locked out, indicating missed decompression; in total, 24 minutes of decompression had been omitted. After surfacing, both divers developed tingling in their lower limbs. They were placed on oxygen and transferred to DHU by lifeboat. They both received an unmodified RN 62 recompression treatment and remained symptom free after surfacing.

CASE 3

A 39-year-old male, described as an "experienced" open-water diver had recently bought a *Buddy Nexus*™ dive computer in order to start mixed-gas diving. At the time, the *Buddy Nexus* was being marketed as "*the first affordable, multi-mode dive computer designed for the full span of your diving career...*". The *Buddy Nexus* supports a number of diving modes: open-circuit sport (conventional scuba with air); open-circuit technical (nitrox, with up to two mixes); or closed-circuit rebreather (CCR) with a constant inspired oxygen partial pressure ($PPO_2$). In this case, the diver intended to perform a dive with the computer believed to be set in open-circuit air mode. According to his computer, the diver dived to a maximum depth of 48.7 msw for a total dive time of 50 minutes, breathing air throughout (Figure 3). He completed all decompression stops advised by the computer, but experienced some back pain during ascent. This was eased by breathing oxygen on the boat. Upon returning ashore, he went to his mother's house nearby and fell asleep exhausted. When he awoke he was unable to stand or pass urine. Diving friends took him to the local emergency department, where they had to carry him in. The relationship of his symptoms to a diving incident was not recognised for several hours, but eventually he was transferred to Hull Hyperbaric Unit where he received a series of treatments over several days. Prior to this accident, he was a fit firefighter. One year after the incident, he had been unable to return to his previous employment and was paraparetic, with both motor and sensory changes in the legs, and bladder and bowel dysfunction. A more detailed account of this case is given by Walker and Laden.[14]

**Chamber compression trials**

Case 1 required no further investigation as it was clear that the error lay primarily with a misunderstanding of the computer response to the adjustments made. Cases 2 and 3 did require investigation, Case 2 because of inconsistencies in the surfacing times and Case 3 because of the severe outcome caused by a seemingly well-controlled dive.

CASE 2

Three models of dive computer were compared: the *UWATEC Aladin Ultra Pro*™ (an older generation dive computer based on Bühlmann algorithms derived from the Haldane/Spencer tissue compartment principles); the *Mares Nemo*™ (a modern-generation, deep-stopping RGBM-controlled computer); and the *Suunto Vytec*. They were immersed in water in a clear perspex™ tank within the DHU compression chamber to permit direct observation. Where adjustable, the computers were set to their default, least conservative settings.

*Trial 1*

The previously undived computers were subjected to an approximate simulation of the dive profile of the incident sixth dive in case 2. All three models went into decompression and registered maximum decompression obligations of between 30 and 40 minutes (Figure 4). The simulated profile of Case 2, dive six was allowed to run during the decompression phases of the three models of computer in order to record when they cleared their respective decompression obligations. The *UWATEC Aladin Ultra Pro* cleared after 80 minutes, the *Suunto Vytec* after 90 minutes and the *Mares Nemo* after 93 minutes.

**Figure 4**
**Three models of dive computer were subjected to a single compression profile designed to mimic the incident profile of Case 2 (light grey profile). The y-axis denotes the displayed decompression information of the respective computers; positive values are times remaining before entering decompression; negative values are the decompression times required; values over 99 minutes denote that the computer is clear of any decompression obligation**

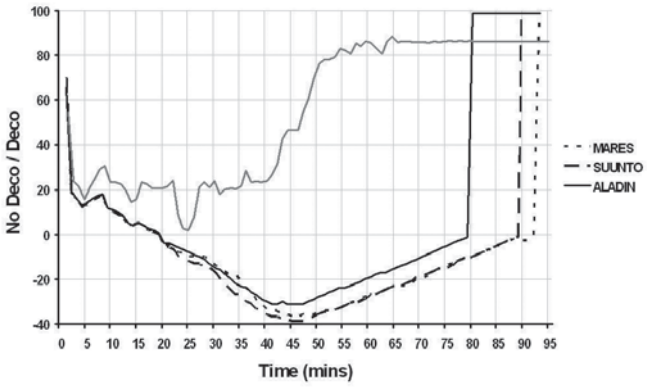
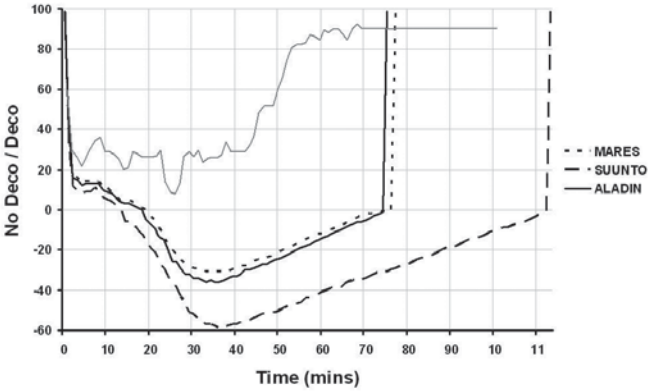



**Figure 5**
**Three models of dive computer were subjected to a compression profile designed to mimic the incident profile of Case 2 (light grey profile) but after 5 other chamber dives over the preceding 3 days. The y-axis denotes the displayed decompression information of the respective computers; positive values are times remaining before entering decompression; negative values are the decompression times required; values over 99 minutes denote that the computer is clear of any decompression obligation**




*Trial 2*

Because the sixth dive had occurred as part of a series, the simulated chamber profile was repeated following five chamber compressions with similar depths, times and surface intervals as indicated from the case downloads. With this gas loading, the decompression profiles of the sixth dive now differed markedly (Figure 5), with the *Suunto*, in particular, accumulating significant decompression obligations. Whereas the *Mares* and *Aladin* accrued decompression requirements of 30–40 minutes and surfaced at about 75–80 minutes, the *Suunto* had a maximum of almost 60 minutes of decompression to carry out and surfaced at 115 minutes (Figure 5).

Of additional note was the rate at which the decompression obligations reduced. From the start of the ascent to surfacing almost 200% in actual against estimated time was taken for the two RGBM models and 150% for the *Aladin* (Table 1). Taken from the time at which the profile reached 6 metres, the RGBM computers took 130% of their indicated time to surface; the *Aladin* about 110%.

Finally, some computer-controlled reduction in decompression can operate on threshold depths. In Figure 6, the same three models of computers described above were held at depths fluctuating between 9 and 11 msw. Where this is a threshold depth range, the information given by some computers ranged wildly and caused large differences in apparent decompression requirements (Figure 6). In a similar way, some dive computers will reduce the decompression in relation to the ascent profile; others will not count down until the threshold depth of the decompression stop is either close or exceeded.

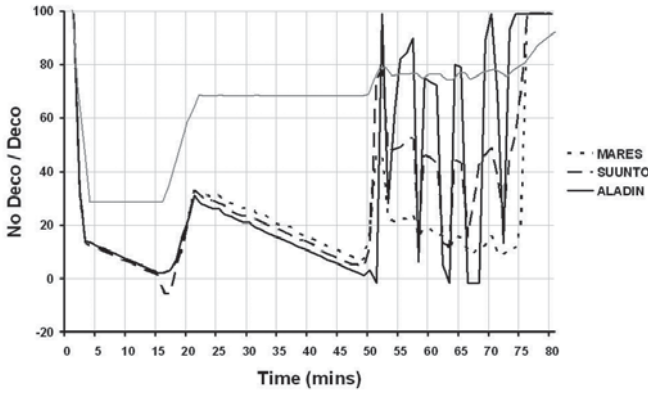
CASE 3

The features of the *Buddy Nexus* dive computer were

**Table 1**
**Reduction rates of displayed decompression times recorded in compression chamber trials for three models of dive computer. Rates are calculated as the display decompression time divided by actual time; a value of 1.00 would be returned if display equalled actual decompression time**

| Computer make/model | Decompression theory | Decompression reduction from point of first ascent (minute/minute) | Decompression reduction from reaching 6 m (minute/minute) |
|---|---|---|---|
| **Mares Nemo** | RGBM | 0.54 | 0.77 |
| **Suunto Vytec** | RGBM | 0.55 | 0.76 |
| **UWATEC Aladin Pro Ultra** | Bühlmann | 0.67 | 0.89 |

**Figure 6**
**The reaction of three models of dive computer subjected to a nominal compression chamber dive (light grey profile) of 13 minutes at 30m, 28 minutes at 13m and then 23 minutes varying between 9 and 11m to illustrate the decompression variations caused by travelling through threshold values. The y-axis denotes the displayed decompression information of the respective computers; positive values are times remaining before entering decompression; negative values are the decompression times required; values over 99minutes denote that the computer is clear of any decompression obligation**




outdated in quality. The download facility would work only on computers running *Windows 2000*™ or earlier operating systems. Data were recorded only at three-minute intervals; however, it was unclear what the sampling frequency and accuracy actually were. Depth increments were recorded at a resolution of only 0.3 msw; it was unclear how the depth was recorded (e.g., as a threshold or statistically-derived value). These features complicated an accurate recreation of the incident dive.

An initial investigation of the incident dive profile was undertaken using the dive simulation feature of the *Suunto Dive Manager*™ software package. The first run simply employed the dive profile estimated from the download information and failed after 28 minutes because of a staged decompression schedule violation. The second run altered the dive profile to comply with the decompression schedule recommended by the *Suunto* software and stops were 9 minutes at 12 msw, 11 minutes at 6 msw, and 25 minutes at 3 msw. Including travel times between stops, a total of 53 minutes of surfacing time was indicated. This compared with the approximately 23 minutes (at 3 msw) undertaken by the diver in Case 3 (Figure 3).

*Trial 1*

Compression trials were carried out at the Millport Hyperbaric Unit near Glasgow because the facility better delivers rapid compression and decompression rates necessary for dry-diving trials. The *Nexus* computer from the actual diving incident was used in the trial and the incident profile was reproduced using the downloaded depth profile to inform the chamber depth. A *UWATEC Aladin Ultra Pro* computer was subjected to the same profile; both computers were immersed as before and the *Nexus* performance was videoed. The *Nexus* was clear of any decompression obligation as the chamber surfaced from 3 msw. The *Aladin* had gone into "SOS" mode but, when downloaded, the initial decompression stop for the *Aladin* was at 9 msw after 29 minutes of the dive; at the same time, the *Nexus* was indicating a first stop at 3 msw. As this deeper stop was missed, the *Aladin* recomputed the decompression profile. However, at the point at which the decompression stop was missed, a total of 40 minutes of decompression was being indicated as necessary; the *Nexus* showed 20 minutes of decompression. Throughout the profile, the *Nexus* depth display read between 0.7–1.4 msw shallower than the chamber gauge and *Aladin* download respectively.

*Trial 2*

To correct for depth differences, a second trial was carried out a few weeks later. The chamber depth reproduced the depth/time profile to match the observed *Nexus* depth. Depth control was slightly compromised because the *Nexus* displayed at increments of only 0.3 m. The *Aladin* again required an initial decompression stop at 9 msw after 29 minutes of the dive, but a total decompression time of 36 minutes; the *Nexus* indicated an initial stop at 3 msw with a total of 19 minutes of decompression.

It was unclear why the *Nexus* permitted a much reduced decompression obligation compared with the *Aladin* for the identical dive profile. The *Nexus* download provided very few secondary data but it was certain that it was set to air and an altitude range of 0–300 m (A0). A recorded 'violation' only highlighted the fact that the computer had entered decompression mode. There was an indication that the computer was set at a $PPO_2$ limit of 121 kPa (1.2 bar). There was no supporting explanation in the *Nexus* manual as to what this recording referred.[4] Given that the computer could be used both for open-circuit nitrox and constant $PPO_2$ CCR, the recording could relate to the depth-related maximum permissible $PPO_2$ limit for open circuit, or the value chosen for a CCR. Given the large differences in decompressions demonstrated, it is possible that the diver in Case 3 was diving on open-circuit air with a dive computer unknowingly set to rebreather mode. Unfortunately, because of the limitations of the download quality for the *Nexus*, it was not possible to determine the mode to which the computer was set.

**Discussion**

Recreational and scientific diving both depend considerably on dive computers to control decompression. The low published incident rates of DCS[8–13], however, may mask the DCS rates of smaller within-sector groups employing

dive computers for deeper and longer dives (e.g., deep wreck diving), or for multi-day diving programmes.[15] Although deep, multi-day diving groups probably carry a higher risk of DCS for many reasons it cannot be discounted that the efficacy of decompression management by dive computers decreases with added diving complexity.[1] It is not suggested that these cases add to any discussion on complexity-affected efficacy of dive computers. However, their dive profiles, primarily through entering decompression, exceed the limits of the computers that their respective manufacturers recommend.[4]

The potential to misunderstand outputs from some dive computers, matched possibly by peer pressure, may be a contributing factor in some decompression incidents. Case 1 was well aware that her previous episodes of DCS, possibly associated with a PFO, meant that to continue diving she needed to dive more conservatively. This she attempted, but then either ignored or forgot about the changed computer settings; both divers should have been aware of the adjusted levels on the computer and modified their dive practices accordingly. That she then swapped to another unused dive computer with no residual nitrogen loading and possibly no altered conservatism settings could have contributed to her subsequent injury and could easily have been avoided.

The newer generation of dive computers vary markedly in terms of complexity, diversity and size. A lack of knowledge of how the computers worked contributed to all three diving incidents. Case 2 demonstrated that large differences in decompression schedules can be generated by different computer models. However, in Case 2, not only did the divers continue their dive after the computer entered decompression mode, but they also dived deeper. This resulted in a decompression obligation when the dive ascent started. In addition, the decompression requirement did not reduce linearly with ascent time, producing an impending predicament of required decompression exceeding the remaining gas supplies, and resulting in the divers surfacing with missed decompression and developing DCS.

This notwithstanding, the related chamber trials show that whereas single, non-decompression dives produced little difference between the computers trialled, there were important differences between them in how decompression was calculated as the decompression obligation increased, and with multi-day diving. In the multi-dive scenario, all the computers tested reduced decompression times at slower than the estimated rates (Table 1) and the *Suunto* model , which is closest to the DCIEM tables model, generated a much greater decompression requirement. This increases the possibility of divers running out of breathing gas before completing their decompression even though with some computer models the decompression obligations may increase disproportionately to those required as more multi-day diving is performed.

Of concern for divers is the difficulty in understanding the performance of some dive computer models new to the market, e.g., the *Buddy Nexus*. This model is manufactured by *Benemec*™ in Finland; other *Benemec* computers are sold under various brand names including *Orca, Zeagle, Ocean Reef* and *Dacor*. What is surprising about a computer model released within the past seven years is the crude levels of measurement resolution, data storage and download information. No information was available on how the computer was controlled and the only mention of the decompression models employed is of "modified Bühlmann's". With rebranded computers the decompression algorithms employed may come as a "black box" about which the manufacturers themselves have no knowledge.

During diver training, considerable time is taken teaching decompression tables that are rarely used. Since the majority of recreational diving uses decompression computers, computer-awareness training needs enhancing and divers and treating physicians need to be aware of the limitations of computer performance. Knowledge of how a computer should perform would be enhanced by the provision of better technical manuals by manufacturers. Tame reviews in a diving press dependent on advertising revenue do not help and more independent testing of computers should be encouraged. For a recreation/occupation that is heavily into redundant support systems, it would not be unreasonable to suggest diving with two computers, with the diver always defaulting to the more conservative model.

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# Article reprinted from other sources

## Rate of delivery of hyperbaric oxygen treatments does not affect response in soft tissue radionecrosis [Abstract]

Hampson NB, Corman JM

**Background:** Soft tissue radiation necrosis (STRN) is effectively treated with hyperbaric oxygen ($HBO_2$), believed to result from stimulation of angiogenesis in radiation-injured tissue. Thirty to forty $HBO_2$ treatments are usually recommended for STRN. For various reasons, different hyperbaric facilities offer these treatments once or twice daily and from 5–7 days weekly. It is not known whether the clinical response differs as a result of the rate of administration of $HBO_2$ treatments.
**Methods:** Details of hyperbaric treatment courses of patients treated for radiation enteritis/proctitis (n = 65) and cystitis (n = 94) at a single institution were reviewed. Outcomes were compared with the total number of $HBO_2$ treatments administered and also rate of treatment administration.
**Results:** Responses were similar for both forms of STRN whether the patient averaged fewer or greater than 5 treatments per week, or even 3 versus 7 treatments weekly. Outcome did differ, however, dependent on the total number of treatments administered. Response was better in patients receiving 30 or more total treatments, as compared with fewer.
**Conclusions:** Soft tissue radionecrosis of the gastrointestinal tract or bladder is (1) effectively treated with hyperbaric oxygen, (2) has a higher response rate if at least 30 treatments are administered, and (3) is equally responsive to rates of hyperbaric treatment ranging from 3 or fewer to 7 or more treatments per week.

*Center for Hyperbaric Medicine, Section of Pulmonary and Critical Care Medicine and Section of Urology and Renal Transplantation, Virginia Mason Medical Center; Seattle, Washington*

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**Key words**
Reprinted from, hyperbaric oxygen therapy, treatment, soft tissue radionecrosis

# Case report

## Two unusual presentations of probable decompression sickness after deep mixed-gas recreational diving

Andrew Fock and Ian Millar

**Key words**
Case reports, decompression sickness, decompression illness, technical diving, risk factors

**Abstract**

(Fock A, Millar I. Two unusual presentations of probable decompression sickness after deep mixed-gas recreational diving. *Diving and Hyperbaric Medicine*. 2008; 38: 40-2.)
We present two unusual presentations of decompression sickness manifesting at previous injury sites after deep mixed-gas recreational diving. While previous injury is often cited as a predisposing factor in decompression illness, formal case reports of this phenomenon are rare in the diving literature.

### Introduction

Deep mixed-gas technical diving has gained in popularity over the last 20 years. It has allowed divers to access many dive sites previously out of the reach of recreational diving. Expeditions to explore deep wrecks have been popularised over the last few years and often involve multi-day multiple dive profiles to depths greater than 70 metres' sea water (msw) on live-aboard dive vessels in remote locations. These dives are often conducted using closed-circuit rebreather apparatus (CCR) and helium-containing gas mixtures. Not surprisingly, the dives involve substantial decompression stress and are generally viewed as standing outside the established boundaries of the known decompression tables. We present two case reports with unusual manifestations of decompression sickness in otherwise well divers after a series of deep mixed-gas dives.

### Case 1

An otherwise well, 44-year-old male diver with some 20 years' diving experience, and on no medication, had a past history of a fractured right humerus 18 years previously, which had required open reduction and internal fixation after an atrophic non-union. The plate and screws remained *in situ*. The diver had had one previous episode of musculo-skeletal decompression sickness (DCS) involving pain-only symptoms in the other arm some six years previously. This had completely resolved with recompression.

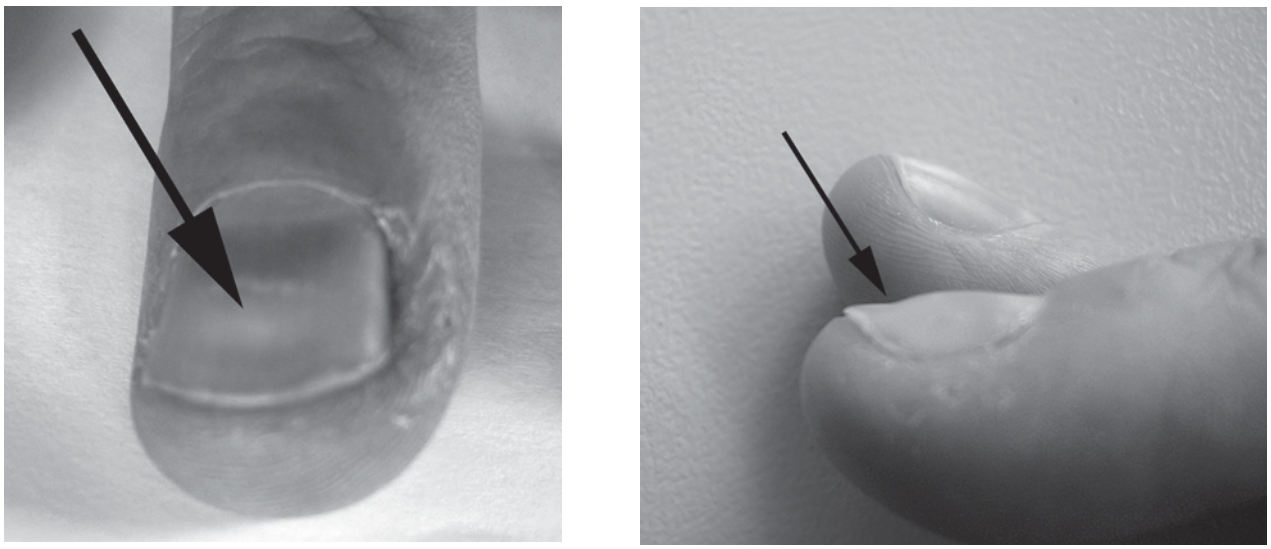
The diver conducted a series of 18 dives over nine days to between 56 msw and 69 msw using a CCR. A trimix diluent consisting of 10% oxygen ($O_2$) and 60% helium (residuum nitrogen) was used for all dives with the onboard computers adjusting the mix by adding $O_2$ to maintain a constant partial pressure of oxygen ($PPO_2$) of 131 kPa. Decompression was controlled by on-board mixed-gas diving computers (VR3™ and VISION™). These utilise modified versions of the Buhlmann ZHL-16 decompression model. Two dives per day were conducted with a surface interval of approximately four hours between dives. Bottom times were generally between 30 and 40 minutes' duration, with total dive times of approximately two hours. The diver increased his $PPO_2$ at the final stop to greater than 152 kPa by using surface-supplied $O_2$ or manually increasing the $PPO_2$ of the CCR unit.

After dive 15 of the series (67 msw for 35 minutes, total dive time (TDT) 1 hour 50 minutes) the diver experienced minor symptoms of DCS (equivocal pain in the right elbow) within a few minutes of surfacing. He immediately returned to 6 msw on the decompression station and remained there on 100% oxygen for 20 minutes. On return to the surface, all symptoms had resolved and did not subsequently return.

After dive 16 of the series (the second dive of the day, 68 msw, 31 minutes, TDT 149 minutes) the diver made an uneventful decompression, but on removing his wetsuit noticed palpable crepitus in the scar and over the plate in his right arm. This was confirmed by one of the authors (AF). The crepitus lasted for approximately 15 minutes before resolving spontaneously. The diver did not develop any further signs or symptoms of DCS; notably there was no rash associated with the area of crepitus or at any other site. Crepitus did not develop after dive 17 the following morning, but returned after dive 18 (68 msw, 31 minutes, TDT 2 hours 7 minutes) later that afternoon. Again, the crepitus resolved spontaneously over about 15 minutes. The diver did not develop any other symptoms of DCS and flew home 36 hours later without incident.

The diver was reviewed nine weeks later. In that time, the diver had developed transverse sulci consistent with Beau's lines across the thumb, index and ring fingernails of his right hand (Figure 1a and b). Close examination also revealed a slight sulcus across the nail of the left thumb, but no abnormalities of the nails of the other fingers or toes. The sulci on the affected fingers were located approximately 10 mm from the eponychium on each finger. The diver had been

**Figure 1**
**Horizontal indentations (Beau's lines) in the nails of the fingers of the right hand of Case 1**



well in the intervening period and at no time had developed any symptoms consistent with decompression sickness.

**Case 2**

The diver was an otherwise well, 56-year-old male with more than 30 years' diving experience. He had an injury to his right eye as a child, complicated by acute glaucoma some years later and resulting in total blindness in that eye. The diver was using a CCR on the same expedition as Case one. However, due to marked hyperoxic-induced myopia, he generally limited himself to one long dive per day. $PPO_2$ was maintained at 141 kPa, and bottom times were usually of 40 to 60 minutes' duration, for total dive times of approximately three hours.

After dive 7 of the series, the diver returned to the surface after an uneventful decompression. Over the next 10 minutes or so he developed scintillating visual symptoms in the blind eye. He placed himself on 100% oxygen via his rebreather unit. After approximately 10 minutes the symptoms had resolved. No other neurological or systemic symptoms were noted. On close questioning, the diver revealed he had had similar episodes, usually after deep, long dives, which had also responded to oxygen. The diver continued to dive for the next two days without the reappearance of symptoms. He then had a two-day break before continuing on another similar diving expedition. Symptoms did not recur.

**Discussion**

Repetitive, deep, mixed-gas diving would be expected to produce considerable decompression stress. However, a previous study on a similar group of technical divers failed to show a significant increase in diver health status score.[1] During this most recent expedition, seven out of the eight divers developed marginal symptoms of DCS at one time or another during the expedition.[1] All cases responded rapidly to either surface oxygen or immediate in-water recompression on 100% oxygen and no overt cases of DCS required formal recompression treatment.

Previous injury is often quoted as a predisposing factor for the development of DCS.[2] However, case reports are rare. Several skin manifestations are also described:[3]

- Pruritus with no rash
- Scarlatiniform rash with pruritus
- Erysipeloid rash
- Cutis marmorata
- Emphysema
- Lymphatic obstruction

Emphysema may be further divided into

- intracutaneous (minute blebs associated with mild pruritus reported in aviators only)
- subcutaneous emphysema associated with gas embolism.

In the first case, the subcutaneous emphysema observed did not conform to either of the above patterns. It was not associated with pruritus and was deeper than intracutaneous blebs. There was no obvious gas embolism. The location of the palpable gas over a potential nidus for bubble formation, in this case the stainless steel plate screwed to the humerus, or in the scar tissue from the previous surgery, suggests that the bubble formation may be a result of the reduced off-gassing due to alterations in local blood flow. An ultrasound to accurately locate the exact site of the bubbles would have been of great interest. It is conceivable that a build-up of tissue inert gas occurred in what is probably poorly perfused, damaged tissue over the course of the multiple daily exposures, with inadequate off-gassing occurring between dives despite no other overt symptoms developing in normally perfused tissues.

Beau's lines (transverse sulci of the nails) have been associated with a number of conditions (Table 1). They are

**Table 1**
**Some conditions associated with Beau's lines [4]**

| |
|---|
| Typhus |
| Acute rheumatic fever |
| Diphtheria |
| Syphilis |
| Malaria |
| Vitamin deficiencies |
| Myocardial infections |
| Subacute and chronic pancreatitis with malabsorption |
| Chemotherapy |
| Acute gastrointestinal bleeding |
| Other severe metabolic stresses |

thought to represent a temporary arrest of the nail growth. They manifest as furrows in the nails that begin at the lunula and gradually move distally with growth. Fingernails grow at a rate of between 0.5 and 1.2 mm per week in a normal adult. The appearance of the Beau's lines in our case report, some 10 mm from the eponychium, would be consistent with the causative event occurring during the expedition.

Nail changes have been previously recorded in the diving literature. Schwartz reported changes similar to those that we observed in the nails of six divers during a helium–oxygen saturation dive to 1,100 feet of sea water (fsw) in 1986.[4] A similar dive to 1,000 fsw in 1988 also produced Beau's lines in a further two divers. Similar changes were noted by Hutchinson in a Mount Everest climber who spent six weeks at an altitude of above 5,500 m .[5] In all these cases, the lines were observed in the nails of all of the fingers. This is in contrast to our case, where the changes were restricted to three fingers of the right hand. It is interesting to speculate that the development of the Beau's lines may have been associated with the subcutaneous emphysema, which occurred some weeks earlier in the same arm during the diving expedition, and may represent more generalised bubble formation in peripheral sites. To the authors' knowledge, this is the first report of their occurrence after non-saturation decompression diving.

The second case presents more of a diagnostic dilemma. While the onset of the scintillating photopsia in his blind eye was temporally associated with his diving, such phenomena have also been associated with visual migraine. A recent Cochrane review has indicated that migraine is responsive to both hyperbaric and normobaric oxygen.[6] As this diver had had a prolonged exposure to 141 kPa of oxygen prior to the onset of symptoms, the diagnosis of migraine would be less likely. The diver does not normally suffer from migraine or photopsia, tending to implicate DCS as a more likely cause. The rapid response to surface oxygen might just as plausibly be expected from either migraine or DCS.

As with the first case, this diver continued to dive after symptoms were noted, without developing other symptoms of DCS (other than subjective worsening of his hyperoxic-induced myopia). This would seem at odds with the commonly held belief that recent mild DCS predisposes to severe DCS if diving continues. In both cases, the subsequent dives were also in the 'extreme exposure' category according to dive tables. The lack of subsequent symptoms may be due in part to the high helium content of the diluent, allowing for continued off-gassing during the surface interval and the use of 100% or near 100% oxygen during the final phases of decompression. Whilst the authors would not encourage the practice of self treatment, continuation of diving after symptoms of DCS would seem to be common amongst technical divers. Anecdotally, this appears thus far to have produced few complications of a serious nature, at least in the divers with whom we have had contact.

## Conclusions

We present two unusual cases of probable DCS associated with previous injury, either resolving spontaneously or responding to normobaric $O_2$. Neither case developed more classical symptoms of DCS despite ongoing diving to substantial depth using mixed gases.

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# The diving doctor's diary

## Two case reports of epileptic seizures related to probable cerebral arterial gas embolism

Colin M Wilson and Martin DJ Sayer

**Key words**
Case reports, cerbral arterial gas embolism (CAGE), decompression illness, recreational diving

**Abstract**

(Wilson CM, Sayer MDJ. Two case reports of epileptic seizures related to probable cerebral arterial gas embolism. *Diving and Hyperbaric Medicine*. 2008; 38: 43-6.)
Two cases are presented of divers suffering epileptic seizure and loss of consciousness as a result of probable cerebral arterial gas embolism (CAGE). Both cases had apparently problem-free dives with no obvious provocation for CAGE, though one case may have been having repeated embolisms for some time in their diving career. Demonstrated also is the Type III form of decompression sickness, where spinal cord disease follows CAGE in a biphasic manner.

### Introduction

Cerebral arterial gas embolism (CAGE) is a serious, potentially fatal complication of scuba diving. It is associated with rapid or poorly controlled ascents or breath-holding on ascent, but may occur with apparently normal ascent profiles even in individuals without pre-existing pulmonary pathology. It may also occur in decompression sickness (DCS) where a clinically significant right-to-left heart communication, such as a large patent foramen ovale (PFO), is present. We report here two cases of divers with probable CAGE complicated by tonic-clonic seizures following open-circuit scuba dives.

### Case 1

A 41-year-old male, with three-and-a-half years of diving experience, was making his first dive of the weekend on a shallow wreck a short distance from Oban on the west coast of Scotland. He entered the water at 1030 hr, diving to a maximum depth of 16 metres' sea water (msw), using a dive computer for decompression management. He surfaced after an uneventful dive of a total duration of 43 minutes having stopped for a precautionary one minute at 6 msw. He got back aboard the rigid-hulled inflatable boat (RIB) quickly, but then developed a visual disturbance described as "all going dark". This was followed by severe spasms in his arms and legs that progressed to a tonic-clonic seizure, all within 10 minutes of surfacing.

The dive buddies in the RIB understandably panicked and made their way to shore while radioing the coastguard; as a result of their panic their message was incoherent, incomplete and confusing. However, an ambulance was quickly on the scene, oxygen was commenced, and the diver was transferred to the Dunstaffnage Hyperbaric Unit (DHU) a short distance away. The diver arrived at 1145 hr (32 minutes after surfacing) and was found to be confused, agitated and disorientated. Cooperation with examination was limited. He was lying with his hips flexed and his arms, hands and legs were rigid with muscular spasm. His hands were white and cold with poor capillary refill. There were no respiratory symptoms or abnormal physical findings in the chest. Full neurological examination was difficult at this time; he had been incontinent of urine. He had improved since the ambulance paramedics had attended him, probably because of oxygen administration, though the passage of time post-seizure might also have been a factor. Radiological facilities are not available at the DHU, but a chest X-ray at Oban Hospital after recompression was normal.

He was recompressed to 282 kPa (18 msw) at 1200 hr, less than an hour after surfacing, using the Royal Navy Treatment Table 62 (RN 62). Within ten minutes of being on 100% oxygen he claimed to be symptom free. The treating doctor (CMW) locked into the chamber to re-examine him at the first air break. Peripherally, he was now warm and pink with normal capillary refill. His vision was normal and he had normal muscular power that enabled him to walk normally. A Romberg test was negative and he was fully orientated in time and space and capable of delivering normal 'serial sevens'. However, it was noted that he had brisk reflexes in his arms and legs and had disdiadochokinesis of his right arm. He had no recollection of events from just after surfacing until going on oxygen under pressure. Having had a rapid and complete resolution of his signs and symptoms following the first cycle of 20 minutes of oxygen, he continued on an RN 62 treatment without modification.

Following treatment, the patient reported some discomfort in his calves and slight soreness in his right upper arm; both symptoms had not been mentioned previously. He felt considerably better but remarked on feeling tired. It was noted that he had slight impairment in heel-toe walking and

some difficulty in writing his address and signing his name. He was admitted to Oban Hospital for monitoring.

The following morning, there was a reduction in the discomfort in his calves and arms and his heel-toe walking was improved. A vast improvement in his writing was noted when compared with the efforts made the previous evening immediately post-treatment. He was returned to the DHU and was treated later that morning with a Comex 12 (222 kPa) hyperbaric oxygen table. All his residual symptoms and signs had settled following this treatment.

He admitted to some past diving-related medical history. Three years before he had had a similar seizure-like event while diving. After some delays he had been flown to a recompression facility. On arrival he was asymptomatic with a normal examination; he was given a precautionary treatment of an unmodified RN 62. Following that incident he had been assessed for a PFO and found to be negative, though he had no information on the examination method used. He continued to dive and, over the subsequent years, he had four further episodes following diving. These occurred typically within five minutes of surfacing and followed a pattern of visual upset, coordination problems, blue lips and some variable degree of loss of consciousness. He tended to be "sat in the corner" by his buddies as his symptoms resolved over the next hour. The 'diving doctor' associated with this dive club had put these episodes down to "carotid stimulation from a tight neck seal".

Following his treatment at DHU, he was diagnosed as having had a CAGE. It was thought probable that he had had repeated embolisms during his diving career. He was advised not to dive again and discharged home the day after his second treatment.

**Case 2**

A 22-year-old male scaffolder was learning to dive with shellfish divers on the west coast of Scotland. He had not undergone any dive training course or medical assessment and, in fact, had a history of using inhaled salbutamol when he had respiratory tract infections. The incident dive was only his sixth dive in total. Initially, there was very little information relating to the incident and even on arrival at the DHU he was unable to provide a full story of what had happened. During his recompression treatment, numerous telephone calls eventually collected the required information to piece together the chain of events.

He had entered the water with a seasoned shellfish diver as his buddy to what was initially reported to the DHU as a depth of 50 msw. In the fullness of time, this was corrected to 50 feet (16 msw) for 18 minutes. On the surface, at about 1230 hr, he had difficulty in swimming back to the boat and had a marked weakness of his left arm. He was unable to climb the ladder back on board and required help from the other divers. On the boat he suffered a tonic-clonic seizure, becoming rigid for 30 seconds with mouth clenching followed by rhythmic contractions of all his limbs. He remained deeply unconscious for 5–10 minutes, and thereafter his conscious level improved slowly. An ambulance was alerted as they returned the short distance to the harbour. During this time, the diver reported visual loss, being unable to see his hand in front of his face. He also reported a right temporal headache, paraesthesia and weakness in his left arm and leg. On meeting the vessel on the quayside, the ambulance paramedics commenced him on high-flow oxygen and found it necessary to remove him from the boat by stretcher.

In Scotland, there is a national emergency telephone support service run from Aberdeen Royal Infirmary, where the National Health Service has the only funded registration service in the UK. The Aberdeen consultant on call advised that the diver should be transferred to a recompression facility for urgent assessment and treatment. Air transportation was not available at that time but, as the diver's condition was improving, the ambulance continued toward the DHU at Oban. Transfer took approximately two hours and during this time his conscious level continued to improve. On arrival at DHU at 1500 hr, examination demonstrated a fit, muscular individual with no abnormal physical signs and he passed urine without difficulty. With a diagnosis of a probable CAGE, intravenous fluids were commenced and he was recompressed on an RN 62 at 1515 hr.

During the second oxygen cycle at 282 kPa (18 msw) he reported paraesthesia with altered sensation in the anterior aspects of both his thighs running to his feet and also paraesthesia and numbness in his left wrist. The treatment table was extended at 282 kPa, with the symptoms in his left hand completely settling, before decompressing to 191 kPa, though his legs remained little changed. On surfacing, he had persisting altered sensation in L3 to L5 dermatome distribution in both his legs, with intermittent paraesthesia in both his feet. He was transferred to Oban Hospital for monitoring, where a chest X-ray was normal.

The following morning, his muscle power remained normal but he was found to have altered sensation from dermatome T9 on the right and T10 on the left to L5. He had noticed difficulty in micturition both in initiating and maintaining his flow. He was diagnosed as having had a relapse and was transferred back to the DHU for further recompression therapy on an RN 62 table.

During the second treatment he had some improvement with the level of altered sensation slowly moving distally; he was able to pass urine more easily. The treatment table was extended by two additional oxygen:air cycles at 282 kPa and one at 191 kPa. On surfacing, the altered sensation showed little improvement, with a level of T10 on the right and T12 on the left. He was returned to the hospital for further post-treatment monitoring.

The diver's girlfriend had driven the 200 miles to be with him the next morning. His behaviour was reported by the nursing staff as being "odd" in her presence; in fact, she accompanied him to the shower. His altered sensory level remained unchanged and he reported his micturition as being normal. However, his girlfriend confided in nursing staff that he still had some problems. He was reluctant to undergo further recompression, claiming claustrophobia, but eventually agreed to further treatment. He was treated with a Comex 12 (222 kPa) hyperbaric oxygen table on the morning of the third day of treatments and returned to hospital afterwards with no apparent benefit.

With the imminent departure of his girlfriend for home two hours after surfacing from the third treatment, he decided he was going to accompany her and was not persuaded to stay, discharging himself against medical advice. However, he said he had no intention of continuing to dive; there has been no further contact with this man.

**Discussion**

The onset of symptoms resulting from CAGE will occur within 10 minutes, as happened in both cases reported here. Neither of them had a deep dive or any provocative ascent problems that could result in pulmonary barotrauma (PBT), though this cannot be completely excluded. There is also the possibility of a right-to-left shunt as a contributing factor. However, we know in Case 1 that investigations had excluded a PFO before his treatment at the DHU, although he gives a history of probable repeated, though possibly less severe, gas embolisms over his diving career. Both these men suffered a tonic-clonic seizure soon after surfacing.

Seizure associated with DCS is uncommon, but in association with CAGE is more common. The involvement of epilepsy in the two cases of seizure reported here is not discounted although neither diver had any past medical history suggestive of epilepsy. There may be a group of people for whom there is a lower threshold for seizure activity and a gas embolism could be a precipitating event. With the push to consider individuals with epilepsy as fit to dive, we may see more patients having seizures with less provocation, and in a hostile environment they may enter the fatality statistics.[1,2]

The most common reason for divers to succumb to an arterial gas embolism (AGE) is gas entering the circulation from PBT. This is the result of damage to the lungs from the over-expansion of pulmonary gas during ascent. The common symptoms of PBT of cough, haemoptysis, retrosternal chest pain and dyspnoea were all absent in the cases reported above. In the majority of divers with AGE, PBT often cannot be diagnosed radiographically.[3] These cases, of course, may have had asymptomatic PBT. It is also possible that venous gas bubbles could have been transferred to the arterial tree by right-to-left shunting, as a result of cardiac or respiratory anomalies.

Case 2 demonstrates the Type III form of DCS with a biphasic pattern where patients, initially with symptoms of AGE, then exhibit signs of spinal DCS.[4–6] Individuals who suffer CAGE when associated with a gas load, even if this load is thought to be trivial when applying standard decompression schedules, may suffer a severe form of spinal DCS that is often resistant to therapy.[3] It is postulated that the second phase of this DCS is caused by the presence of bubbles from an embolism precipitating further bubble formation in saturated spinal tissues. Greer reported a case where a scientific diver had a gas embolism with epileptic seizure and then developed severe spinal involvement although, in that case, there was a delay of over nine hours before recompression.[4] It is also observed that some divers suffering CAGE have a relapsing disease and, despite additional recompression, little benefit is achieved.[3]

A number of studies on CAGE have compared the results of submarine escape tower (SET) training with sport diving.[3,7] These noted differences in the respective presentations and results of treatment. These differences were not related solely to the speed of treatment but were also considered to be caused by the differences in inert gas loading, which was assumed to be virtually absent in the SET cases.

In a review of 300 consecutive cases of decompression illness (DCI) (84% of whom were sport divers) treated at DHU, 10.3% were diagnosed as having suffered CAGE, and of those 10% presented with a history of having had a seizure (i.e., 1% of 300, or three cases; Wilson CM, Sayer MDJ, unpublished observations). All of those 300 cases would be assumed to have had gas loadings greater than SET trainees. The incidence of CAGE in the sport-diver population has been falling over recent years. Reports of DCI collected by the Divers Alert Network (DAN) indicate that those cases attributed to CAGE have fallen from 18% of the total in the 1980s to less than 7% in 2001.[8] DAN have speculated that this decrease may be attributable to the use of modern dive computers indicating ascent rates that, in turn, may contribute to divers better controlling their ascents.

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# Cerebral arterial gas embolism in a diver using closed-circuit rebreather diving apparatus

Si Jack Chong and Teck Wei Tan

**Key words**
Cerebral arterial gas embolism (CAGE), closed-circuit underwater breathing system, recompression therapy

**Abstract**

(Chong SJ, Tan TW. Cerebral arterial gas embolism in a diver using closed-circuit rebreather diving apparatus. *Diving and Hyperbaric Medicine*. 2008; 38: 46-7.)
A Singaporean naval diver suffered symptoms and signs of cerebral arterial gas embolism (CAGE) following a panicked ascent from a depth of 5–6 metres' sea water while using a Dräger LAR V closed-circuit oxygen rebreather system. He presented with altered mental status and paresis. CAGE due to gas mixtures with high oxygen content has seldom been reported. The diver had no sequelae following prompt recompression therapy. This positive clinical outcome may be attributable to the high oxygen content in the diver's inspired gas and/or the promptness of recompression.

## Introduction

Pulmonary barotrauma following ascent from depth whilst diving is one of the most serious forms of all barotraumas. Gas in the lungs expands during ascent and may rupture into adjacent lung tissues, resulting in mediastinal emphysema, pneumothorax or arterial gas embolism.[1] Cerebral arterial gas embolism (CAGE) is of particular concern as the vascular damage, hypoxia and the triggering of the inflammatory cascade in cerebral vessels may result in high rates of relapse, neurological deficits and even death.[2] CAGE has been widely reported with the use of open-circuit compressed gases, but there have been few reports of pulmonary barotrauma associated with closed-circuit rebreather (CCR) diving systems.[3,4] This may be due to several factors, including the experience of the divers using the CCRs and the high oxygen ($O_2$) content in the gases used in these diving systems.

We present the case of a young, healthy Royal Singapore Navy (RSN) diver who presented with symptoms and signs of CAGE after an uncontrolled ascent while using a CCR system. He responded clinically to recompression therapy and did not demonstrate any sequelae at follow up.

## Case report

A 21-year-old RSN diver with nearly two years' diving experience was performing a routine compass dive for 60 minutes just outside the naval camp, with a maximum depth of 5–6 metres' sea water (msw) and using the Dräger LAR V oxygen CCR system. The $O_2$ concentration of the breathing gas in the LAR V CCR has been shown to be up to 74%.[5] The diver had completed about 35 minutes of his training dive when he developed difficulties with his mouthpiece and subsequently panicked and ascended uncontrollably to the surface as he seemed to be "choking".

At the surface, the dive supervisor noted that the diver had altered sensorium as he could not recognise his supervisor. He was thrashing about wildly but was relatively weak. The dive supervisor rapidly rescued him and administered 100% oxygen while evacuating him to the recompression facility within the nearby naval camp.

At the recompression facility, intravenous hydration was commenced. Blood pressure and heart rate were normal but there was tachypnoea of 22 breaths/min and pulse oximetry showed a decrease in oxygen saturation (92%) on 40% oxygen. There was altered mental status with a GCS score of 11 (E3 M4 V4) and the patient was not orientated to time, place or person. Neurological examination revealed generalised weakness with Medical Research Council grade of 4/5. An urgent chest X-ray did not demonstrate emphysema or pneumothorax.

A diagnosis of CAGE was made and recompression therapy was initiated within 35 minutes of the accident using the USN Treatment Table 6 (USN T6). The patient's symptoms began to improve at depth and achieved complete resolution by the first air break. A neurological examination done at depth demonstrated complete mental acuity and full power. The entire treatment table was completed and the patient monitored overnight with no relapse or sequelae. The patient underwent a repeat USN T6 the following day.

The blood investigations done showed a normal full blood count and urea, glucose and electrolyte levels within normal limits. A computed tomography (CT) scan of the patient's chest and a spirometry performed three months later did not reveal any abnormality. He returned to active diving six months after the incident.

**Discussion**

The expert opinion is that serious symptoms that develop immediately after ascent must be regarded as AGE and treated accordingly until a definitive diagnosis can be made.[1] Experimentally, cadaveric lungs have been shown to burst at pressures as low as 70 mmHg. In addition, there have been reports of intensive-care patients with lung ruptures following positive pressure of more than 70 mmHg.[1] This is approximately equivalent to an ascent of one metre in water. There have also been reports of CAGE occurring following ascents of one metre depth of water.[6]

The diver in this case suffered a CAGE following a rapid uncontrolled ascent from a depth of about 6 msw while using the LAR V CCR system. The rapid manifestation of his clinical symptoms on surfacing and signs of altered mental status with paresis were classical for CAGE. However, early recompression following CAGE carries a good prognosis, as seen in our patient.

The interesting aspect of this case was that the diver suffered a CAGE while using a Dräger LAR V oxygen CCR system. Using the single fill/empty cycle purge procedure developed by Thalmann and Butler, a diver can effectively increase the oxygen concentration in his breathing gas to 74%, in contrast to the 20.9% found in normal air.[5] This was the most likely oxygen concentration in the breathing gas of our patient at the time of the incident. With this oxygen-rich gas mixture, the gas bubbles responsible for the pathogenesis of this case of CAGE should theoretically be more easily metabolised by the surrounding tissues, reducing in size rapidly, and the oxygen content should mitigate the hypoxic effects of air embolism to tissues. The rapid and complete resolution of the symptoms may thus be contributed to by the high oxygen content. In a similar report by Carstairs the patient also achieved complete resolution.[4] The rapid initiation of treatment (within 35 minutes of the incident), thanks to the proximity of the recompression facility to the dive site, also would have contributed to the positive outcome.

Mediastinal emphysema, pneumothorax and local pulmonary damage may be associated with CAGE. However the subsequent CT scan of this patient's thorax did not reveal any pulmonary changes or pathologies. There were also no changes in the spirometry results of this patient. These follow-up medical investigations help to ascertain the future risk factors for diving, as well as shed light on the origins of pulmonary barotrauma. Even if there has been a history of rapid ascent, the presence of pulmonary bullae or other abnormalities must be sought.

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**Editor's comment:**
Overdistension and overpressurisation of the lung are thought to be the underlying causes of pulmonary barotrauma and arterial gas embolism (AGE), but the exact mechanisms of injury remain unclear. Much of the clinical work on AGE relates to the submarine escape training environment, which may differ in several important ways from scuba diving injuries. Most importantly the diver has a greater degree of gas loading and recompression is often delayed, sometimes for many hours, resulting in much poorer outcomes than in the immediate recompression of escape trainees[1] or of the military diver described by Chong and Tan. A subgroup of AGE victims demonstrates a relapsing or secondary deterioration course, either as a result of the neutrophil-mediated secondary injury that occurs or from the onset of decompression sickness, the so-called Type III decompression sickness, as may possibly have been the situation in the second of Wilson and Sayer's cases.

Symptoms and signs typically develop within five minutes of the incident – frequently an uncontrolled or rapid ascent due to running out of air or buoyancy problems. There is an apparent inverse relationship between severity and the time to onset. Neuman reports an 18% incidence of convulsions in 74 diving accidents with cerebral arterial gas embolism (CAGE).[2] However, this rate appears much higher than that in the Scottish series, and in our own experience in South Island, New Zealand (Davis FM, unpublished observations). In 83 decompression illness cases treated in the Christchurch unit over eight years, 17 (1 in 5) divers had a definitive diagnosis of CAGE and four others had possibly suffered a CAGE. Of these, only one had a documented seizure either at the site of the accident or subsequently. Seizure associated with CAGE tends to carry a very poor prognosis according to anecdotal reports from other hyperbaric units and may commonly be associated with cardio-respiratory arrest and death.

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# Position available

## Hyperbaric Medicine + Anaesthesia or Emergency Medicine Specialist

**Vacancies #:** 6308 + 6946 (Anaesthesia; no # for EM)
**Closing date:** 09 May 2008 (Late applications will be accepted)

The Hyperbaric Medicine Unit (HMU), Christchurch Hospital has a 0.4 FTE position to replace a retiring member of the senior staff. This vacancy will be combined with either Anaesthesia or Emergency Medicine sessions to create a full-time specialist position.

CDHB is a busy tertiary referral centre in South Island of New Zealand, with close links to the University of Otago Christchurch School of Medicine and Health Sciences situated on the Christchurch Hospital site. Surgical services include all the main surgical specialties, whilst the Department of Medicine covers all major sub-specialties. There is a regional Spinal Injuries Unit and a Bone Marrow Transplant Unit, and the Emergency Department is the busiest in New Zealand.

HMU is administered within the Anaesthetic Department and is currently being assessed by the ANZ College of Anaesthetists for approval for resident training in hyperbaric medicine. The HMU provides a full range of hyperbaric services for the Canterbury District Health Board (CDHB), and is a referral centre for other DHBs in South Island and some parts of North Island. Clinical practice is based on the UHMS and ANZHMG guidelines. We operate a 4 ATA-capable rectangular multiplace chamber providing around 1,300 treatments per year. Our patients range from ambulatory through critically ill, and we provide a 24/7 emergency service [visit <www.cdhb.govt.nz/hbu/>].

Applicants should have appropriate training and experience in hyperbaric medicine, and an Emergency Medicine or Anaesthesia post-graduate qualification recognised for vocational registration by the Medical Council of New Zealand. Foreign graduates will need to meet all immigration and medical council requirements. A successful appointee in Anaesthesia would preferably have generalist skills with a focus on day surgery, acute theatre work, preassessment, and elective orthopaedics. Flexibility in covering other lists is also required and the ability to work in obstetrics would be an advantage.

Christchurch ("The Garden City") is a university city (Canterbury, Lincoln and Otago Universities) of about 365,000 people. It is situated on the East Coast of South Island close to the Banks Peninsula and the commercial harbour town of Lyttelton, and is an hour's drive from the Southern Alps. A wide range of outdoor recreational activities are readily accessible locally and throughout South Island [visit <www.ccc.govt.nz>].

**For the position description contact:**
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# 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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## SPUMS website news

Hello to all from your friendly SPUMS Webmaster. There have been a couple of things happening with the website over recent times. Firstly (and most importantly) we have started uploading full copies of the SPUMS Journal to the Rubicon Foundation Website. These will be updated to all the journals except for the last three years, which will eventually become a members only access portal on our website. This is a significant advance for SPUMS as it raises our profile in the hyperbaric world and I feel a few of our northern hemisphere colleagues will be more than a little surprised at the quality and breadth of research from the southern half of the planet. Currently we have Volumes 1–30 being uploaded at the Rubicon Foundation, which can be accessed by going to the Links page on the SPUMS website. Rubicon has also indexed the journal to make it searchable online. Thanks to Gene Hobbs from The Rubicon Foundation for doing this and also to all the members who have supported this initiative.

I have added another link area to our site called "Other notices", which currently has one section of SPUMS-approved notices namely job opportunities in hyperbaric medicine. SPUMS has had a general policy of minimal advertising in its journal but it was thought that advertising job opportunities could help members increase their participation in the organisation so this area is more of a

community notice area. Anyone who wants to utilise this facility should submit a request to the SPUMS Committee via the Secretary and we will deal with it on a case-by-case basis.

Finally, I have added a link to the Historical Diving Society–Asia Pacific as there has been a warm relationship between SPUMS and HDS-AP involving complimentary journal swapping and genuine cross-topic interest. If you have any interest in diving history, I recommend visiting the HDS–AP site (see link on <www.spums.org.au>) as they have some great stories that would make most hyperbaric physicians have nightmares!

Regards to all,

*Glen Hawkins, SPUMS Webmaster*

## SPUMS Annual General Meeting 2008

The AGM for SPUMS 2008 is to be held at Liamo Resort, Kimbe WNB, PNG, at 1000 hr, Wednesday 28 May 2008.

**Agenda**

**Apologies:**

**Minutes of the previous meeting:**

Minutes of the previous meeting will be posted on the notice board at Liamo Resort and were published in *Diving and Hyperbaric Medicine.* 2007; 37(2): 101-4.

**Matters arising from the minutes:**

**Annual reports:**

President's report
Secretary's report
Education Officer's report
Annual financial statement and Treasurer's report

**Subscription fees for 2009:**

Proposed by the Treasurer, seconded by the Secretary:
Full members AUD$150.00 (internet transaction); AUD$170 (manual/paper-based transaction)
Associate /other members AUD$80 (internet transaction); AUD$100 (manual/paper-based transaction)

**Election of office bearers:**

President
Secretary
Education Officer
Committee Members (2)

**Appointment of the Auditor 2008:**

Proposed by the Treasurer, seconded by the Secretary:
Barrett, Baxter and Bye, 60 Albert Road, South Melbourne 3205

**Business of which notice has been given:**

Motion to elect Neal Pollock, PhD, to full Membership of the Society.
Proposer: Mike Davis; seconder: Simon Mitchell

## ANZCA Certificate in Diving and Hyperbaric Medicine

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

**Eligibility criteria are:**

1 Fellowship of a Specialist College in Australia or New Zealand. This includes all specialties, and the Royal Australian College of General Practitioners.
2 Completion of training courses in Diving Medicine and in Hyperbaric Medicine of at least 4 weeks' total duration. For example, one of:
   a ANZHMG course at Prince of Wales Hospital Sydney, **and** Royal Adelaide Hospital or HMAS Penguin diving medical officers course **OR**
   b Auckland University Diploma in Diving and Hyperbaric Medicine.
3 **EITHER:**
   a Completion of the Diploma of the South Pacific Underwater Medicine Society, including 6 months' full-time equivalent experience in a hyperbaric unit and successful completion of a thesis or research project approved by the Assessor, SPUMS.
   b **and** Completion of a further 12 months' full-time equivalent clinical experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA.
   **OR:**
   c Completion of 18 months' full-time equivalent experience in a hospital-based hyperbaric unit which is approved for training in Diving and Hyperbaric Medicine by the ANZCA
   d **and** Completion of a formal project in accordance with ANZCA Professional Document TE11 "Formal Project Guidelines". The formal project must be constructed around a topic which is relevant to the practice of Diving and Hyperbaric Medicine, and must be approved by the ANZCA Assessor prior to commencement.
4 Completion of a workbook documenting the details of clinical exposure attained during the training period.
5 Candidates who do not hold an Australian or New Zealand specialist qualification in Anaesthesia, Intensive Care or Emergency Medicine are required to demonstrate airway skills competency as specified by ANZCA in the document "Airway skills requirement for training in Diving and Hyperbaric Medicine".

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

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

# 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

## Themes:

The Treatment Tables
Tropical/Envenomation Medicine Update
Resuscitation Update

Alf 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*. Richard Moon is Associate Professor of Anesthesiology at Duke University Medical Center, USA, and 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.

For registration go to the SPUMS website: <www.spums.org.au>
click on 'Conference Registration'. Early registration/booking is recommended.

Abstracts for presentations are very welcome and should be submitted to the Convenor before 30 April 2008 as a Word file of up to 250 words (excluding references – 4 only) and with only one figure.

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

Convenor: Dr Chris Acott
E-mail: <cacott@optusnet.com.au>
Telephone: +61-(0)8-8431-2295
Facsimile: +61-(0)8-8431-8219
Mobile: +61-(0)412-618417

# 34th Scientific Meeting of the European Underwater & Baromedical Society

## Graz, Austria, 3 to 6 September 2008

### Welcome to Graz!

We are proud and happy to organize this year's EUBS conference for you. Come and join us in Graz, elected European cultural capital in 2003, capital of Styria, the "green heart of Austria"!

Univ. Prof. Dr Freyja Maria Smolle-Jüttner Dr Beatrice Ratzenhofer-Komenda
Dr Winfried Beuster, Presidium

### Main topics

This conference will be devoted to interdisciplinary HBO therapy as well as to basic science and to aspects of nursing in hyperbaric medicine.

**Hyperbaric medicine topics:**

- Application of HBOT in burn injury, critical care, neurological disorders,
- oncology, paediatrics, radiotherapy and traumatology
- Critical incidence reporting – discussion of cases
- Basic research
- Nursing the HBO therapy patient – a challenge?

**Diving medicine topics:**

- Advances in diving research
- Validation of decompression profiles
- Restart of diving after neurological DCI
- Special environmental conditions: high altitudes, cold water
- Diving with the handicapped

### Call for abstracts and deadlines:

Submission of abstracts: 4 May 2008
Early registration: 1 June 2008
Submission of full papers: 22 June 2008

Detailed instructions for abstract submission (electronic only) and publication may be found on the conference website: <http://www.eubs2008.org>. Peer-reviewed abstracts will be published as a supplement to the journal *Diving and Hyperbaric Medicine* in December. Participants are also invited to submit their full manuscript for consideration of publication in *Diving and Hyperbaric Medicine*. Student travel grants are available and participation in the Zetterstrom Poster Award is encouraged. Further information is available on the website.

Satellite meetings, social events, and prices are shown on the conference website.

**Venue:** Main Auditorium at Graz University Hospital, Auenbrugger Platz 36
**Hotel Reservation:** via Graz Tourism Agency, available via the conference website <http://www.graztourismus.at>
**E-mail:** <info@graztourismus.at>
**Phone:** +43-(0)316-807547
Refer to EUBS 2008 when booking, please. Please do not hesitate to contact us if you need further assistance:

**Conference office:** Martina Neuhold, Auenbrugger Platz 29, Graz
**Office phone:** +43-(0)316-385-81923 or +43-(0)664-859-6146

## Minutes of the EUBS General Assembly held on 14 September 2007, Sharm el Sheikh, Egypt

The President, Alf Brubakk, welcomes the members. The agenda and the minutes from the General Assembly 2006 are accepted, followed by the report of the General Secretary of the 33rd Annual Conference, Dr Adel Taher. There were 230 paying members, 22 guests and 320 participants to the gala dinner all in all, from 31 different countries. The meeting is considered a success.

In his presidential report, Alf Brubakk states that there has been a 10% increase in membership up to an actual total of 350 members. The members should, however, increase their participation in the Society's affairs, for instance only 20% have taken part in the elections of the new Vice President and new Member at Large. Maybe this is due to the fact that the Society should do more for the members than only organising annual meetings and publishing the *EJUHM*. There is a lot of good research done, the DAN group, the Brubakk group, Uwatec, Suunto, EDTC, etc. The role of the Society is to participate in this, to educate new young researchers in order to facilitate its future in diving and hyperbaric medicine. Research in diving medicine can have an impact on other science, for instance the research done in Norway showing that a single exercise before diving can diminish the number of circulating bubbles has an impact on sports medicine in general. EUBS has to expand on this. One step in this direction could be the expansion of our journal.

Peter Mueller, editor in chief of the EJUHM, announces the merger of *EJUHM* with *Diving and Hyperbaric Medicine*, formerly the SPUMS Journal (South Pacific Underwater Medicine Society). *EJUHM* will publish its final edition this year. After that, EUBS will collaborate with SPUMS, and maybe others, i.e., Asian societies, to expand *Diving and Hyperbaric Medicine*, the ultimate goal being to be rapidly indexed in Medline in order to attract more quality articles. The major focus of this new journal will remain diving medicine, as some HBOT papers should rather go into the journals of the corresponding medical specialties. Peter Mueller also reminds the members that this journal is their journal and that they need to submit quality material to it to make it a success. An increase of the membership fee, which amount is not yet known, will be necessary. The Executive Committee of the EUBS will write a letter of intention to SPUMS for a trial period of two years. Comments from the audience encourage this proposal. Alf Brubakk asks the members to send more quality articles in order to increase the chances of being indexed in Medline.

Peter Germonpré, webmaster of the Society's home page, reports some improvements aiming to ease communication between members and to increase the attractiveness of the site. A personal password will be attributed to each member allowing them to vote electronically and to update their personal data. The membership directory will be available online and a large English database of scientific articles, compiled and indexed by the German Hyperbaric and Diving Society, will be offered to the members. These modifications should be available by the end of this year. The inclusion of the proceedings of the annual meetings in PDF format will begin, starting with this year's material. The addition of previous proceedings is also planned. All members are requested to send their e-mail address to Tricia Wooding (patriciawooding@btinternet.com) immediately if they are interested in the new features of the Society's website.

The financial statement of the Society is depressing. Even though there is no audit report at this time, the account has been audited and found to be correct. The Society is poor with only £3,220 in our account.

This year the Zetterstöm award has been won by Dr Emmanuel Gempp and co-workers for their work entitled: Preventive Effect of Pre-dive Hydration on Bubble Formation. The poster has been awarded for its good methodology and immediate practical relevance to the field.

Alf Brubakk thanks Jacek Kot for his service to the Society as Member at Large.

The new Vice President and Member at Large of the Society are respectively Dr Peter Germonpré from Belgium and Dr Phil Bryson from the UK.

Next annual meetings are to be held in Graz (September 2008) and the UK (2009). Greece has cancelled its candidature for 2010 and will be replaced by either Turkey or Poland.

Some general comments from the audience express the need for more discussion time after the presentations. The organising committee expresses disappointment about the bad payment ethic of members, for congress fees as well as for membership fees.

Dr Adel Taher and his crew of volunteers are congratulated for an excellent congress and venue.

*Jörg Schmutz, Secretary*

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**Call for candidates: Member at Large 2008–2011**

Armin Kemmer ends his term as Member at Large this year.

All members of EUBS wishing to propose a candidate are invited to send a short presentation and CV of their candidate to the Secretary of EUBS, Joerg Schmutz (joerg.schmutz@hin.ch), copy to the President, Alf Brubakk (alf.o.brubakk@ntnu.no).

# Letters to the Editor

## NHMRC guidelines for clinical practice for ASD and PTSD

Dear Editor,

Recently I described the case of a scuba instructor suffering from acute stress disorder (ASD), a type of post-traumatic stress disorder (PTSD), following the death of one of her students.[1] The treatment described was a combination of eye movement desensitization and reprocessing (EMDR) and cognitive-behavioural therapy (CBT) exposure based exercises.

As it happens, in August the Australian Centre for Post Traumatic Mental Health published Australian clinical practice guidelines for ASD and PTSD.[2] These have been endorsed by the National Health and Medical Research Council (NHMRC). The treatment described in the diver injury case is consistent with these guidelines.

The NHMRC guidelines suggest that immediately following a traumatic episode (e.g., diver death or serious injury) the most helpful response is to offer psychological first aid. This includes providing information on traumatic stress reactions, encouraging self care and using available social support. It is recommended that the medical practitioner monitor the patient, watching for improvement, plateau or deterioration, and be ready to offer assistance or appropriate referral if needed.

The guidelines recommend the use of trauma-focused psychological therapy as the first-line intervention for ASD and PTSD. EMDR, with *in vivo* exposure included, and CBT are considered the most effective treatments. If medication is required, selective serotonin re-uptake inhibitor antidepressants are considered the best choice.

For the benefit and convenience of patients and practitioners, the NHMRC guidelines and a comprehensive set of information guides on ASD and PTSD are available online as pdf file downloads at <http://www.acpmh.unimelb.edu.au>. An update in *Medical Journal of Australia* provides traumatic stress information for medical practitioners including screening questions that can be used to identify patients suffering with ASD and PTSD.[3] This article is available online at <http://www.mja.com.au/public/issues/187_02_160707/for10467_fm.html>. Brief articles and summary sheets specifically for divers on the subjects of traumatic stress reactions, death and panic are available at <http://psychodiver.com>.

*Gary Ladd*
*Clinical Psychologist, Vancouver BC, Canada*
***E-mail:*** *<Gary@PsychoDiver.com>*

**References**

1 Ladd G. Treatment of psychological injury after a scuba-diving fatality. *Diving and Hyperbaric Medicine*. 2007; 37: 36-9.
2 Forbes D, Creamer M, Phelps A, Bryant R, McFarlane A, et al. Australian guidelines for the treatment of adults with acute stress disorder and post-traumatic stress disorder. *Aust New Zeal J Psychiatr*. 2007; 41: 637-48.
3 Forbes D, Creamer M, Phelps AJ, Couineau A-L, Cooper JA, et al. Treating adults with acute stress disorder and post-traumatic stress disorder in general practice: a clinical update. *Med J Aust*. 2007; 187: 120-3.

**Key words**
Letters (to the editor), psychology, trauma and stress

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## "Paradoxical embolism" – a misnomer?

Dear Editor,

I wish to comment on two articles published in the December 2007 issue. Firstly I sympathise with the plight of the European doctors wishing to perform diving medicals that are accepted by the Queensland Government.[1] As you will be aware SPUMS is not a regulatory body and had no input into the decision of the Queensland Government to use the SPUMS Diving Doctors List (DDL) as a reference list for doctors able to perform diving medicals. The DDL is a service offered to the SPUMS membership, and not all doctors who are members of SPUMS are included on the list. Rather it contains only those who have done the appropriate courses/training and then have applied annually to be listed. Doctors who are not members of SPUMS but are 'qualified' are also not on the DDL. To me the solution is easy for our European colleagues – become a SPUMS member and apply. As Dr J Wendling indicated the EDTC/ECHM standards are acceptable to SPUMS so the process would be simple. The application forms can be found on the SPUMS website, <www.spums.org.au>.

Secondly the term 'paradoxical embolisation' was used in the book review by Dr Boon von Ochsée.[2] It was an excellent review and has prompted me to buy the book but I would like to question the use of the word 'paradoxical' when referring to air embolism associated with a patent foramen ovale. I know it is a term frequently used and, therefore, I am not arguing with Dr Boon. However, the word 'paradox' means "statement contrary to accepted opinion;…person or thing conflicting with preconceived notions of what is reasonable or possible".[3] By this definition, surely there is nothing paradoxical about an air embolism associated

with two connected chambers of the heart – any connection would mean a mixing of the chamber's contents? I believe the term should be reserved for embolisation in which no communication between the atria or any other right-to-left communication is found.

*Christopher Acott*
*SPUMS President*

**References**

1 Wendling J. Recognition of diving medicals in Queensland. *Diving and Hyperbaric Medicine*. 2007; 37: 226. (letter)
2 Boon von Ochsée. Balestera C, Cronjé FJ, Germonpré P, Marroni A, editors. PFO and the diver. Flagstaff, AZ: Best Publishing Company; 2007. *Diving and Hyperbaric Medicine*. 2007; 37: 227-8. (book review)
3 *The concise Oxford dictionary*, 6th ed. Oxford: Oxford University Press; 1976. p. 798.

**Key words**
Letters (to the Editor), patent foramen ovale (PFO), medicals – diving

## Diving deaths – use words wisely

Dear Editor,

I have just been reading the article by Andrew McClelland in the last issue and am suffering from the horrors and sleepless nights![1] In case report SC 03-01, for example (the healthy [sic] diver with a BMI of 36.5), it is reported "the deceased rapidly found it hard to swim". Was this establishing rigor mortis? The thought of "the deceased panicked and had a knife in his hands" and later, 'the deceased was lunging with the knife" brings back to mind some zombie-type horror movies. Could you please give these articles a suitable warning rating?

*Dr Roger Capps*
*Senior Consultant, Department of Anaesthesia and Hyperbaric Medicine, Royal Adelaide Hospital*

**Reference**

1 McClelland A. Diving-related deaths in New Zealand 2000-2006. *Diving and Hyperbaric Medicine*. 2007; 37: 174-88.

**Key words**
Letters (to the Editor), diving deaths, writing – medical

**Editor's note:**

We apologise to Dr Capps for not editing "deceased" to read "victim", as the diver, indeed, was not dead at the time the described events were observed. Perhaps a censorship rating of 20+, extreme violence and some scenes may disturb, would be appropriate? Despite such flippancy, all readers must recognise the profound sadness with which this journal publishes these reports. The stated intention of such is to improve diving safety by learning from the errors of others. However, the fact that certain features of scuba and snorkelling fatalities and near misses crop up time after time after time suggests that recreational divers do not really learn from either the past mistakes of others or their own errors.

# The poetry doctor

## Lethal lessons

To read reports of diving deaths is depressing and quite frightening
And yet to scan the details is revealing and enlightening.
The stories of these deadly dives, so tragic and heart-rending
Expose a chain of small events that have this lethal ending.
Separation from a buddy or a sudden pain in ear,
Becoming over-weighted or a vis that's not too clear.
These provoke anxiety and the heart begins to race.
Air seems harder to inhale as the breathe increases pace.
And now a second problem looms, a loose or flooded mask,
Cramping muscles in the calf or a narcosis-clouded task.
Fatigue and cold, so commonplace, further muddle thinking,
Then panic rears its ugly face, both body and mind start sinking.
All early training is in doubt, the weight belt stays in place,
Finning fast till air runs out and drowning ends the race.
Alternatively the diver heads at full speed to the top,
Embolising on the way, ignoring safety stop.
The body is recovered and rescue is begun
Till the diver's declared dead in a sport that should be fun.
As I read I feel unease and start to wonder why?
Then realise that I'm sad for "There, but for luck, go I".
For I have made these dive mistakes; too deep, too long, too fast,
Or dived alone, run low on air, all common in my past.
Yet I survived to repeat dive for luck was on my side
As then the deadly chain stayed still or otherwise I'd died.
These thoughts are very humbling and I now appreciate
That I must keep fit and strong and my training up to date.
So if you want a tragic tale no need to read Macbeth.
Find your latest SPUMS Journal and read more diving deaths.

*John Parker*
*<www.thepoetrydoctor.com>*

# Book review

## Wound care practice, 2nd edition (Volumes 1 and 2)

### Paul J Sheffield and Caroline E Fife, editors

Hard cover, 602 (1) and 607 (2) pages
ISBN 1-930536-38-8 (1), 1-930536038-0 (2)
Flagstaff, AZ: Best Publishing Company; 2007
Price: US$189.00 + P&P
Copies can be ordered online from <www.bestpub.com> or from Best Publishing Company, P O Box 30100, Flagstaff, AZ 86003-0100, USA
**Phone:** +1-928-527-1055; **Fax:** +1-928-526-0370
**E-mail:** <divebooks@bestpub.com>

The skin requires a quarter to a third of the cardiac output, and is the largest organ in the body, with huge nutritional needs. When skin breakdown occurs this is often regarded as a "failure of care" regardless of the underlying health status of the patient. Wound care crosses most specialty boundaries, but tends to be regarded by doctors as the preserve of nurses, receiving scant attention in the training of most physicians and surgeons. This book aims to cross these boundaries. This is a comprehensive, well researched and referenced update from the first edition, with a focus on the assessment and management of both acute and chronic wounds using a modern wound care approach. Whilst wet-to-dry gauze dressings are, in practice, still the mainstay of wound care, anyone reading these volumes would be hard pressed to justify continuing such an archaic approach.

At 1,210 pages long, divided into two volumes, this is not a book to be read in its entirety, nor one for the beginner, but rather to use as a reference book covering the principles of wound management and the treatment of underlying causes and co-existing risk factors in the aetiology of chronic wounds. Despite 65 contributors, sourced from many basic and applied sciences and from clinical practice, it is reassuring that everyone seems to be dancing to the same tune. The text has 47 chapters arranged into six logical sections: the problem wound, principles of wound assessment and principles of wound management in the first volume, and pain, infection, adjunctive therapies, communication and trust and finally healthcare delivery (which has an American focus, but for which many of the principles are universal) in the second volume.

The information in each section is informative and presented in a logical manner, often extending our knowledge and understanding in areas of wound care with which we were not familiar. Each chapter is laid out in sub-sections with a brief chapter overview of topics to be covered prior to the in-depth information. Descriptions of the tests used in wound assessment are comprehensive, if somewhat repetitive. The authors demonstrate their relevance to clinical practice through detailed case studies, taking the reader through the processes of assessment and diagnosis (there is a strong emphasis on wound diagnostics), planning and applying treatment in a holistic manner and reviewing outcomes. There are useful mnemonics and management algorithms scattered throughout the text. Whilst there are several stand-out chapters, the one on biofilm should be read by every practising nurse and physician. Both editors work in the hyperbaric field, but many of the contributors do not. It is therefore pleasing to see hyperbaric oxygen therapy (HBOT) taking a small but rightful place in many of the chapters on the clinical management of specific wound problems. There is also a brief chapter devoted specifically to HBOT. Chapters are also devoted to many ancillary services, such as physiotherapy, occupational therapy, nutrition services, podiatry and orthotics.

Chapters are individually and extensively referenced, with review questions at the end of each allowing the reader to clarify their understanding of the topics discussed; a welcome addition missing from the first edition. Inevitability there is a degree of repetition between chapters written by different authors on related topics, but each approach tends to complement the other as they clarify areas discussed from different perspectives. The text is of a high quality with relatively few proof-reading errors. Colour has been introduced to the tables making them clearer. All photos are in colour and, whilst similar to the previous edition, the size of some has been altered sometimes to the detriment of clarity. Some of the photos need updating, for instance those showing old models of the same machine.

There are a few deficiencies. There is a definite need for an introductory chapter on the structure and function of normal skin and the processes of normal wound healing before embarking on the aetiology of the problem wound. The modern concepts of TIME and "wound bed preparation" as the core of chronic wound care teaching are not mentioned in the text. Certainly the same principles are here, but have not been brought together in the way they are now taught, at least in Australia and New Zealand. There is an irritating lack of consistency in quoting international units, as well as USA units, for various parameters. Correcting this would enhance the international flavour of future editions.

This is a highly relevant text for all involved in this field. It should be on the shelf of every wound care and diabetic centre, vascular, plastics and general surgery department, hyperbaric medicine unit, family medicine group practice and district nursing service in the world.

*Yvonne Denny, Charge Nurse Manager*
*Margaret Mossop, Vascular Nurse Specialist*
*Pam Mitchell, Wound Care Nurse Specialist*
*Michael Davis, Medical Director, Hyperbaric Medicine Unit, Christchurch Hospital, New Zealand*

**Key words**
Book reviews, wounds, chronic wounds, textbook

# Articles reprinted from other sources

## Acute decompression illness and serum s100ß levels: a prospective observational pilot study [Abstract]

Poff DJ, Wong R, Bulsara M

**Background:** S100ß, a calcium binding protein associated with astroglial cells and other tissues has been shown to be raised in the serum of patients with a number of neurological pathologies. As there are no published data on serum s100ß determinations in recreational divers affected by decompression illness (DCI) this pilot study determines whether s100ß is a possible biochemical marker of DCI worthy of further investigation.
**Methods:** Venous blood samples were drawn from patients diagnosed with, and treated for acute DCI at a hyperbaric facility and analysed for serum s100ß concentration and creatine kinase (CK) activity. Samples were taken at initial presentation, and again following final treatment.
**Results:** Twenty-one patients were included in the study. Neither s100ß, nor CK levels were significantly raised above population normal limits.
**Conclusion:** S100ß is not a clinically useful serum marker of acute DCI.

*Australasian College for Emergency Medicine and Royal Flying Doctor Service, Western Operations, Western Australia; Department of Diving and Hyperbaric Medicine Fremantle Hospital, Western Australia; and School of Population Health, University of Western Australia, Faculty of Medicine and Dentistry*

**Reprinted with kind permission from Poff DJ, Wong R, Bulsara M. Acute decompression illness and serum s100ß levels: a prospective observational pilot study. *Undersea Hyperb Med*. 2007; 34: 359-67.**

**Key words**
Reprinted from, decompression illness, decompression sickness, investigations

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## Influence of bottom time on preflight surface intervals before flying after diving [Abstract]

Vann RD, Pollock NW, Freiberger JJ, Natoli MJ, Denoble PI, Pieper CF

Previous trials of flying at 8,000 ft after a single 60 fsw, 55 min no-stop air dive found low decompression sickness (DCS) risk for a 11:00 preflight surface interval (PFSI). Repetitive 60 fsw no-stop dives with 75 and 95 min total bottom times found 16:00. Trials reported here investigated PFSIs for a 60 fsw, 40 min no-stop dive and a 60 fsw, 120 min decompression dive. The 40 min trials began with a 12:05 PFSI (USN guideline) which was incrementally reduced to 0:05 (three DCS incidents in 281 trials). The 120 min trials began with a 22:46 PFSI (USN guideline) which was reduced to 2:00 (nine incidents in 281 trials); 2:00 was rejected with six incidents. Low-risk PFSIs for the 40 min dive were nearly 12 hours shorter than for the 55 min dive, and low-risk PFSIs for the single 120 min decompression dive were 12 hours shorter than for the 75–95 min repetitive dives. With the dry, resting conditions of these dives, low-risk PFSIs appeared to be sensitive to dive profile characteristics such as bottom time, repetitive diving, and decompression stops. Whether this is so for wet, working dives is unknown.

*Center for Hyperbaric Medicine and Environmental Physiology, Divers Alert Network, Department of Anesthesiology, Center for Aging, Department of Biostatistics and Bioinformatics, Duke University Medical Center*

**Reprinted with kind permission from Vann RD, Pollock NW, Freiberger JJ, Natoli MJ, Denoble PI, Pieper CF. Influence of bottom time on preflight surface intervals before flying after diving. *Undersea Hyperb Med*. 2007; 34: 211-8.**

**Key words**
Reprinted from, flying (after diving), diving research, hyperbaric facilities, DAN – Divers Alert Network

## The Hyperbaric Research Prize

The Hyperbaric Research Prize has been introduced to encourage the scientific advancement of hyperbaric medicine and will be awarded annually whenever a suitable nominee is identified. It will recognise a scholarly published work or body of work(s) either as original research or as a significant advancement in the understanding of earlier published science. The scope of this work includes doctoral and post-doctoral dissertations. The Hyperbaric Research Prize is international in scope. However, the research must be available in English.

The Hyperbaric Research Prize takes the form of commissioned art piece and US$ 10,000.00 honorarium.

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

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## Third Congress of US–Japan Panel on Aerospace-Diving Physiology & Technology, and Hyperbaric Medicine (3rd New UJNR)

**Dates:** 7 to 8 November 2008
**Venue:** Grand Plaza Nakatsu Hotel, Nakatsu City, Japan
**Website:** <www.maruroku.co.jp/grandplaza/>
**Registration:** 20,000 yen
**Sponsors:** Japanese Society of Hyperbaric and Undersea Medicine <http://www.coara.or.jp/~gensin/3rdcongress/> and Undersea and Hyperbaric Medical Society

**Abstract submission:** 15 January–31 July 2008

**For further information contact the Congress office:**
Kawashima Orthopaedic Hospital, 14-1 Miyabu, Nakatsu city, Oita prefecture, Japan, 871-0012
**Phone:** +81-979-24-0464
**Fax:** +81-979-24-6258
**E-mail:** <newujnr_3rdcongress@yahoo.co.jp>

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## Latest results on insect repellents

A consumer report in the USA (June 2006) tested 18 repellents on human subjects by thrusting their arms into cages with 200 Aedes or Culex mosquitoes. In general, the higher the concentration of DEET in the repellent, the more effective the repellent. However, a 7% concentration lasted only about one hour. Most non-DEET repellents offer little protection. So if you are going to the ASM in PNG make sure you buy a repellent with a concentration of 20% or more of DEET.

## British Hyperbaric Association
The 2008 Annual Meeting, 20-23 November 2008

**Venue:** King's College Conference Centre, University of Aberdeen, Aberdeen

**Guest Speakers:** Professor John Yarnold, Institute of Cancer Research, Mr Richard Shaw, University of Liverpool, and Professor Richard Moon, Duke University

**For further information and on-line registration go to the BHA2008 website:**
<http://www.hyperchamber.com/BHA2008/index.html>
**For more information on the BHA go to:**
<www.hyperbaric.org.uk>

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**EAST OF ENGLAND HYPERBARIC UNIT**
**Hyperbaric Medicine**
**A Team Course for Health Care and Diving Professionals**

**Dates:** 14 to 18 April 2008
**Venue:** James Paget University Hospital, Norfolk
**Cost:** £475

40 hours of theory and practical experience with a multiplace chamber incorporating the BHA Core Curriculum. Approved for CHT and CHRN/ACHRN by The National Board of Diving & Hyperbaric Medical Technology (NBDHMT). Approved for CEPD points by the RCA.

**Course Director:** Roly Gough-Allen
**For information and application forms contact:**
Ms Tricia Wooding
**Tel:** +44-(0)208-5391222
**Fax:** +44-(0)208-5391333
**E-mail:** <mail@londonhyperbaric.com
**Website:** <www.londonhyperbaric.com>
**Postal address:** London Hyperbaric Medicine Ltd, Whipps Cross University Hospital, Leytonstone, London E11 1RG

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**2008 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS' UNDERWATER MEDICINE COURSE**

**Dates:** 10 to 21 November 2008
**Venue:** HMAS Penguin, Sydney
**Cost:** $1,833.00 (tbc)

The Medical Officers' Underwater Medicine Course seeks to provide the medical practitioner with an understanding of the range of potential medical problems faced by divers. Considerable emphasis is placed on the contra-indications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses.

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

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**AUSTRALIAN and NEW ZEALAND COLLEGE OF ANAESTHETISTS**
**Annual Scientific Meeting 2008**

**Dates:** 3 to 7 May 2008
**Venue:** Sydney Convention Centre, NSW

**Title: What anaesthetists and divers have in common**
Chair: Margaret Walker

1 The uptake, distribution and elimination of a novel anaesthetic agent – nitrogen: A/Prof. Mike Bennett
2 Rebreather diving – a portable anaesthetic machine with a twist: Dr Simon Mitchell
3 A tale of narcosis: Dr David Wilkinson
4 A sad ending – the limits of rebreather performance: Dr Simon Mitchell

There will be a Diving and Hyperbaric Medicine Special Interest Group session during the meeting.
**For additional information contact:**
**E-mail:** <emmab@icmsaust.com.au>
**Website:** <www.anzca.edu.au>

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**DIVING HISTORICAL SOCIETY AUSTRALIA, SE ASIA**

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

**ROYAL ADELAIDE HOSPITAL DIVER MEDICAL TECHNICIAN (DMT) & DIVING MEDICAL OFFICER COURSES 2008**

**DMT Courses**

**November/December 2008**
Unit 1: 24 – 28 November
Unit 2: 1 – 5 December
Unit 3: 8 – 12 December

**DMT Refresher courses 2008**
27 – 31 October

**Medical Officers Course 2008**

**Basic:** 16 – 20 June
**Advanced:** 23 – 27 June

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

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**ASIAN HYPERBARIC & DIVING MEDICAL ASSOCIATION**
**Annual Scientific Meeting 2008**

**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>

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**UNDERSEA & HYPERBARIC MEDICAL SOCIETY**
**Annual Scientific Meeting 2008**

**Dates:** 26–28 June 2008
**Venue:** The Salt Lake City Marriott Downtown, Salt Lake City, Utah

A pre-course '*Decompression and the Deep Stop*' will be offered on June 24-25 and a '*Wound Healing*' pre-course will be offered on June 25.

**Register online:** <www.regonline.com/UHMS-SLC08>
**For more information follow the link:**
<http://www.uhms.org/MeetingsEvents/2008AnnualScientificMeeting/tabid/97/Default.aspx>

# Instructions to authors

**(revised February 2008)**

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

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

**Requirements for manuscripts**
Documents should be submitted electronically on disk or as attachments to e-mail. The preferred format is Microsoft Office Word 2003 . Paper submissions will also be accepted. All articles should include a **title page**, giving the title of the paper and the full names and qualifications of the authors, and the positions they held when doing the work being reported. Identify one author as correspondent, with their full postal address, telephone and fax numbers, and e-mail address supplied. The text should generally be subdivided into the following sections: an **Abstract** of no more than 250 words, **Introduction**, **Methods**, **Results**, **Discussion, Conclusion(s), Acknowledgements and References**. Acknowledgements should be brief. Legends for tables and figures should appear at the end of the text file after the references.

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

The preferred length for original articles is 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. If figures are created in Excel, submit the complete Excel file. Large files (> 8 Mb) 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:

1 Freeman P, Edmonds C. Inner ear barotrauma. *Arch Otolaryngol.* 1972; 95: 556-63.
2 Hunter SE, Farmer JC. Ear and sinus problems in diving. In: Bove AA, editor. *Bove and Davis' diving medicine*, 4th ed. Philadelphia: Saunders; 2003. p. 431-59.

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

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

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

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## DIVER EMERGENCY SERVICES PHONE NUMBERS

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

**NEW ZEALAND**
**0800-4-DES111 (in New Zealand, toll-free)**
**+64-9-445-8454 (International)**

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

**EUROPE**
**+39-06-42118685 (24-hour hotline)**

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

**SOUTHERN AFRICA**
**outside South Africa +27-11-254-1112 (may be called collect)**
**within South Africa 0800-020-111**

**The DES numbers above are all generously supported by DAN**

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### DAN Asia-Pacific DIVE ACCIDENT REPORTING PROJECT

This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. 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>**

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### DIVING INCIDENT MONITORING STUDY (DIMS)

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

**Diving Incident Report Forms (Recreational or Cave and Technical)**
**can be downloaded from the DAN-AP website: <www.danasiapacific.org>**
**They should be returned to:**

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

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