Hyperbaric oxygen therapy in the treatment of skin ulcers due to calcific uraemic arteriolopathy: experience from an Australian hyperbaric unit

Mark Edsell, Michael Bailey, Keith Joe and Ian Millar

Key words
Calciphylaxis, hyperbaric oxygen therapy, chronic wounds, outcome

Abstract

Introduction: Calcific uraemic arteriolopathy (CUA), also known as 'calciphylaxis', is a syndrome of ischaemic necrotic ulcers occurring in uraemic patients with end-stage renal failure. It is a debilitating condition with a high morbidity and mortality. Hyperbaric oxygen (HBO) has been used to treat such wounds for many years but evidence of its efficacy is limited.

Aim: We aimed to study the efficacy of HBO on the healing of problem ulcers secondary to CUA.

Method: A retrospective case review of all patients with chronic skin ulcers secondary to CUA treated at the Alfred Hospital Hyperbaric Unit from July 1997 to March 2006 (n = 20).

Results: HBO was beneficial in eleven (55%) patients, with six of these (30%) experiencing complete resolution of their ulcers on completion of their treatment. Advancing age was identified as a predictor of a positive outcome (P = 0.02). There was no statistical correlation between the number of HBO treatments and ulcer healing.

Conclusion: HBO can benefit patients with chronic non-healing wounds secondary to CUA, but its precise role remains undefined.

Introduction
Calcific uraemic arteriolopathy (CUA), also known as 'calciphylaxis', is a syndrome of ischaemic necrotic skin ulcers caused by subcutaneous and small vessel calcification. It occurs mainly in patients with uraemia caused by end-stage renal failure (ESRF). Hypercalcaemia, hyperphosphataemia and hyperparathyroidism are also thought to be precipitating factors and can feature as part of the syndrome. The condition usually presents as nodules of subcutaneous calcification that develop into painful violaceous ulcers (livedo reticularis) which become progressively necrotic. Secondary infection of the ulcers is common in the setting of underlying uraemia and immune compromise. This can progress to sepsis, which is associated with a mortality of 60–80%. CUA more commonly affects females and younger patients on long-term dialysis and has a prevalence of up to 4% in patients undergoing haemodialysis.

The term calciphylaxis was first used by Sleye in 1962 when he induced metastatic soft tissue calcification in rats in a two-stage process. He first sensitised rats with Vitamin D analogues and parathyroid hormone (PTH) and then, after a critical period, challenged them with injections of irritant substances (egg albumin, iron) or local trauma. The areas of skin challenged developed the characteristic ulceration described above. The word was coined because the mechanism of calcification was proposed to be a hypersensitivity reaction, and originates from the combination of calcium and anaphylaxis.

Calciphylaxis was first described in humans by Rees and Coles in 1969 who found medial calcification and intimal hyperplasia of arterioles occurring in the presence of uraemia. The arteriolar calcification led to ischaemic tissue necrosis which then itself became calcified and necrotic. Skin breakdown and wound development was then triggered by minor trauma and progressed to extensive ulceration. In 1998, Coates et al found that the previously described derangements in calcium, phosphate and PTH were often not present in patients with calciphylaxis and so proposed the use of a more descriptive term of “calcific uraemic arteriolopathy” (CUA).

Hyperbaric oxygen (HBO) therapy involves breathing 100% oxygen at an elevated ambient pressure, usually 203–253 kPa. In hypoxic tissue, such as that found in and around problem ulcers, it restores tissue oxygen tension (P_{O_2}) to physiologically normal or supra-normal levels. This enhances fibroblast proliferation, collagen formation and angiogenesis to aid ulcer healing. Wound healing may also be enhanced by systemic effects such as the mobilization of vasculogenic stem cells. The elevation of P_{O_2} also improves neutrophil function and polymorphonuclear leukocyte-mediated bacterial killing of organisms commonly found in associated ulcer infection. It is by these mechanisms that HBO probably exerts its effect in CUA.

HBO has been used in the treatment of CUA wounds for many years but published evidence of its efficacy is limited to isolated case reports and small case series. The first case series by Podymow and colleagues reported complete
healing in two out of five patients with CUA. A year later Basile et al published a larger series of nine patients with distal lesions, eight of whom had their ulcers heal after an average of 41 HBO treatments. Not all reports have shown benefit, however, and HBO has failed to become a universally accepted treatment. This review aimed to study the benefit of HBO in those patients treated at the Alfred Hospital, Melbourne.

Methods

After submission to the Hospital Ethics Committee, the study was approved as a quality assurance project. All patients treated at The Alfred Hospital Hyperbaric Unit for chronic non-healing skin ulcers secondary to CUA from July 1997 to March 2006 inclusive were retrospectively studied. Patients were identified by searching the unit patient database (FileMaker Pro 5.5, Filemaker Inc, Santa Clara, California). Patient information was obtained regarding age, gender, cause for renal failure, presence of diabetes, dialysis type and the distribution of CUA lesions. Lesions were defined as either ‘proximal’, referring to lesions of the trunk, buttocks or upper thighs, or ‘distal’, referring to lesions of the arms and lower legs. Where both proximal and distal lesions were present, patients were assigned to the proximal group (being indicative of more extensive disease).

HBO data retrieved included the duration and number of treatments, and the degree of pressurisation. Patients typically received HBO for 90 minutes at between 203 and 243 kPa for five days per week. A therapeutic course of treatment was, for the purpose of this study, defined as more than two weeks (ten treatments) of therapy as this is the minimum prescribed to treat non-healing wounds of other aetiologies at our institution. Eight patients who received less than two weeks (ten treatments) of HBO were therefore excluded from further study. Patients were defined as having multiple treatment courses if there was a break in treatment of more than one month.

Efficacy of HBO was assessed using a six-point outcome score (Table 1), which was created from outcome measures used in the previous case studies. This was aided by photographs when available. In the absence of photographs, outcome was based on the wound assessment made in the medical record. Healing was defined as a stepwise improvement of the ulcers evidenced by resolution of necrotic areas, granulation, reduction in wound size, and subsequent scar formation. Outcome assessment was made when each patient’s treatment at the hyperbaric unit came to an end. Patient follow up was undertaken in mid 2008, aiming to evaluate mortality and progression of wounds subsequent to treatment.

Results were analysed using SAS version 8.2 (SAS Institute Inc., Cary, NC, USA). Relationships between continuous variables were determined using Spearman correlation coefficients, whilst relationships between continuous and categorical variables were determined using Wilcoxon rank sum tests. A two-sided P-value of 0.05 was considered to be statistically significant.

Results

Twenty-eight patients with a diagnosis of CUA were identified from the hyperbaric unit database. Eight of the 28 patients received ten treatments or fewer (median 2.5, range 0–7) and were excluded from further analysis. Reasons for discontinuation or refusal of therapy included patient refusal, anxiety/claudophobia, respiratory co-morbidity and withdrawal by referring medical staff for palliation. Of the remaining twenty patients, two patients had missing data pertaining to their calcium/phosphate levels. The mean age of the treated patients was 56 years (SD 10.7) and twelve were female. Study patient characteristics are summarised in Table 2. The study group received between 17 and 83 treatments (median 51, mean 50.6) and five patients received more than one course of HBOT (median 1, range 1–3).

Eleven of the 20 patients treated had improvement in their wounds with HBO (an outcome score of 3 or more), of whom six healed completely. The five patients who received multiple courses all showed improvements in healing by the end of each course, only to subsequently deteriorate and require further treatment. Surgical debridement, skin grafting, antibiotics and wound care were administered as indicated.

In half of the patients, the decision to stop HBO was due to factors external to the hyperbaric physician. Reasons included being stopped by referring physician (three patients), withdrawal for palliation (two patients), attendance issues (two patients), patient decision (two patients) and death due to other causes (one patient). No demonstrable dose-response relationship was seen between the number of HBO sessions and ulcer healing (P = 0.42).

There were no statistically significant correlations between wound healing and gender, diabetic co-morbidity, or the type of dialysis therapy used to treat the renal failure (Table 3). Similarly, normal serum calcium and potassium levels at the time of assessment, or a history of parathyroidectomy,
Table 2  
Clinical data for patients undergoing HBO for skin ulcers secondary to CUA (n = 20)

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Aetiology</th>
<th>Type of dialysis</th>
<th>Previous PTH surgery</th>
<th>Serum Ca²⁺/PO₄⁻</th>
<th>Ulceration site (biopsy-proven)</th>
<th>HBO treatments</th>
<th>Outcome score</th>
<th>Additional therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>45</td>
<td>M</td>
<td>Diabetic nephropathy</td>
<td>PD</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>77</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>31</td>
<td>F</td>
<td>Lupus nephritis</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Proximal (Yes)</td>
<td>76</td>
<td>1</td>
<td>Debridement</td>
</tr>
<tr>
<td>73</td>
<td>F</td>
<td>Diabetic nephropathy</td>
<td>PD</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>75</td>
<td>4</td>
<td>Debridement and skin grafting</td>
</tr>
<tr>
<td>42</td>
<td>F</td>
<td>Glomerulonephritis</td>
<td>PD</td>
<td>Yes</td>
<td>Normal</td>
<td>Proximal (No)</td>
<td>37</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>M</td>
<td>IgA nephropathy</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (Yes)</td>
<td>52</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>42</td>
<td>F</td>
<td>Hypertension</td>
<td>HD</td>
<td>No</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>45</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>63</td>
<td>M</td>
<td>Hypertension</td>
<td>HD</td>
<td>No</td>
<td>Elevated</td>
<td>Distal (Yes)</td>
<td>60</td>
<td>5</td>
<td>Debridement and skin grafting</td>
</tr>
<tr>
<td>58</td>
<td>M</td>
<td>Diabetic nephropathy</td>
<td>HD</td>
<td>Yes</td>
<td>Unknown</td>
<td>Distal (Yes)</td>
<td>20</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>65</td>
<td>F</td>
<td>Lupus nephritis</td>
<td>None</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (Yes)</td>
<td>79</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>46</td>
<td>M</td>
<td>Reflux nephropathy</td>
<td>None</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>33</td>
<td>0</td>
<td>Debridement and BKA</td>
</tr>
<tr>
<td>58</td>
<td>F</td>
<td>Hypertension</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>83</td>
<td>2</td>
<td>Debridement</td>
</tr>
<tr>
<td>58</td>
<td>F</td>
<td>Reflux nephropathy</td>
<td>HD</td>
<td>No</td>
<td>Elevated</td>
<td>Proximal (No)</td>
<td>33</td>
<td>2</td>
<td>Parathyroidectomy</td>
</tr>
<tr>
<td>62</td>
<td>M</td>
<td>Diabetic nephropathy</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Proximal (Yes)</td>
<td>79</td>
<td>3</td>
<td>Debridement</td>
</tr>
<tr>
<td>67</td>
<td>F</td>
<td>Obstructive nephropathy</td>
<td>HD</td>
<td>No</td>
<td>Unknown</td>
<td>Proximal (No)</td>
<td>54</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>68</td>
<td>F</td>
<td>Hypertension</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Proximal (Yes)</td>
<td>50</td>
<td>5</td>
<td>Surgical debridement</td>
</tr>
<tr>
<td>53</td>
<td>F</td>
<td>Reflux nephropathy</td>
<td>HD</td>
<td>Yes</td>
<td>Normal</td>
<td>Distal (Yes)</td>
<td>17</td>
<td>3</td>
<td>Died during HBO (sepsis)</td>
</tr>
<tr>
<td>57</td>
<td>M</td>
<td>Diabetic nephropathy</td>
<td>HD</td>
<td>No</td>
<td>Normal</td>
<td>Distal (Yes)</td>
<td>35</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>64</td>
<td>F</td>
<td>Polycystic kidneys</td>
<td>PD</td>
<td>No</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>22</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>49</td>
<td>M</td>
<td>Diabetic nephropathy</td>
<td>PD</td>
<td>No</td>
<td>Normal</td>
<td>Distal (No)</td>
<td>28</td>
<td>3</td>
<td>Debridement</td>
</tr>
<tr>
<td>62</td>
<td>F</td>
<td>Glomerulonephritis</td>
<td>HD</td>
<td>No</td>
<td>Normal</td>
<td>Distal (Yes)</td>
<td>57</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

PD – Peritoneal dialysis, HD – Haemodialysis, PTH – parathyroid, BKA – below-knee amputation
Table 3
Statistical correlation between patient characteristics and skin ulcer healing (outcome) when treated with HBO

<table>
<thead>
<tr>
<th>Patient characteristic</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advancing age</td>
<td>0.02</td>
</tr>
<tr>
<td>Gender</td>
<td>0.73</td>
</tr>
<tr>
<td>Number of HBO treatments</td>
<td>0.42</td>
</tr>
<tr>
<td>Site of CUA lesion</td>
<td>0.39</td>
</tr>
<tr>
<td>Type of dialysis</td>
<td>0.42</td>
</tr>
<tr>
<td>Co-existing diabetes</td>
<td>0.54</td>
</tr>
<tr>
<td>Abnormal Ca²⁺/PO₄⁻</td>
<td>0.94</td>
</tr>
<tr>
<td>History of previous parathyroidectomy</td>
<td>0.63</td>
</tr>
<tr>
<td>CUA proven on biopsy</td>
<td>0.45</td>
</tr>
</tbody>
</table>

was not a predictor of a successful outcome. There was no correlation between the site of the lesion and a worse outcome (P = 0.39). Advancing age was the only predictor of positive outcome (P = 0.02), older patients tending to do better with HBO.

Follow-up information was available for 16 of the 20 patients. Ten had died and six remained alive at a mean follow-up of 51 weeks. Of the nine patients who had not responded to HBO (scores of 1 or 2), two were unable to be contacted and the other seven had died; two patients immediately followed cessation of HBO and withdrawal of active therapy and, in the remaining five, the longest survival was 15 weeks. In contrast, of the five patients who had healed at the end of their courses of HBO, the four who were contactable were alive and had remained healed after 12, 34, 46 and 89 weeks. One had experienced new CUA lesions at a previously unaffected site but these had again responded completely to HBO treatment. Of the four patients with a very limited response to HBO (score of 3), one was not contactable, and one died of systemic sepsis shortly after ceasing HBO, but the other two continued to heal and remained alive after 27 and 98 weeks. The two patients who substantially, but not completely, healed (score of 4) both survived for a further four years.

Discussion

In our study, the age, sex distribution and prevalence of diabetes in the patients were all similar to those of the two previous published series. However, we were unable to reproduce a healing rate of 89% (8 of 9 patients) described in Basile’s case series. By comparison, only 30% of our patients experienced a complete resolution of their ulcers, similar to the original series by Podymow et al. This study, combined with our own, probably represents a more accurate picture of the healing rate for CUA ulcers. Historically, a worse outcome has been associated with patients with proximal lesions. In our study, more patients had proximal lesions (six patients), but on analysis we found no correlation between the site of the lesion and a worse outcome.

The high mortality at follow up probably reflects the natural history of patients who develop CUA but there are insufficient data in our series and in the literature to confirm this. Response to HBO appears to be a predictor of mortality, the eight early deaths all occurring in patients with poor or no response.

Of the patient factors studied, age was a significant predictor of success with older patients being more likely to have a positive outcome with HBO. The exact reason for this cannot be ascertained from our study. However, it may be reasonable to postulate that more aggressive forms of CUA, renal failure, or of the underlying cause of said renal failure are seen in younger patients. Whatever the reason, it would be difficult to base a decision to treat with HBO on age alone – the patient with the most dramatic turnaround from life-threatening, extensive proximal disease to good long-term function was only 45 years old.

Regarding HBO, all treatments were given at pressures between 203 and 243 kPa, but there was no relation between the pressure or number of treatments and outcome. Thus the selection of which patients will respond to HBO, and the most effective treatment regimen (number of sessions) remains largely inconclusive.

A multimodal, multidisciplinary approach to treatment is usual for CUA. The complex nature of calciphylaxis and its co-morbidities can produce many barriers to successful treatment. In half of our patients, the decision to stop treatment was not made by the hyperbaric physician. For some patients, receiving daily hyperbaric oxygen therapy in conjunction with intermittent dialysis and inter-hospital transfer is too demanding. Close liaison between patient, renal unit and hyperbaric unit is essential to improve efficiency and minimise the chance of patients missing valuable dialysis or HBO. During periods of HBO, many of our patients received dialysis at The Alfred to avoid patients spending time and energy commuting between treatment centres.

In addition to providing HBO, most hyperbaric units can provide an important coordinating role due to their position of being able to review patients daily and follow the progress of healing. Routine wound care and antibiotics when necessary are administered. CUA skin ulcers are often associated with severe ischaemic pain and distress. Pain management and psychological support are other aspects of treatment well managed by hyperbaric physicians many of whom have a background in anaesthesia.

Referral for surgical debridement and skin grafting may also be necessary. Half of our patients healed after skin grafting. A key role for HBO may be as a pre-operative optimizer of the wound base by improving the microcirculation and encouraging healthy granulation prior to skin grafting. However care must be taken in patients with widespread
calcific deposits. The donor site must first be investigated for the presence of calciphylaxis as new ulcers can be generated from the surgical insult. On the evidence of this study, grafting may be best reserved for older patients with isolated lower limb CUA.

Control of calcium and phosphate levels and surgical parathyroidectomy are recognised treatments for CUA. A retrospective case series by Angelis and colleagues reported an 80% healing rate in patients who underwent parathyroidectomy. This was reinforced by a recent study by Duffy and colleagues who retrospectively studied two cohorts of patients with CUA ulcers. Those treated with total or partial surgical parathyroidectomy (six patients) demonstrated a 100% healing rate compared with those treated with medical therapy (nine patients) of whom only two had improvement in the skin lesions. The surgery group also showed a survival benefit over the 80-month follow up (median survival 39 versus three months using Kaplan-Meyer analysis). The majority of our patients had previously undergone parathyroidectomy and had good control of calcium and phosphate prior to starting HBO. Given this was a group who had largely failed to show ulcer healing despite ‘conventional’ therapy our 30% healing rate may represent significant benefit, but without comparative controls no definitive conclusions can be drawn.

A common observation in our patients was that the severe pain often associated with CUA lesions usually diminished and in some cases ceased after several weeks of treatment with HBO, but unfortunately objective scoring of this was not undertaken. This is something which deserves further study: if HBO can contribute to an improved quality of life by reducing pain then it may be justifiable, even in some terminal cases. It is important to qualify this with the fact that for some patients, the logistics of travel to a hyperbaric facility could represent an inappropriate burden during the final weeks of life.

When examining therapeutic options for CUA in the absence of higher order evidence, it is useful to consider mechanisms of action. Medical and surgical therapies involve controlling metabolic derangement to arrest or reverse the process of systemic calcification. In contrast HBO acts to reverse tissue hypoxia at the wound site and as such can be considered a local treatment for hypoxic ulceration but not for the underlying CUA (Table 4). However, a further observation in our patients was that long-term responders reported softening of their tissues. This may represent some reversal of the CUA process but assuming any causal relationship would be inappropriate at this stage.

The pathogenesis of CUA is incompletely understood. However, it is reasonable to postulate that calcium deposition in small vessels will lead to local tissue hypoxia around the wound margins akin to many chronic wounds. Restoration of tissue normoxia, the goal of therapy in hypoxic wounds, may be difficult to achieve in patients with arterial calcification leading to a reduced macro-vascular blood flow. Any benefit demonstrated from HBO in calciphylaxis seems just as likely to be due to enhanced neutrophil function in patients with multiple barriers to healing, including diabetes and uraemia. In addition, optimising neutrophil function may render the patient resistant to secondary infection, the commonest cause of death in CUA.

This study reflects our belief that prolonged treatment with HBO is often required to heal these wounds. We believe this probably induces microvascular angioeneogenesis not only in hypoxic areas at the wound site but also at other sites of calcification. This may in turn provide some degree of protection from ischaemic injury to these sites in the future.

The results of this study can be compared with the recently published interim analysis of the study by Hawkins et al, which evaluates outcomes of chronic hypoxic wounds treated with HBO. This prospective study of 110 patients showed an overall good response in 52.3% receiving HBO. Skin ulcers due to CUA were not examined as a subgroup in this report but analysis of wounds caused by peripheral vascular disease and venous disease demonstrated a good response in 23.5% and 45.5% respectively at the end of treatment with HBO. The healing rate of 30% in our study is, therefore, consistent with these results and supports the view that whilst HBO is not a cure-all for refractory wounds, it is at least one treatment option for patients with skin ulcers secondary to CUA. However, patients should be informed of the potentially prolonged treatment course that may be required and the effect this may have on other aspects of their ongoing medical care.

For the future, there have recently been a number of case reports of treatment of CUA with agents aimed at modifying calcium and/or phosphate kinetics, including chelating agents and bisphosphonate therapy. It seems reasonable to speculate that such strategies could be synergistic with

<table>
<thead>
<tr>
<th>Table 4</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Therapeutic options for patients with CUA</strong></td>
</tr>
<tr>
<td><strong>Systemic therapies</strong></td>
</tr>
<tr>
<td><strong>Medical</strong></td>
</tr>
<tr>
<td>Reduce dialysate calcium</td>
</tr>
<tr>
<td>Anticoagulation</td>
</tr>
<tr>
<td>Phosphate binders</td>
</tr>
<tr>
<td><strong>Surgical</strong></td>
</tr>
<tr>
<td>Parathyroidectomy (partial or total)</td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

http://archive.rubicon-foundation.org
HBO in managing this difficult problem but this remains to be explored.

Limitations of our study are those inherent in all retrospective reviews, such as record keeping and a lack of comparative controls. Patients who received more than one treatment course all showed improvement with each course but analysis was done on the patient’s eventual outcome rather than that of the individual course, as this would have positively biased the outcome. Out of necessity, the outcome score was developed from previously published wound assessment outcomes but has not been validated. More robust, prospective studies of CUA therapeutics will probably need to be multi-centred and given the difficulty in organizing and funding randomized trials, this would seem an ideal area for a well-designed registry.

Conclusion

In a proportion of our CUA patients, HBO was beneficial in improving ulcer healing and resolution. However, this study does not support the healing rates achieved in the largest previous study. The ability to predict from clinical signs and symptoms which patients will benefit, and the most effective HBO regimen remains undefined. Given CUA is a painful and debilitating condition, it remains reasonable to consider HBO as part of the multidisciplinary, multimodal treatment approach in those patients able to undertake the prolonged treatment that is often required. Prospective, case-controlled trials are needed to clearly define the healing effect of HBO in this condition.

Acknowledgements

The authors would like to thank Cate Venturoni RN for her assistance with data collection and patient follow up.

References


Submitted: 03 November 2006
Accepted: 13 August 2008

Mark Edsell, MB ChB FRCA, was, at the time of this study, a registrar in Hyperbaric Medicine at The Alfred Hospital, Melbourne.

Michael Bailey, PhD MSc BSc (hons), is Statistical Consultant, Department of Epidemiology & Preventive Medicine at Monash University, Melbourne.

Keith Joe, MB ChB FACEM, is Staff Specialist in Emergency Medicine at the Royal Melbourne Hospital.

Ian Millar, MB BS FAFO M, is the Unit Director, Hyperbaric Medicine, The Alfred Hospital, Melbourne.

Address for correspondence:
Dr Mark Edsell, Clinical Fellow in Anaesthesia, The Heart Hospital Westmoreland Street London W1G 8PH England

Phone: +44-(0)20750-43160
Fax: +44-(0)20750-48847
E-mail: <markedsell@doctors.org.uk>
Review article

Compressed breathing air – the potential for evil from within
Ian L Millar and Peter G Mouldey

Key words
Air, compressors, carbon monoxide, toxins, exogenous poison, clinical toxicology, standards

Abstract

Human underwater activities rely on an adequate supply of breathable compressed gas, usually air, free from contaminants that could cause incapacitation underwater or post-dive or longer-term health effects. Potentially fatal but well-known hazards are hypoxia secondary to steel cylinder corrosion and carbon monoxide (CO) poisoning due to contaminated intake air. Another phenomenon may be behind some previously unexplained episodes of underwater incapacitation and perhaps death: low-level CO poisoning and/or the effects of gaseous contaminants generated within the compressor, including toluene and other volatile compounds. Many low molecular weight volatile contaminants are anaesthetic and will be potentiated by pressure and nitrogen narcosis. In sub-anaesthetic doses, impaired judgement, lowered seizure threshold and sensitisation of the heart to arrhythmias may occur. Toxic compounds can be volatilised from some compressor oils, especially mineral oils, in overheated compressors, or be created *de novo* under certain combinations of temperature, humidity and pressure, perhaps catalysed by metal traces from compressor wear and tear. Most volatiles can be removed by activated carbon filtration but many filters are undersized and may overload in hot, moist conditions and with short dwell times. A compressor that passes normal testing could contaminate one or more cylinders after heating up and then return to producing clean air as the filters dry and the systems cool. The scope of this problem is very unclear as air quality is tested infrequently and often inadequately, even after fatalities. More research is needed as well as better education regarding the safe operation and limitations of high-pressure breathing air compressors.

Introduction

Contamination of a diver’s breathing air makes diving unpleasant at the very least and, in the worst cases, can prove fatal. In between these extremes, contamination may cause impairment of judgement and consciousness predisposing to accidents underwater, it can trigger cardiac arrhythmias or lower seizure threshold and may cause headache or respiratory compromise. Some long-term health effects of inhaled contaminants are also possible, but will not be addressed in this paper.

The diving environment carries special risks with respect to the use of compressed air breathing apparatus, as the increased pressure associated with depth results in a proportional increase in the partial pressure of gaseous contaminants breathed for any given volumetric level of contamination. Air that is acceptable at the surface may thus become toxic as the diver descends. To assist in preventing air-contamination deaths and critical incidents, as well as avoiding long-term health effects, various authorities publish divers’ air quality standards which specify the maximum levels of various contaminants. These are generally set at a more stringent level than used for compressed air breathed at normobaric pressure.

The risk of compressor-intake air being contaminated is generally well understood, although installations are still periodically noted where intakes are capable of entraining engine exhaust or volatile contaminants such as paint and solvent vapours. What appears less well known, however, is the potential for generation of such contaminants from within the compression process and the limitations and failure modes of commonly used filtration systems. This paper aims to summarise some of these issues in the hope that readers will play a part in minimising future risks for divers.

A chance meeting between the two authors led us to conclude that there was a widespread lack of knowledge regarding the potential for compressed air to be contaminated and that it is very likely that some, and possibly many, compressed air contamination-related deaths have been unrecognised and unreported. We have, therefore, drawn upon our experience and a wide range of reference sources to prepare this overview document for readers of *Diving and Hyperbaric Medicine*. This is not a formal literature review, but a summary of issues and a set of personal recommendations that we hope will stimulate further research and discussion, and help with the ongoing refinement of standards, procedures and training aimed at minimising risk for divers and others, such as fire fighters, who rely upon compressed breathing air.
Oil mists, water vapour and particulates

The principal contaminants of concern are carbon monoxide and volatile hydrocarbons. With respect to oil mist and droplets, the lubricants used in breathing air compressors are generally chosen for their biological inertness and it has usually been assumed that small amounts of aerosolised liquid should be well tolerated. Data specifically addressing compressor oils are limited, so some caution remains appropriate. Water vapour itself is desirable with respect to the diver’s airways but must be limited to avoid regulator freezing for cold-water divers and to reduce corrosion in steel cylinders. Dry air also inhibits the growth of bacteria in compressed air systems. More importantly with respect to this discussion, moisture substantially degrades adsorption filter function and is an indicator of systems at risk of supplying contaminated air. The degree of dryness required depends significantly upon pressure and compressed air standards and guidelines usually take account of this. To date it has generally been considered that particulates are well captured by filtration but this may not be so: pollens have been reported to have contaminated scuba cylinders, triggering dyspnoea in an individual with borderline airway hyper-responsiveness.\(^1\) It can be speculated that micro-particles might have other health effects and might even act as catalysts for chemical transformation of oils and other contaminants within the compressor system but little information seems available on this subject at this time.

Air quality standards

There are some major differences between jurisdictions with respect to the testing methodology required and acceptable contaminant levels for compressed breathing air. The most dramatic difference is that both Canada and the United States set standards for volatile hydrocarbons and require air samples to be tested by a laboratory whilst the UK, most of Europe, Australia and New Zealand allow field testing with colorimetric tubes and do not specifically call for volatile contaminant testing, instead setting only general requirements for divers’ air to be free from toxic contaminants (Table 1).

There are also requirements for there to be “no odour” or “no objectionable odour”. The lack of a specific volatile hydrocarbon test requirement in Europe and Australasia means that, although subjective, odour detection is extremely important as at very low concentrations these substances will not be detected by testing with oil detector tubes but will generally have a detectable smell.

Carbon monoxide (CO)

CO is a colourless, odourless product of combustion which reversibly binds haemoglobin, inhibiting oxygen transport and resulting in a chemically induced hypoxia. Excess CO interacts with various intracellular energy-chain enzymes causing oxidative stress and direct cellular toxicity. CO is also a feature of normal biology, being generated from haem by the action of haem oxygenase. Endogenous CO acts as a neurotransmitter and appears to have anti-oxidant and cytoprotective qualities that are only now being elucidated. Some of the toxic effects of CO poisoning probably result from disruption of this normal CO biology but at low levels exogenous CO may not be as detrimental as previously thought, provided the person is at rest and at the surface. Clinical trials have been commenced using CO as a therapeutic gas to modulate processes such as ischaemia-reperfusion injury. Interestingly, the doses used have been as high as 500 ppm, 10 times higher than commonly set as the occupational health and safety limit for ambient air.\(^2,3\) These trials have triggered criticism as such levels have been demonstrated to carry significant risk in patients with cardiovascular or respiratory disease.\(^4\) Even relatively low levels of CO inhalation therefore remain undesirable for older or unfit divers as uptake will increase with depth and exertion, bringing with it an increased risk of cardiac ischaemia and/or arrhythmia. Also critical to the case for avoiding low-level CO inhalation is the significant reduction in exercise tolerance that results.\(^5\)

Volatile hydrocarbons

Volatile hydrocarbons are those lower molecular weight hydrocarbons that exist in the gaseous form at temperatures
associated with diving. These should be differentiated from the higher molecular weight hydrocarbons and fluorocarbons that are used as lubricants in compressors and to lubricate seals and breathing equipment components.

One of the most common groups of volatile hydrocarbons of concern is the so-called BTEX group: benzene, toluene, ethylbenzene and xylene. These are most commonly found in petroleum fuels and as solvents in paints, paint thinners, inks, degreasers and cleaning fluids. Unlike oil mists and droplets, which do not generally pass beyond the lungs, volatile hydrocarbons are in gaseous form at body temperature and are readily absorbed and widely distributed in the body, including the brain, in the same manner and with similar pharmacokinetics to volatile anaesthetic agents. They also act as anaesthetic agents; albeit with a less than ideal side-effect profile. They often cause initial excitation and increased cardiac irritability, predisposing to arrhythmias and sudden cardiac death, in addition to the high risk of death if divers achieve anaesthetic levels resulting in loss of consciousness underwater. At intermediate levels, cognitive impairment may increase the risk of lack of buoyancy control or other errors that may lead to injury or death. Some agents appear to lower seizure threshold.6

Although all volatile hydrocarbons can cause such problems, one of the most discussed reports of diving air contamination involved toluene.7 This readily vaporised liquid is most commonly encountered as a major component of automotive petrol but is also used as a cleaning solvent, in paints and in glues including some dive suit repair glues. The lethal concentration for human toluene exposure has been estimated at 1,800–2,000 ppm for one hour but for a scuba diver underwater the threshold for impairment is probably more important.5 The American Industrial Hygiene Association’s ERPG-2 limits estimate the airborne concentration limits at which individuals are still capable of taking protective action. For toluene, this is 300 ppm and the US Occupational Health and Safety Authority sets a ‘Permissible Exposure Limit’ of 200 ppm.9

For divers’ air, the British version of EN 12021 recommends that contaminant levels should be below 10% of the eight-hour time-weighted average (TWA) allowed for surface workplaces.10 Using this principle would suggest the danger limit for toluene in divers’ air may be around 20–30 ppm and the recommended limit may be as low as 2–5 ppm; from 10 per cent of the TWA up to 10 per cent of the “no observed adverse effects level” which appears to be around 50 ppm.11 It is likely that any compressor-generated contamination would contain a mix of toxic substances and their effects are almost certainly additive, if not synergistic. In addition to this, nitrogen will further potentiate the consciousness-impairing effects. This provides a good basis for the conservative limits for volatile hydrocarbons as adopted in the United States and even more stringently in Canada.

Other volatile solvents

In addition to the BTEX group, many other agents used as industrial cleaning fluids are highly volatile and very narcotic if inhaled. Examples are acetone and chlorinated solvents such as trichloroethylene. If these are not adequately removed from compressor systems they can show up in compressed air.

Production of CO and volatile hydrocarbons inside compressors

In oil-lubricated compressors, small quantities of oil entering the compression chambers can undergo oxidative and/or thermal breakdown given the presence of oxygen (within the air being compressed) and/or sufficient heat. Some hydrocarbon breakdown pathways may also require water vapour and catalysts, which can be provided by intake air humidity and traces of metals from the wearing of compressor components respectively. This problem is most common in high-pressure (HP) compressors used for filling scuba cylinders as these are usually oil-lubricated and higher heat production is a necessary by-product of high pressure compression. HP compressors are usually three- or four-stage, air-cooled machines with inter-coolers between compression stages. Overheating is a relatively common problem, however, particularly in hot climates and when small compressors designed for filling individual cylinders are used continuously, for instance in low-budget commercial or club diving installations. HP compressors should be fitted with over-temperature alarms with automatic shut-down mechanisms but this is not always the case.

It has been hypothesised that the particular toxic oil breakdown products produced from any one oil type and compressor combination may depend upon a critical temperature range. Partial pressure of oxygen (determined by air pressure), water content and trace-metal catalyst type may also be important. An overheated or poorly maintained compressor can thus be considered analogous to an oil refinery catalytic cracking tower, which breaks down higher molecular weight hydrocarbons into smaller molecules, including acidic gases and other toxic compounds. This concept allows for a phenomenon of major concern: a compressor that produces clean air when cool may produce dangerous contamination of one or more cylinders as it heats up under load. Where cylinders are individually filled this could produce seemingly random contamination that would escape detection by routine air quality testing of the compressor with air samples taken shortly after a cold start. Where the compressor fills a large capacity air bank, any short period of contaminant production may be of less concern, as the contaminated air will be diluted in the bank with all fills from the bank receiving low levels of contamination rather than one or two cylinders being filled to a level associated with the potential for underwater toxicity.
The oil-type debate

Much is made of the advantages of one oil type over another for breathing-air compressors. Most HP compressor oils are of mineral or synthetic origin as these are generally more stable at higher temperatures. Some are classified as ‘food grade’. Suitability for ingestion does not, however, imply that any particular oil is less harmful for inhalation. Oil droplet and mist contamination of breathing air is universally limited by standards and, in practice, any non-volatile oil mist inhalation that occurs seems to be relatively well tolerated. The widely used United States ACGIH limits for mineral oil contamination of ambient breathing air in a work place allow time-weighted averages of 5 mg.m⁻³ for an eight-hour working day. These sorts of levels are most commonly encountered in machine shop workers operating equipment such as lathes and metal milling machines, where the cutters and the item being drilled or shaped are cooled and lubricated by a spray of ‘cutting oil’.

Whilst contamination of breathing air by mists of lubricant oil is not unknown in the setting of on-line, low-pressure, surface-supply diving compressors, the major concern for HP compressors is the stability of lubricants at high pressures and temperatures and avoidance of breakdown into volatile toxic by-products. It may be that newer synthetic oils with increased anti-oxidant and thermal resistance properties are the least likely to be broken down into lower molecular weight gases. However, it remains difficult to compare manufacturers’ product claims, given the non-standardized testing regimens employed. Unfortunately there is limited reporting of the actual composition of oils as this is generally considered proprietary information. Currently, the best available guidance is to review both the oil suppliers’ and the compressor manufacturers’ guidelines when selecting the oil for a breathing-air compressor installation.

Filtration

Compressor first-stage intakes should be protected by dust filters. Inlet-air carbon dioxide (CO₂) absorption is also sometimes employed, especially in urban or industrial settings where elevated CO₂ levels may be such as to represent a risk when breathed at up to six times atmospheric pressure. (CO₂ is not removed by standard post-compression filter systems).

Considerable quantities of oil/water mist are produced during post-compression cooling of compressed air and HP compressors typically employ oil-water separators immediately after some or all of the three or four compression stages. The oil-water condensate that is drained carries with it some of the contaminant load but the higher the intake air humidity, the more water will need to be removed and this is critical, as water logging of downstream adsorbents such as activated charcoal (AC) severely degrades their function. Some more elaborate HP installations include refrigerated dryer/separators after the final compression, providing improved protection against water overloading of the final filters.

After it is compressed, air requires filtration before it will be suitable for breathing. Filtration can be relatively minimal for low-pressure compression of clean air using ‘oil-free’ compressors but for high-pressure compressors, systems are needed that will eliminate not only particles but also the oil mists, residual water vapour and any hydrocarbon contamination or carbon monoxide produced by the compression process. This requires a number of different components and these may be contained in separate filter elements or combined into combination cartridges.

In addition to particle filtration, filter systems usually incorporate a desiccant bed to remove residual moisture. An AC bed then removes volatile hydrocarbons plus odour. AC beds are capable of adsorbing most low molecular weight hydrocarbons and their performance is, therefore, critical in the prevention of contamination of breathing air. AC beds have a number of critical performance factors and failure modes, however. Their performance degrades over their normal service life, but degradation increases dramatically with excessive adsorption of hydrocarbons, with elevated temperature or if desiccant failure allows moisture through to the AC bed.

AC does not absorb CO and, therefore, better filter systems incorporate a catalytic element such as a manganese/copper oxide combination (Hopcalite), which oxidises CO to CO₂ with the small amounts of additional CO₂ generated usually not presenting a problem for the diver. As with AC, most catalytic beds function best with very low moisture contents and some are extremely moisture sensitive.

The need for dry AC and catalyst means that the overall filter-system processing capacity is often limited by the type and volume of desiccant in the cartridge, and another feature of better quality filter systems is, therefore, larger capacity desiccant and AC beds to provide some redundancy. Probably the most critical factor affecting filter performance, however, is the temperature of the air entering the desiccant and AC elements. As the temperature of breathing air increases, most often due to a poorly installed compressor or high ambient temperatures, the processing life of the cartridge must be corrected downwards. As an example, one major manufacturer suggests limiting filter life to 20 per cent of normal if the filter is exposed to an air stream temperature of 50°C rather than the ‘normal’ 20°C.

A final critical performance element is the pressure of the air passing through the filters. Filters perform optimally if air passes through them relatively slowly, allowing adequate ‘dwell time’ for contaminants to be trapped, absorbed into media such as desiccant or adsorbed onto media such as activated carbon. If the pressure within the filter system is low, any given atmospheric volume will pass more rapidly, reducing dwell time. In a worst-case scenario, the high initial
air velocity can also physically damage filters, releasing particles to contaminate the downstream system. Such problems can be avoided by installing a pressure-maintaining valve downstream of the filters. This will minimise the risk of contaminant breakthrough at the start of filling an empty cylinder or receiver.

Regrettably, it would appear that many compressors are purchased with a standard combination filter unit that does not include CO catalyst and which is not sized for operation at high ambient air temperature or in some cases even for the compressor’s free air delivery at the start of compression when pressures are low and flows highest. Any filter that is relatively undersized will carry a high risk of allowing volatile hydrocarbon ‘breakthrough’, especially in the latter part of its nominal life and if used in hot and humid conditions.

The limitations of current air quality testing

Most HP compressor air testing is undertaken by periodically sending samples for analysis by a laboratory, or sampling on site by passing air through proprietary colour-change detector tubes. The maximum intervals allowable between tests generally range from three to six months. Such intermittent testing is an inherently weak system, as test samples can be taken when the compressor is first started or after installation of new filter elements, providing no certainty that the air will be clean once the compressor is hot or the filters are nearing their end of service.

To date, screening for low levels of volatile hydrocarbons has required laboratory testing, using either gas chromatography with flame ionisation detection (GC/FID) or infrared spectroscopy (IR) techniques. There is no colour-change detector tube available that is sensitive or has a sufficiently broad spectrum to detect the large number of potential volatile hydrocarbons found in compressed breathing air. In both Canada and the USA air must be tested for volatile hydrocarbons by laboratories which use GC/FID or IR techniques.

Continuous, on-line hydrocarbon monitoring would be ideal and suitable analysers have been available for some time, but these are expensive and rarely used. Recently, portable volatile hydrocarbon IR analysers have become available to the diving industry, initially to address the problem of commercial divers being affected by contamination of the diving bell with volatile hydrocarbons from pipeline or oil-well sources. Some compressors are fitted with moisture detectors to alert users to desiccant exhaustion and this seems a useful feature that is available now. Continuous CO detection technology is readily available but surprisingly rarely used. Low cost CO monitors are now becoming widely available and inexpensive electronic-chip-based gas detectors of many types are expected to dramatically increase monitoring possibilities over the next few years.

What is the evidence this is a real problem?

Although deaths continue to occur as a result of compressor-intake contamination, there is relatively little concrete evidence of a widespread problem related to compressor production of either CO or volatile hydrocarbons. There is some, however, and it is of particular concern that we could be missing a wider problem. In one cave-diving incident in Florida, a near miss occurred due to toluene contamination, with the diver initially becoming disoriented and swimming in an agitated fashion before becoming lethargic, requiring rescue. Another interesting case involved a breathing-air compressor installation at a Canadian Fire Department station, which produced CO on several occasions. The contamination disappeared after a full overhaul and filter change only to recur shortly afterwards and appears most likely to have been due to a poor installation location which allowed recirculation of hot exhaust air and resulted in compressor overheating with consequent oil breakdown contaminating the breathing air.

A substantial deficiency in our knowledge arises from the fact that many fatalities do not have their air tested using techniques that would detect low levels of CO or volatile hydrocarbons. Levels that do not cause loss of consciousness may still be important if they have pre-disposed the diver to cardiac arrhythmia or underwater impairment of judgement leading to fatal error. In the 1995–2000 DAN fatality data, 145 fatalities were recorded as a result of drowning or near-drowning with the initial injury or problem “unknown”. It seems reasonable to speculate that gas contamination may have contributed to some of these deaths. A DAN review of 451 fatalities over a five-year period suggests that only 15 per cent of the divers had a carboxyhaemoglobin (COHb) measurement taken at the time of death. Three per cent of those sampled had a fatal concentration of COHb at the time of measurement.

In 2006, the UK Health and Safety Executive reported on an examination of diving equipment implicated in 54 accidents and incidents of all types. Whilst only five involved a suspected ‘bad fill’, 41 of the 54 air samples tested failed the moisture content standard. The Swedish Consumer Agency sampled air from nine dive suppliers in 1996, finding one case of oil contamination. In 2007, five of 20 failed, two due to excess CO2 and three due to moisture. In parts of the USA where laboratory analysis of air is required rather than simple detector tube sampling, rates of failure to meet acceptable CO levels have been as high as 3 per cent in recreational dive air (10 ppm limit), and the US Navy has encountered similar problems at a frequency of 2.5 per cent using an CO specification of 20 ppm.

Whilst this does not confirm there is a specific problem with volatile hydrocarbon contamination produced within compressors, it does suggest there is probably a systematic deficiency in the quality and performance of compressor installations.
Nitrox compression

With the rapid increase in the recreational use of nitrox, there are many instances of conventional HP air compressors being used with oxygen-enrichment systems feeding the intake in order to provide nitrox scuba cylinder fills. Although special nitrox compression systems are available commercially, make-shift arrangements are of concern with respect to the risk of fire as well as contamination of breathing air. The increased oxygen concentration passing through nitrox compressors degrades the compressor oil much more rapidly than would happen normally, which may generate toxic by-products, shorten the compressor and filter life, and increase the risk of contamination of breathing air. High-quality synthetic oils should, in theory, be less susceptible to thermal and oxidative degradation than mineral oils. Even so, evolving recommendations suggest oil changes may be needed after only 25 per cent of the time usually allowed.

Discussion

Whilst it remains unclear to what extent there may be a problem with contamination of breathing air generated by compressors, it is clear that air quality is an important issue that has been inadequately addressed. In order to better discern the extent of this potential problem and to reduce risk, we offer the following recommendations:

- All accident investigations should include laboratory testing of air by a specialist air-quality laboratory. The diver’s blood should also be sampled for COHb and the time interval recorded from cessation of potential CO exposure to blood sampling with a record of whether the diver was breathing air or oxygen.
- Existing requirements for divers’ breathing air to have very low levels of CO (generally < 10 ppm) remain appropriate and it may be that even lower limits would be wise to minimise risk for older and less fit individuals. Certainly all measures necessary should be taken to avoid the risk of substantially higher amounts entering breathing-gas supplies.
- Standards authorities should consider mandating maximum levels of volatile hydrocarbon contaminants and, where this does not presently exist, should also consider requirements for third-party testing or other means of quality control.
- Micro-particulates should also be considered further.

With respect to compressor installations, we suggest the following:

- Manufacturer’s installation, use, oil type and maintenance recommendations should be strictly adhered to and all maintenance should be logged.
- Compressors designed for periodic filling of small numbers of cylinders should not be used for continuous service installations.
- Even basic HP compressor systems should have high temperature alarms with automatic compressor shutdown plus real-time moisture and CO monitoring.
- Installations should be in well-ventilated locations with intakes that guarantee uncontaminated air.
- If there is choice of size, filter systems should be larger rather than smaller and should be changed at intervals corrected for high ambient operating temperatures using the filter manufacturer’s correction factors.
- Air purifier systems should incorporate a catalyst system to convert any entrained or internally produced CO to less dangerous CO₂.
- Air test samples should be taken with the compressor well warmed up so it is running at full temperature. Whilst testing after installing new filters will validate that the filter has been installed correctly and is not faulty, there is a case for also testing towards the end of nominal filter life so as to obtain measurements of air quality indicative of ‘worst-case’ scenarios.

For divers, we would suggest asking questions, looking for certificates of compliance with appropriate standards or codes of practice and investigating standards of air-quality control at destinations before travel. Particular caution should be applied for hot, humid locations, especially if compressors are installed near walls, in small rooms or if run in the heat of the day rather than at cooler times.

The most accessible and remarkably sensitive analytic method for hydrocarbons is to get into the habit of smelling cylinder air well before a dive. If the diver does not have a clear nose and intact sense of smell they should ask someone else to perform this service. Many contaminants have a significant “oily”, “rubbery” or “solvent” type smell. A “musty” smell may indicate excessive moisture is present. Being odourless, CO will not be detected by smell, but CO analysers have become significantly cheaper and could well be used alongside the oxygen analysers that have become routine for nitrox divers.

If CO or unusual odour is noted, abort the dive.

Finally, it would be useful if the industry, consumer agencies and researchers were to conduct regular surveys of air quality to provide a clearer picture of how often low-level contamination is occurring.

Sources and acknowledgements

The authors have attempted to summarise information which has been gained from a variety of overlapping sources and would be difficult to individually reference. These include manufacturers’ representatives, equipment suppliers, technical manuals, accident reports, various Standards and Codes of Practice, internet sources and personal experience in diving and hyperbaric medicine. We acknowledge gratefully the many who have helped inform and educate us, especially those analytical laboratory managers who have shared their insights, and, of course, the fire fighters and divers who have shared their experiences of breathing compressed air.
A 35-year-old male, with 20 years diving experience and no relevant medical history, undertook a solo crayfish dive. He told the boatman that he would be 15 minutes, but failed to surface. A search by police divers found him the following day at a depth of 9 metres’ sea water. The autopsy was limited due to decomposition of the body from sea lice. The police investigation suggested that he was diving over-weighted with 17.5 kg on the weight belt which was not released. All his equipment was intact and working correctly and the cylinder pressure was 194 bar so he died very early in the dive.

Analysis of the cylinder contents revealed an extremely high carbon monoxide level, 13,600 +/- 300 ppm (NZ standard < 10 ppm), as well as increased levels of carbon dioxide and methane. A second cylinder owned by the diver returned similar analysis. Both cylinders were filled at the same time of any other cylinders filled on that day reported as contaminated, so this was an isolated finding, the cause of which was unknown.

This case is from the New Zealand diving fatality data.
Case report

Diving-related pulmonary oedema as an unusual presentation of alcoholic cardiomyopathy
Helen Kenealy and Kenneth Whyte

Key words
Immersion, pulmonary oedema, scuba diving, cardiovascular, exogenous poison, case reports

Abstract

Diving-related pulmonary oedema (DRPO) is an uncommon and incompletely understood phenomenon. Pulmonary oedema has been rarely documented in shallow water. It is also associated with cold water and exertion and has been seen in swimmers as well as divers with no underlying heart disease. We describe an otherwise well 69-year-old lady who developed diving-related pulmonary oedema on her second and third dives in a shallow, heated pool. Follow-up echocardiogram revealed moderate global left ventricular dysfunction with an ejection fraction of 37%. There was a history of alcohol consumption of half a bottle of wine per day, which combined with the echocardiographic findings led to the diagnosis of alcoholic cardiomyopathy. We believe this not only to be the oldest patient with a documented case of DRPO but also the first report where it has clearly unmasked clinically significant underlying heart disease.

Introduction

Pulmonary oedema is a rare and probably under-reported, diving-related event with only infrequent reports in the literature since the first report in 1981. However, the pathophysiological mechanism remains elusive. To date this phenomenon has been reported only in patients with no evidence of underlying cardiac disease.

Case history

Mrs A was a previously fit and well 69-year-old lady. She originally denied any exertional dyspnoea in the period preceding her decision to start diving. In retrospect, she reported some minor exertional dyspnoea in the preceding months; however, she had not felt it significant. Her only past medical history was that of treated hypothyroidism. Her social history was significant in that she was still working and consumed half a bottle of wine per day. She had no previous history to suggest ischaemic heart disease.

She presented with a history that, at her second diving lesson one week prior, she had developed chest tightness and dyspnoea following ascent from four metres’ depth. This first episode was not severe and settled within 24 hours. No medical attention was sought.

On her third dive, she had successfully ascended without difficulty midway through the lesson in a chlorinated pool heated to 28°C. She then descended again for the duration of the lesson. At the end of the lesson she had to swim underwater the entire length of an Olympic-sized swimming pool (50 metres) to ascend near steps. On this second ascent, she noted marked shortness of breath and chest tightness. Onlookers described her as being blue.

On initial medical assessment, she was hypotensive and in acute pulmonary oedema. There was a left bundle branch block on electrocardiogram (ECG), which was presumed to be new as there were no previous ECGs to compare with. Symptoms improved with oxygen, intravenous frusemide and morphine. Initial chest X-ray showed unequivocal pulmonary oedema (Figure 1a).

Subsequently in-patient angiography showed only mild coronary artery disease. Echocardiogram revealed global systolic dysfunction with an ejection fraction of 45% and no regional wall motion abnormalities. Fasting glucose was normal, fasting cholesterol mildly elevated. Her renal function was normal. Repeat chest X-ray showed resolution of pulmonary oedema (Figure 1b). No troponin rise was noted; B-type naturetic peptide was not performed. Spirometry as an inpatient was normal with a forced expiratory volume in one second of 2.76 litres (112% of predicted) and forced vital capacity of 3.25 litres (106% of predicted).

In this case, we strongly recommended that she never dive again. She has been followed up by the cardiology service and has not dived again. Her repeat echocardiogram, however, revealed a mildly dilated left ventricle with abnormal septal motion consistent with persisting left bundle branch block. There was evidence of ongoing moderate global left ventricular dysfunction with a calculated ejection fraction of 37%. The left ventricular diastolic parameters...
Diving and Hyperbaric Medicine  Volume 38 No. 3 September 2008 153

were consistent with abnormal left ventricular relaxation with mildly increased end-diastolic pressure. There were no valvular or right-sided abnormalities noted.

She continues to have symptoms of shortness of breath on exertion consistent with NYHA class II. It is now considered that her diving-related pulmonary oedema (DRPO) was an unusual presentation of a mild, occult alcoholic cardiomyopathy.

Discussion

PATHOPHYSIOLOGY

Pulmonary oedema is thought to be a rare complication of diving.1 The mechanism is not completely understood. The first case series was published in 1981 by Wilmhurst et al.2 They described 11 divers in cold British waters who developed pulmonary oedema. The proposed mechanism was that of increased peripheral vascular resistance due to cold exposure thus raising both preload and afterload. They showed that healthy controls did not show the same degree of increased vascular resistance in the forearm when exposed to cold stimulus as those that developed pulmonary oedema. However, this finding was not replicated in a further study.1 Cases arising from warm-water diving have been reported, shedding doubt on the causative role of cold water in the development of DRPO.3

There have since been reports of pulmonary oedema while swimming (not diving) in cold waters.4 Studies in military personnel suggest there is an association with strenuous activity.1,3 Immersion in water leads to redistribution of blood and an increase in pulmonary capillary pressure, thus increasing the risk of pulmonary capillary leak. Cold immersion is thought to increase this redistribution of blood.4 Although cold may be a risk factor because it increases peripheral vascular constriction and thus cardiac preload and afterload, it is clear that cold-water diving is not a pre-requisite for DRPO.

It appears that multiple factors may contribute to the risk of DRPO and in any individual a complex interaction of factors may precipitate an episode of DRPO.

DEMOGRAPHICS AND DIVE CHARACTERISTICS

Cases reported in the literature number over 30 as described in a recent review.5 The cases have ranged in age from 23 to 61, mean ages have been quoted as 43.3 years and 45.6 years.1,6-8 The water temperatures have ranged from approximately 0°C to 25°C.6,9 The diving depths range from 3.3 to 42 metres’ sea water.1,9

DRPO is thought to be under reported given that spontaneous recovery has been observed to occur, as probably occurred in our patient during her second diving lesson the previous...
week. Symptoms often resolve with supportive treatment only once patients are removed from the water, such that diuretics are not always necessary.

**DRPO AND CARDIAC DISEASE**

Our patient is the first unequivocal report of DRPO unmasking underlying cardiac disease, in her case a probable alcoholic cardiomyopathy. There has been one case reported in a series by Hampson et al in which a 60-year-old lady with pre-existing hypertension developed DRPO and was found to have mitral valve prolapse with mitral regurgitation on echocardiogram. She had normal left ventricular systolic function and successfully completed seven subsequent dives in waters of approximately 25°C with a wetsuit. In this case we would suggest that the role, if any, of her mitral valve disease in the development of DRPO was uncertain.

During a dive the water pressure on the thoracic cage alters chest wall compliance, pooling of blood in the lungs alters the lung compliance and gas density increases airway resistance. These factors result in the need to generate a more negative pleural pressure to maintain tidal volume. This will, in turn, lead to a higher pressure gradient across the left ventricular (LV) wall, which results in increased LV wall stress and work – in contra-distinction to the effect of CPAP in acute pulmonary oedema where the positive pressure decreases the pressure gradient across the LV and hence reduces the LV wall stress and work. We postulate that the additional load imposed on the LV during these dives by the effect of immersion on both central vascular pressures and on the LV transmural pressure precipitated pulmonary oedema in our patient.

**LIKELIHOOD OF RECURRENCE AND RETURN TO DIVING**

These reports indicate that some but not all will have recurrence of symptoms when re-challenged. There is no way to predict who will or will not re-develop symptoms. Slade et al reported a case series of eight middle-aged divers with DRPO. Their subjects ranged from people doing their first dive to very experienced divers. Subsequently three of their patients successfully resumed diving.

Tetzlaff and Thorsen support the practice of advising sufferers to not dive again. For those who will not follow this advice, there is anecdotal evidence in 15 cases that a 5 mg dose of nifedipine taken orally before a dive may prevent recurrent attacks. During follow-up, it has been shown that most patients with DRPO will develop systemic hypertension. In our patient we believe the underlying cardiomyopathy would make further attempts at diving hazardous for the reasons outlined above.

**Disclaimer**

We declare no conflicts of interest. The authors are salaried employees and no additional funding was provided for this article.

**References**


Submitted: 24 June 2008
Accepted: 25 July 2008

*Helen Kenealy, MBChB, is Respiratory Registrar and Kenneth Whyte, MD, FRACP, FRCP(Glasg), is Respiratory Physician at the Green Lane Respiratory Medicine Service, Auckland Hospital, New Zealand.

**Address for correspondence:**

**Helen Kenealy**

Green Lane Respiratory Medicine Service
Level 7, Support Building, Auckland City Hospital
Park Road, Grafton, Auckland 1023
New Zealand

**Phone:** +64 (0)9-307-4949

**Fax:** +64-(0)9-631-0770

**E-mail:** <hdkenealy@gmail.com>
Longitudinal field study of lung function in scuba divers – a progress report

Anne Wilson

Key words
Scuba diving, diving research, pulmonary function

Background and objective

There have been relatively few studies examining the prevalence and characteristics of lung function changes occurring in recreational scuba divers. A pilot study conducted by the author identified a number of divers with low FEV1/FVC ratios.¹ The current field study, using spirometry for the assessment of lung function in recreational scuba divers, has two objectives:

1. To identify the physiological effects of recreational seawater scuba diving on lung function.
2. To monitor forced expired air volume in one second (FEV1) in a cohort of divers over a period of three years.

This project is ongoing, and this report summarises the work done in 2006.

Materials and Methods

Ethics approval was received from The University of Adelaide. Following written consent, data were collected from qualified scuba divers while at the SPUMS ASM in New Zealand in 2007. Participants completed a questionnaire requesting information on relevant health history and scuba diving experience. Data on the dive profiles of each diver were collected post-dive. EasyOne® spirometers, known for their stability and reliability,² were used to measure divers’ forced vital capacity (FVC), forced expired air volume in one second (FEV1), and forced expiratory flows at 25, 50 and 75% of the forced vital capacity (FEF25, FEF50, FEF75). Spirometry was conducted pre- and early post-dive on board the diving vessel.

Results

Data have been collected from 100 subjects. Results of 38 were rejected from analysis as they did not meet ATS/ERS international standard criteria for acceptability and reproducibility. Of the 62 remaining subjects, 43 were male and 19 female. Ages ranged from 19 to 68 years (mean 47 years).

Using body mass index (BMI) scales, 16 had a BMI < 25, 37 were overweight (BMI 25–30) and 9 obese (BMI ≥ 30). Sixteen had pre-dive FEV1/FVC ratio values less than 75%. Data were analysed using paired samples t-tests. There was a statistically significant decrease in FVC from pre-dive (4.78 +/- 1.04 L) to post-dive (4.67 +/- 1.00 L, t(61) = 2.41, P = 0.02). The eta squared statistic (0.09) indicated a moderate effect size.

Commentary

Previous research has shown that lung function can be affected by several factors, such as the physiological reduction in lung volume on immersion, breathing in saltwater, hyperoxia or hypoxia and decompression stress. These studies, however, have focused on professional divers and have been conducted in artificial environments rather than in the natural environment.³⁻⁵

Due to the popularity of recreational diving, and because of the need to meet less stringent medical standards than for professional diving, it is important that information on risks related to recreational diving be available to the diving public. It is hoped that the present study will increase our understanding of the physiological effects of scuba diving thus allowing for better-informed decisions and improved medical support of recreational diving.

References

Antimalarial drugs and diving – a brief review

David Cosh

Key words
Malaria, medications, travel medicine, tourism, infectious diseases

Travellers to Papua New Guinea are advised to take prophylaxis against malaria. All four human Plasmodium species are present in the country, P. falciparum being dominant, and the disease is endemic in many regions, especially the lowlands.¹

Diving in a location that requires divers to consider malaria prophylaxis raises the question: "Are there adverse drug effects of antimalarial prophylaxis that may be relevant to the recreational scuba diver?" This question can be sub-divided into two:

• Does taking prophylactic drugs compromise diver safety?
• Do these drugs interact in any clinically meaningful way with other drugs that divers might commonly take, either on a regular basis for the treatment of common medical conditions that do not themselves preclude diving, or with drugs that might be taken specifically to facilitate diving itself (e.g., antihistamines, decongestants and analgesics)?

SPUMS conferences attract a diverse group of attendees with respect to age, diving ability and health. Divers over 50 years of age are not uncommon and many would be taking medications for common medical conditions, e.g., hypertension, hyperlipidaemia, osteoarthritis and others. Antihistamines, decongestants, simple analgesics and non-steroidal anti-inflammatory drugs (NSAIDs) would not be uncommon additions to a diver’s kit and, accepting that no drug is free from adverse effects, there is little evidence to suggest that taking the aforementioned agents significantly compromises actual diver safety during a dive.

Anecdotal evidence derived from discussions with conference attendees suggests that most who do take malaria prophylaxis choose between doxycycline and mefloquine, with the majority choosing the former despite the need to take the drug for four weeks after leaving the malarious region. Simply considering either drug alone without consideration for any other drugs that the diver might take, it is well known that doxycycline can cause photosensitivity and gastrointestinal effects commonly seen with many antibiotics, e.g., nausea, loose bowels and specifically for this tetracycline, oesophagitis. Mefloquine is reported to commonly cause nausea, vomiting, abdominal pain, headache and dizziness. Less common are severe neuropsychiatric effects such as delirium and the drug can prolong the QT interval.² Kitchener et al carried out an open-label prospective study of over a thousand Australian soldiers serving in East Timor in 2001, who were taking either doxycycline or mefloquine. They found an incidence of severe sleep disturbance, headache and fatigue of less than 2% for both drugs (4% severe nausea with mefloquine, c.f. no reports on doxycycline) and concluded that mefloquine was generally well tolerated and should continue to be used in those intolerant of doxycycline.³

However, does this recommendation change when one considers other drugs that a diver might be taking? Assessing the likelihood and clinical significance of an interaction between two or more drugs is a more precise science now that the role of the cytochrome P450 enzyme family in drug metabolism is better understood. For many drugs the enzymatic pathways of their metabolism and in turn how they may affect the metabolism of other drugs is well understood.⁴ Nevertheless pharmacists and prescribers are bombarded, mostly via prescribing and dispensing software, with warnings of drug interactions that in reality have minimal if any clinical significance.

Doxycycline given together with warfarin usually results in an increase in the international normalised ratio (INR). Mefloquine lowers seizure threshold and can antagonise the effect of anticonvulsants and should be avoided with other drugs that can lower seizure threshold e.g., the tricyclic (TCA) and selective serotonin receptor uptake inhibitor (SSRI) classes of antidepressants. Mefloquine should not be given with quinine, quinidine or chloroquine because of risk of ECG abnormalities, but such combinations are unlikely to be used by a diver.

In summary, malaria prophylaxis with either doxycycline or mefloquine is likely to be well tolerated by most divers and should not preclude them taking their normal medications, as well as those that might be used on a short-term basis to make their diving a ‘medically trouble-free’ experience. One possible exception might be a diver who takes a tricyclic or SSRI antidepressant where one would suggest doxycycline as the preferred agent. However, many diving physicians would regard these drugs, and the underlying depressive illness, as contra-indications to diving. Diving and warfarin are an unlikely but possible combination, and if an anticoagulated diver were to take doxycycline it is likely that the dose of warfarin would need to be adjusted downwards to prevent a rise in INR and, therefore, an increased risk of bleeding.

References

Dengue fever and Japanese encephalitis – an update

Robyn Margaret Walker

Key words
Travel medicine, tourism, infectious diseases

Dengue fever

Most travellers to Papua New Guinea (PNG) are aware of the risk of malaria and the recommendation for malaria prophylaxis. However, malaria is not the only mosquito-borne disease of significance endemic to the region. Dengue fever is the most prevalent mosquito-borne viral disease in the world and is endemic to PNG. Four antigenically related but distinct flaviviruses are responsible for the four different serotypes of disease seen (dengue types 1–4). Serological surveys in PNG have documented evidence of past infection in up to 63% of urban adults and 5% of rural adults.1 In 1995, dengue type 3 was confirmed to be circulating in Lae and with dengue type 2 circulating in the Port Moresby area.1 Unlike other mosquito-borne diseases where the mosquitoes are active from dusk to dawn, the Aedes aegypti mosquito which transmits dengue fever bites during the day. Rapid population growth, expanding urbanisation, inadequate municipal water supplies and difficulties with refuse disposal result in an abundance of new breeding sites for mosquito vectors, while human migration patterns disperse vectors and viruses into new areas. Insecticide spraying in response to dengue outbreaks is not very effective against Aedes aegypti as they breed inside houses.

Although travellers are at greater risk of ‘mild dengue’ fever, most are at exceedingly low risk of contracting dengue haemorrhagic fever (DHF) due to the lack of previous exposure. Classic dengue fever is an acute febrile illness characterised by headache, retro-orbital pain, marked muscle and joint pains (break-bone fever) and a typically macular or maculopapular rash. The fever typically lasts five to seven days.

The greatest risk factor for development of DHF is a secondary infection with a different serotype from the original infecting virus. This form of the disease is characterised by increasing vascular permeability (plasma leakage syndrome), which is the most specific life-threatening feature. DHF has a case fatality rate of approximately 12% despite aggressive therapy. Warning signs for DHF include severe abdominal pain, persistent vomiting, an abrupt change from fever to hypothermia and abnormal mental status.

Treatment for dengue fever is symptomatic, including maintenance of adequate hydration and administration of antipyretics. Infection provides long-term protection against the particular serotype that caused the disease; however, it provides only short-lived immunity to the other three dengue serotypes.

Japanese encephalitis

Japanese encephalitis (JE) is also endemic in the region and is transmitted by the dusk-to-dawn biting Culex mosquito. The risk to travellers correlates with the duration of exposure in endemic areas. Symptomatic illness is uncommon (about one in 250 JEV infections leads to symptomatic illness), but there is a high fatality rate among those with clinical disease (mortality rates of approximately 30% are seen amongst hospitalised patients) and approximately 50% of survivors have severe neurological sequelae. Surviving children may have a better prognosis than adults. Treatment is supportive with a focus on management of intracranial pressure, optimisation of blood pressure, control of seizures and prevention of secondary complications.

Vaccination against JE is recommended for residents in JE endemic areas and travellers to endemic or epidemic areas during the transmission season, especially if exposure is prolonged and there is a greater likelihood of exposure to vectors, e.g., travel to rural areas. There is a current shortage of JE vaccine within Australia and measures have been put in place by the Department of Health and Ageing to limit supply to those at highest risk.2–4

References

1 Australian Defence Force Health Surveillance Report – PNG. Canberra: Ministry of Defence (internal report, not available);
Milne Bay Province of Papua New Guinea is well known for its tourism and recreational and pearl-diving activities. There are a number of companies providing diving services, probably the highest concentration of diving activities in Papua New Guinea.

Since the author moved to work at the Alotau Provincial General Hospital as the Consultant Anaesthetist in March 2006, there have been three diving accidents attended to at the Emergency Department.

Case report

A 38-year-old, male pearl diver presented at an outlying health centre with headache, cough, chest pain, shortness of breath and numbness from the abdomen downwards after a working dive. A diagnosis of a jellyfish sting was made, and he was given IV fluids and antibiotics. The next day he also had weakness of the legs and urinary retention and was transferred to Alotau Hospital. He continued to be managed conservatively by the internal medical team, and by the eleventh day was mobilising with a frame and improving, but still had abnormal bowel and bladder function. The diagnosis of a jellyfish sting was given on the discharge letter.

Comment

This was a typical case of Type 2 decompression sickness misdiagnosed as “neurotoxicity from a jellyfish sting” despite the contrary diagnosis being advocated by others. It highlights the problems of the PNG health services attending to diving accidents. Paramount in any service delivery is the human resource available, in this case the medical officers. The need for continuing education in underwater medicine is highlighted, and the South Pacific Underwater Medicine Society’s role is appreciated in this regard.

Dr Lucas Samof, Consultant Anaesthetist, Alotau General Hospital, Milne Bay, PNG
The world as it is

Basic life support in a diving bell and deck decompression chamber

Christopher John Acott

Key words
Basic life support, diving bell, decompression chamber

Abstract

Basic life support (BLS) in a diving bell (DB) or deck decompression chamber (DDC) is difficult due to the confined space and limited equipment. Retrieval of the unconscious diver into the DB requires the use of a pulley system. Once inside the bell, space limitations dictate that any resuscitative efforts are attempted with the victim (diver) either suspended in the upright position by the pulley system or lying against the DB’s curved side in a semi-supine position. BLS at best achieves a carotid artery systolic blood pressure of 40 mmHg or 30% of cardiac output in the supine position and, therefore, would be ineffective for a diver suspended on a pulley due to inadequate cerebral perfusion. Compression-only cardiopulmonary resuscitation (CPR) may be the only option with the victim lying against the bell’s side due to an inability to perform rescue breathing. However, compression ventilation CPR is possible in a DDC provided space limitations do not deny the rescuers access to the patient. The use of an automatic external defibrillator in a hyperbaric chamber has not been studied and therefore cannot be recommended in a DB or DDC. The laryngeal mask airway has been recommended by the Diving Medical Advisory Committee, but an accompanying study suggests that a new device, the i-gel™ without an inflatable cuff, may be a more suitable airway option for the future.

Introduction

Basic life support (BLS) in a diving bell (DB) or deck decompression chamber (DDC) is difficult due to the confined space, limited equipment and poor lighting. To date the only study addressing this issue was published by Myers and Bradley in 1981 and reflected the protocols of the time. In 2006, Acott addressed emergency airway management using currently available extraglottic airway devices; however, the latest BLS guidelines were not discussed.

Retrieval of an injured or unconscious diver to a DB is a complicated process that will ensure a considerable delay before any resuscitative efforts can be established. This process involves recognition that the diver is in trouble (known sequence of threatening events, oral communication is lost, a failure by the outside diver to answer signals via the umbilical or an indication from the diver’s bell gauge that the diver is not breathing) and the commencement of the recovery phase. Recovery is multifaceted and involves the bellman recovering the diver’s umbilical, uncoiling his own umbilical, switching to on-board gas supply, preparing the inside hoist, placing his helmet and fins on, exiting the bell after flooding it, locating the diver, attaching him to the hoist and then re-entering the bell and pulling the diver in. Once the injured diver is inside the bell the bellman must evacuate the bell, remove his and the injured diver’s helmet and some of the victim’s gear so that he is able to place the victim in a semi-supine position against the side of the bell or suspend him in the upright position by the pulley.
system. BLS inside a DB is, therefore, extremely hard if not impossible to do (Figure 1).

**Basic life support guidelines**

Since 2000, the management of a sudden cardiac arrest (SCA) has changed. These changes were based on extensive evaluation of the current resuscitation evidence by the International Liaison Committee on Resuscitation (ILCOR). Changes to the BLS guidelines have attempted to simplify and eliminate time-wasting procedures and were summarised in this journal recently.9

- Any attempt at resuscitation is better than none.
- External cardiac massage (ECM) should be started as soon as possible followed by early defibrillation via an automatic external defibrillator (AED).
- Interruptions to external cardiac compressions should be minimized (kept to < 10 seconds).
- The compression rate is 100 compressions per minute (although there is no human evidence identified for an optimal compression-ventilation ratio for CPR in patients of any age).
- The compression to ventilation ratio be 30:2 irrespective of the number of rescuers (this maintains international consistency, simplified teaching and increases the chance of skill retention).

Other changes that are relevant to BLS in a DB are that:

- the carotid pulse is no longer palpated because the ‘no signs of life’ equals being unconscious, unresponsive, not breathing normally and not moving
- the finger sweep to clear the airway only be performed if required
- compressions and ventilation should be continued up until the first defibrillation attempt
- chest compressions are important if defibrillation can’t be delivered within five minutes of collapse.

Current data indicate that prompt bystander cardiopulmonary resuscitation (CPR) is the main determinant of a successful outcome. The proportion of patients who survive a prehospital event varies greatly (quoted rates are between 20 and 50%) but the earlier bystander external cardiac compression (ECM) is commenced with the addition of early defibrillation the greater the chance of survival. Survival decreases by 7–10% for every minute without ECM. The importance of early bystander CPR was clearly shown by a retrospective study of 115 patients who suffered a prehospital cardiac arrest and were managed only by ambulance personnel. Those patients surviving to an emergency department showed a median response interval of seven minutes, whilst no patient survived to hospital where the response interval was greater than 14 minutes.10 Because of the above and other data, it is important that all divers are trained in BLS.

ECM must be performed with the victim supine on a flat, hard surface and achieve a depth of about one-third of the chest diameter (studies have shown this can also be achieved with the rescuer’s leg or foot). Several case studies have also shown it can be performed successfully with the patient prone on a hard surface. Efficient ECM can achieve 20 to 30 per cent of the cardiac output in the supine position with a carotid artery systolic pressure of 40 mmHg. These data suggest that CPR attempted with the victim upright hanging from a pulley (as recommended by the Comex company) would be ineffective in maintaining cerebral perfusion and therefore should not be attempted.

**The precordial thump**

Because there are inherent difficulties in performing BLS in a DB or DDC the use of a precordial thump may be thought to be effective. However, there have been no prospective studies to recommend this, and while there are three case studies showing that it has converted a ventricular tachycardia/ventricular fibrillation (VT/VF) arrest to a perfusing rhythm, other studies have shown it to convert VT to fast VT, VF, asystole or complete heart block. Both the Australian Resuscitation Council (ARC) and the European Resuscitation Council (ERC) recommend that the precordial thump should be used only in a witnessed, monitored VF or VT cardiac arrest within the first 15 seconds and only if a defibrillator is not readily available. The American Heart Association (AHA) has no recommendations for or against its use except that it is not included in BLS training. The precordial thump is, therefore, not recommended for use in a DB or DDC.

**Compression-only CPR**

Compression-only CPR is an option when the rescuers are unwilling or unable to do rescue breathing (mouth to mouth) and should be continuous at a rate of approximately 100 compressions per minute. Some animal studies and extrapolation from clinical evidence suggest that rescue breathing (RB) is not essential within the first five minutes of adult CPR for a VF sudden cardiac arrest (this type of arrest is generally regarded as non-asphyxial). This may be applicable to BLS in a DB because the diver would have been breathing a gas mixture containing approximately 50 kPa of oxygen. If the airway is open, passive chest recoil following compressions may provide some air exchange. Historically the older methods of CPR (Silvester, Shafer and Holger Nielson) provided some ventilation despite the lack of rescue breathing. Observational studies of adult cardiac arrest show that survival rates are better with compression only than with no CPR but were not as good as normal compression-ventilation CPR. Access to the victim’s airway is limited once lying against the wall of the DB and so compression-only CPR may be the only option if the victim has suffered a cardiac event. Compression-ventilation CPR may be effective if performed inside a DDC because the patient would be supine and on a hard surface; however, the position of the resuscitator(s) relative to the patient is dependent on the size and shape of the chamber and access to the victim’s airway may be limited.
Basic life support in a diving bell

There is no current recommendation from the Diving Medical Advisory Committee (DMAC) for an AED to be present at a diving platform or included in the diver’s medical kit for a DB or DDC, although the use of a laryngeal mask has been recommended. Space limitations, plus a lack of testing in a hyperbaric environment often prevent an AED from being carried in a DB or DDC. The insertion of an intravenous cannula and the administration of vasopressors or antiarrhythmics are also clearly not an option due to the inability to identify the victim’s cardiac rhythm. However, as stated by the ARC, ERC and AHA the foundation of good advanced cardiac life support is good BLS and not pharmacological intervention.

The recommended use of a cervical collar to stabilise the head also needs to be reassessed. If applied incorrectly cervical immobilisation can cause airway obstruction in unconscious victims and a rise in intracranial pressure. Application will also waste time before ECM can commence. The use of a cervical collar should only be considered when there is clear evidence for a neck injury, which is infrequent in diving accidents managed underwater.

A new extraglottic airway device (EAD), the i-gel™, was tested by Acott, and considered likely to be suitable for use in a DB or DDC. The i-gel was more effective in achieving an airway from different positions of the operator, and was preferred by diver medical technicians when compared to the classic laryngeal mask airway for emergency airway management. However, the current ILCOR 2005 guidelines provide information on different methods and devices and it is too early to recommend the incorporation of the i-gel into a proposed new BLS algorithm for a DB or DDC except as one of various alternatives. Keeping the airway patent is of the utmost importance in BLS, and not the device used.

Recommendations

The time must be noted when the bellman has recognised that the diver is in trouble. Timing is important because the chances of survival decrease 7–10% per minute. Once the diver is brought into the DB he should be checked for signs of life after his helmet has been removed and his airway cleared/suctioned if required. If there are no signs of life the bellman can try to insert an EAD. If the bellman is unable to insert the EAD, then a nasopharyngeal or Guedel airway can be used. Two rescue breaths (RBs) should be given via the EAD prior to laying the diver against the side of the DB and commencing ECM. If the bellman still has access to the EAD and is able to give rescue breathing, then the recommended ratio of two breaths and 30 compressions should be used. If not, then compression-only CPR should be used at a rate of 100 compressions per minute. If there are no signs of recovery, it is currently unclear after what time interval CPR should be abandoned (the ILCOR 2005 document suggests BLS should be continued till defibrillation or 20 minutes of asystole). However, this length of time is complicated by a number of factors underwater, for instance by how long it took for resuscitation to be commenced from the time it was noted that the diver was unresponsive and requiring rescue.

Two proposed algorithms, one for when signs of life are absent and one for when they are present, for the resuscitation of a diver in a bell are shown in Figures 2 and 3.

Reassessment of CPR skills

CPR skills have been shown to decline rapidly following initial achievement of competency. The ARC recommends that CPR skills are reassessed annually. The assessment should focus on the provision of adequate CPR and not on the technicalities. These recommendations have implications for DMT training.

---

Figure 2

Summary of resuscitation in a diving bell for a diver without signs of life

---

* DMAC recommends the use of a suction device in a DB or DDC.
** Do not attempt to resuscitate with the diver suspended from the pulley in upright position. Patient is placed against the DB wall (which will be semi-supine) or supine in a DDC.
*** This time is determined by the retrieval time to get the diver into the bell.
Summary

ECM should be commenced as soon as the diver is brought into the bell. If RB cannot be performed then compression-only CPR should be used. Timing of events is crucial in determining how long resuscitative efforts are continued.

Acknowledgements

Thanks to Alan Forsyth once again for permission to use the photo in Figure 1.

References


Submitted: 17 May 2008
Accepted: 05 August 2008

C J Acott, MBBS, Dip DHM, FANZCA, is a specialist in the Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, Adelaide, South Australia 5001, Australia.

Phone: +61-(0)8-8222-5116
Fax: +61-(0)8-8232-4207
E-mail: cacott@optusnet.com.au

This paper is based on a presentation by Dr Acott at the SPUMS ASM 2008 in Kimbe, PNG
New thoughts on the correction of presbyopia for divers
Quentin M Bennett

Key words
Eyes, vision, optometry, diving, equipment

Abstract

(Bennett QM. New thoughts on the correction of presbyopia for divers. Diving and Hyperbaric Medicine. 2008; 38: 163-4.)

Some new ideas on the correction of presbyopia for divers, particularly those with demands for critical near vision underwater, are discussed. Progressive lenses cannot be satisfactorily bonded to a dive mask, so efforts to mimic the properties of progressive lenses using bifocal lenses are described.

Introduction

From the age of early to mid-forties, the amplitude of accommodation declines and those in this age group and older begin to have difficulty with near tasks. This is presbyopia, yet another of the signs of maturity that strike at this stage of life.1

One relevant fact for divers is that poorer light conditions prevail underwater. This can result in increased or earlier presbyopic effects, probably due to slightly dilated pupils in the reduced light. This is more marked in dirty water or high-latitude, cold water diving. In the latter, the sunlight is at a much lower angle and less penetrates the water column. Should a diver have interests such as underwater photography, which demands critical near vision underwater, they may well notice presbyopic changes underwater before they notice them in normal life.

Choices in the correction of presbyopia for divers

Correction of presbyopia in normal life comes in many forms. Simple reading spectacles, progressive lenses, bifocal lenses or bifocal contact lenses are the most common. Diving masks certainly bring constraints and limitations. Contact lenses may have physiological compromises imposed.2 Another factor can be the diver’s standard of uncorrected vision, or their refractive error.

The easiest situation is when there is no great distance refractive error so that a near-vision correction is required only for reading a gauge or combo. Some early presbyopes have discovered that a carefully placed pinhole can be sufficient for them to read a gauge if they are diving in conditions with reasonable light levels. This aid is insufficient for any critical tasks.

Many divers have a distance refractive error in addition to their presbyopia. During their day-to-day life they wear progressive spectacles which enable them to focus at any distance from far to near. Progressive lenses are generally regarded as the most convenient everyday form of spectacle correction for presbyopes.3 Because these lenses utilise very complicated curves split between the back and front surfaces it is not possible to bond them to the flat posterior glass surface of a dive mask.

Therefore, in order to be able to see at both distance and near, one has to use bifocal lenses because they can be ground with a flat front surface with all the power on the back surface. Because the facemask utilizes a flat, toughened glass plate, a glass-fused bifocal lens is normally used. The flat front surface of the lens is bonded to the back surface of the mask glass plate in the appropriate position before the eyes. Such a glass-fused bifocal lens has a segment of glass of a higher refractive index fused to it and annealed under prolonged high temperature.

Generally a bifocal lens suffers from the disadvantage of offering sharp vision at distance and at near, but not at intermediate distances. On the other hand, the progressive lens has the great advantage of also offering sharp vision at intermediate distances, such as when viewing a computer monitor. The difference in power between the top distance portion and the lower, reading portion of a bifocal or progressive lens is termed the addition.

When underwater, critical intermediate vision is generally necessary only for specialised interests such as underwater photography, observation of macro life, and some scientific requirements. A technique for achieving this for divers is described below.
Method

Using a variation of a technique known in contact lens practice and refractive surgery as simultaneous vision or monovision, we have been trying to mimic aspects of the effects of a progressive lens for diving. For these techniques, as used above water, the dominant eye is usually corrected for distance and the non-dominant eye for near. Underwater we utilise only the technique for near and intermediate vision.

Monovision works well for some people, but is disliked by others. It does compromise binocular vision and could never be recommended for occupations requiring either good distance judgement or constant work at a single distance. Examples of occupations requiring good binocular vision and accurate distance judgement, making them unsuitable candidates for monovision, would include drivers, pilots and crane operators.

With the variance that we have utilised for diving, distance vision in both eyes is properly corrected and balanced. We use monovision or simultaneous vision at intermediate and near distance only, and this is done by providing different bifocal additions in each eye. We utilize this type of correction only for persons who have good binocular vision with its resulting ability to fuse slightly different images.

An easy illustration would be an emmetrope who requires no correction for distance. Depending on his or her exact requirements we may give them bifocal segments with reading powers of +3.00 diopter in the right eye and +1.50 in the left. With these lenses the right eye’s focus is theoretically at about 0.30 m and the left at 0.66 m. Assuming the diver has good binocular functions the then fused image from both eyes gives a depth of field not dissimilar to that of a progressive lens.

Requirements underwater are very different from those in our normal life on land where an average progressive spectacle addition for someone over 55 years of age is likely to be about +2.25, theoretically giving sharp focus from around 40 cm to infinity. For a presbyopic diver who uses an underwater digital camera and needs to inspect the screen, and who also uses a wrist-mounted computer, I will frequently suggest a +3.00 addition in the dominant eye, and a +1.25 or +1.50 in the non-dominant eye. This way they can comfortably see their camera screen or computer well with a reasonably low addition, then simultaneous vision at near may not be necessary. This is because a lower addition has a greater depth of field.

Comments

One thing that is very helpful to those outside the ophthalmic professions who may be giving advice on corrections is a basic understanding of the dioptric power system. The dioptric power of a lens is the reciprocal of the focal length in metres. For example, a lens with a focal length of 0.50 m has a power of 2 diopters. One with a focal length of 0.33 m is 3 diopters power and so on. If we could look at the earlier example of having a 3 diopter addition before the dominant eye, the focus of the bifocal addition would be at about 0.33 m with this eye, and the other eye with a +1.50 addition would have its focus at 0.66 m. So, from knowing the required focus distance, one can work out an approximation of the required lens power. Remember, however, that if the diver still has some remaining accommodation these figures won’t be exact and the focus will be closer, so a lower addition would be called for.

As with everything when dealing with human beings, there is great variance and no two people are the same, so one must remain flexible when prescribing lens power, and think carefully of the diver’s requirements.

References

Summary of the 2007 report prepared by Colin Wilson

Brian Cumming has been involved for a number of years in collating information on diving incidents and producing excellent reports for the British Sub-Aqua Club (BSAC). The summaries of the years 2005 and 2006 have previously been discussed in this journal and the full reports remain easily available.1−4 We are encouraged to browse the reports to learn from others’ mistakes. Information is collected from various sources: Her Majesty’s Coastguard (HMCG) 40%, BSAC members’ reports 25%, Royal National Lifeboat Institute (RNLI) 14%, newspapers 5% and any other sources available to him. Following an overview, details of incidents are then classified under the following headings: fatalities, decompression incidents, injury/illness, boating and surface incidents, ascent problems, technique problems, equipment problems and miscellaneous incidents. Where incidents have more than one cause or effect, then the most appropriate factor may be associated with each fatality and these are summarised as follows:1−4

- Two incidents involved three people diving together with the likelihood that separation was a contributory factor.
- Two incidents involved divers trapped in wrecks and running out of air. In one incident, there was a double fatality.
- Two incidents involved three people diving together with the likelihood that separation was a contributory factor.
- One diver was diving solo.

The details of the fatalities do not approach the comprehensive reports from New Zealand and Australia but they are still a valuable resource.5,6

From the fatalities section:

Case 1

“A rebreather diver entered the water ahead of his two buddies and dived to place a decompression cylinder on a shot line. He resurfaced and spoke to a fourth diver who was preparing to enter the water. Although they had planned to dive together, when the two buddies entered the water the first diver had already left the surface again. They continued their dive and, several minutes later, they saw the torchlight of the first diver. They found him lying on the bottom at a depth of 50 msw; he was unconscious, his mouthpiece was out of his mouth and he was tangled in some branches and other debris. They freed him and sent him to the surface. They started their ascent and met the fourth diver coming down. The fourth diver returned to the surface and found the casualty on his back with his mask off, he recovered him to the side, removed him from the water and started resuscitation procedures. Ten minutes later the casualty’s two buddies surfaced and contacted the emergency services. The casualty was taken to hospital where he was declared dead. It is thought that the casualty was diving with a rebreather that he had built himself.”

Case 2

“Two rebreather divers entered the water and commenced their dive down a shot line to a wreck. They conducted a bubble check at six msw and exchanged OK signals at 20 msw. They reached the top of the wreck at 32 msw. One of the pair indicated that all was not well. The dive leader signalled that they should ascend and they started to do so. Two metres into the ascent the troubled diver became agitated, closed and removed his rebreather mouthpiece and placed his bale-out regulator into his mouth. He then spat this regulator out and began to panic. His buddy placed the regulator back into his mouth and attempted to purge it; he discovered that it was not turned on. He turned the cylinder on and attempted to put the regulator into the diver’s mouth,
but the diver was not able to take the regulator and he fell unconscious. The pair had sunk to the bottom at a depth of 35 msw. The buddy dropped the casualty’s weight belt and inflated both their jackets. They made a buoyant ascent to the surface. Their total dive time was six minutes. At the surface the casualty was not breathing. The buddy called for help and tried to give rescue breaths; this was difficult to do because of their inflated BCDs and side-mounted gas cylinders. Their boat reached them quickly but they could not get the casualty back into the boat. His rebreather was removed and he was held by the boat. The Coastguard was alerted and five minutes later a helicopter arrived. After some difficulty the casualty was airlifted to hospital where he died three days later. The buddy was airlifted to a recompression chamber for precautionary treatment. The casualty had had a problem with his rebreather one month earlier and had suffered from hypercapnia.”

Decompression incidents

In 2007, there were 81 reports involving 89 cases of decompression illness (DCI), showing an incident rate similar to recent years. However, there has been a dramatic reduction, by over half, from the peak of 173 cases in 2002. There is continued concern voiced about the number of abnormal ascents with or without omitted decompression, as commented on in previous years’ reports. Thankfully ascent problems do not always result in DCI and are thus recorded in the ascent section; a number of these cases are avoidable.

The major causal factors associated with the 89 DCI cases in the 2007 report were:
• 24 involved diving deeper than 30 msw
• 24 involved rapid ascents
• 28 involved repetitive diving
• 13 involved missed decompression
• some cases involved more than one of these causes.
In addition, there were a number of reports from the RNLI of “diver illness”, without further clarification, which may have been further cases of DCI.

From the decompression illness section:

Case 3
“A diver conducted a dive using a rebreather to a maximum depth of 36 msw. During the dive she became separated from her buddy but continued solo as previously agreed. Her dive duration was 48 minutes including a two-minute stop at 14 msw and a three-minute stop at 3 msw. She sneezed several times during the dive. Later she felt a little unwell. This feeling continued and three days later she visited her doctor with symptoms of severe lethargy and loss of balance. She was referred to a recompression facility where cerebral DCI was diagnosed. She received a series of fourteen recompression treatments which fully resolved her symptoms. She was tested for a patent foramen ovale (PFO) and a large shunt was found.”

Case 4
“A diver conducted a dive to a depth of 30 msw. After 26 minutes she conducted a one-minute stop at 9 msw and a six-minute stop at 6 msw. After this dive, her left shoulder, armpit and chest felt very itchy. She took some antihistamine tablets. She ate lunch, felt thirsty but didn’t drink much. After a two-hour surface interval she dived to 23 msw for a drift dive. A delayed SMB was deployed after 25 minutes and she made a slow ascent to 9 msw for a one-minute stop and then to 6 msw for a three-minute stop. During this stop she experienced a discomfort in her left breast, which felt like suit squeeze. She left the water and de-kitted. Within minutes the discomfort became an intense pain, which lasted for about 25 minutes. The following morning the breast was swollen, uncomfortable and felt as if it were on fire. Diving medical advice was sought and she was told that it was not diving related. Then advice was sought from a recompression facility and she was asked to attend for examination. She received recompression treatment, which greatly eased her symptoms. (Coastguard report)”

The BSAC yearly incident report once again is a useful read and Brian Cumming is highly commended for collecting, analysing and producing it. Thanks should also go to those who make their information available.

References


Colin M Wilson, MB, ChB, FRCA, is Medical Director of the Dunstaffnage Hyperbaric Unit, Scottish Association for Marine Science, Dunbeg, Oban, Argyll PA37 1QA, Scotland.

E-mail: <colinwilson@tiscali.co.uk>

Key words
Recreational diving, accidents, diving deaths, abstracts
Articles reprinted from other sources

Joint Committee on Quantitative Assessment of Research Citation Statistics

A report from the International Mathematical Union (IMU) in cooperation with the International Council of Industrial and Applied Mathematics (ICIAM) and the Institute of Mathematical Statistics (IMS) < http://www.mathunion.org/Publications/Report/CitationStatistics >

Executive summary

This is a report about the use and misuse of citation data in the assessment of scientific research. The idea that research assessment must be done using “simple and objective” methods is increasingly prevalent today. The “simple and objective” methods are broadly interpreted as bibliometrics, that is, citation data and the statistics derived from them. There is a belief that citation statistics are inherently more accurate because they substitute simple numbers for complex judgments, and hence overcome the possible subjectivity of peer review. But this belief is unfounded. Relying on statistics is not more accurate when the statistics are improperly used. Indeed, statistics can mislead when they are misapplied or misunderstood. Much of modern bibliometrics seems to rely on experience and intuition about the interpretation and validity of citation statistics.

While numbers appear to be “objective”, their objectivity can be illusory. The meaning of a citation can be even more subjective than peer review. Because this subjectivity is less obvious for citations, those who use citation data are less likely to understand their limitations.

The sole reliance on citation data provides at best an incomplete and often shallow understanding of research, an understanding that is valid only when reinforced by other judgments. Numbers are not inherently superior to sound judgments.

Using citation data to assess research ultimately means using citation-based statistics to rank things: journals, papers, people, programs, and disciplines. The statistical tools used to rank these things are often misunderstood and misused.

For journals, the impact factor is most often used for ranking. This is a simple average derived from the distribution of citations for a collection of articles in the journal. The average captures only a small amount of information about that distribution, and it is a rather crude statistic. In addition, there are many confounding factors when judging journals by citations, and any comparison of journals requires caution when using impact factors. Using the impact factor alone to judge a journal is like using weight alone to judge a person’s health.

For papers, instead of relying on the actual count of citations to compare individual papers, people frequently substitute the impact factor of the journals in which the papers appear. They believe that higher impact factors must mean higher citation counts. But this is often not the case! This is a pervasive misuse of statistics that needs to be challenged whenever and wherever it occurs.

For individual scientists, complete citation records can be difficult to compare. As a consequence, there have been attempts to find simple statistics that capture the full complexity of a scientist’s citation record with a single number. The most notable of these is the h-index, which seems to be gaining in popularity. But even a casual inspection of the h-index and its variants shows that these are naive attempts to understand complicated citation records. While they capture a small amount of information about the distribution of a scientist’s citations, they lose crucial information that is essential for the assessment of research. The validity of statistics such as the impact factor and h-index is neither well understood nor well studied. The connection of these statistics with research quality is sometimes established on the basis of “experience.” The justification for relying on them is that they are “readily available.” The few studies of these statistics that were done focused narrowly on showing a correlation with some other measure of quality rather than on determining how one can best derive useful information from citation data.

Key words
Reprinted from, research, writing – medical, general interest

Editors’s comment: The Research Office of the Christchurch School of Medicine and Health Sciences of the University of Otago recently drew the Editor’s attention to this document. The Executive Summary is reproduced here as these comments have implications for all medical and scientific authors and research workers.
The poetry doctor

Thoughts that Bubble up

Born explosively from my inhalation
Its infancy is short lived.
As it assumes an adult jellyfish form,
Its translucent dome reflecting the rays of sunlight
Dappled by wave and cloud,
Its lens-like properties distorting its surrounds.
It Boyles towards the surface,
An expanding mass dividing amoebically.
Again and again creating a cluster of clones,
Each adding and accelerating the reproductive cycle.
The simplicity and singularity of the origin is
Soon overwhelmed by the complexity and multiplicity of
the masses
Ever expanding like a nuclear cloud.
Suddenly in a gurgling and rippling of the surface
It is lost, an extinction of this creation
Absorbed into the amorphous atmosphere
Yet still present, still existing.
I lay on my back inhaling and exhaling observing the
sequence repeat.
Is this a fast-forwarded, repeated replay of mankind?
Such philosophical dilemmas disturb my peace
So I roll over and continue my dive.

John Parker
<www.thepoetrydoctor.com>

Decompression dives

The primary function of language is to be a tool for communication. In everyday oral communication body language also plays a role, and misunderstandings due to less precise ways of expressing oneself can be clarified on the spot. In scientific writing precision is important.

Divers who descend in water are compressed. When they ascend they are decompressed. Accordingly, all dives ending at the surface are decompression dives. A diver who does not perform a decompression dive will thus remain at depth. Some dives require decompression stops in order to reduce the risk of decompression illness. My point is that, in general, one should pay more attention to semantics, and particularly so in scientific writing.

Otto I Molvær
Norway

Key words
Decompression, letters (to the Editor)

Editor’s comment: I share Dr Molvær’s irritation about this loose usage. Last week in Vanuatu, a group of divers I was with surfaced after a 35 msw dive without having incurred the need for a ‘stop’ on their computers, having done a ‘multi-level’ dive. On the DCIEM tables (which I carry), this dive required two stops of five minutes at 6 msw and ten minutes at 3 msw, which I duly did at the end of the dive. It is time all divers recognised that both ‘multi-level’ diving and ‘safety’ stops are forms of decompression stop diving.

Active sonar and marine mammals

There has been a recent world-wide concern regarding the impact of anthropogenic noise on the marine environment. A series of high profile events have served to heighten our awareness of these problems, particularly the potential impact of active naval sonar on marine mammals. Three specific incidents of beaked whale strandings in association with naval activities with active sonar have been given wide media coverage. These were in 1996 off Greece, 2000 off the Bahamas and 2002 off the Canary Islands. Hypotheses involving symptoms similar to human decompression illness were proposed and published.

Though the precise ways in which sounds can harm marine mammals is not fully understood, very loud noises could lead to auditory and/or non-auditory tissue injury. At lower levels, noise may mask communication, etc, leading to inappropriate behaviours and interference with individual animal interactions. Chronic acoustic stress may also result in increased vulnerability to disease and injury from cumulative effects.

A number of steps could help to lower the risks.
• Human behaviour could be modified; for instance, careful selection of location and timing of operations, and improved overall performance whilst minimizing the emission of acoustic energy.
• The effectiveness of ‘ramping up’ – starting sonar at relatively low levels and then gradually increasing to operational levels – is highly disputed.
• Improving risk assessment and mitigation is very difficult in that the impact mechanisms are so poorly understood.
• Improved marine mammal detection - visual watch, infrared detection and light applification technologies, passive and active sonar detection.

The circumstantial evidence provides enough impetus that the risk must be managed.

Book reviews

Clinical application of hyperbaric oxygen

I Boerema, WH Brummelkamp and NG Meijne, editors

Hard cover, 440 pages
Flagstaff, AZ: Best Publishing Company; 2008
Price: US$49.50 plus postage
Copies can be ordered from Best Publishing Company, 2355 North Steves Boulevard, POBox 30100, Flagstaff, AZ, USA 86003-0100
<www.bestpub.com>
Phone: +1-928-527-1055
Fax: +1-928-526-0370

The explosion-like interest in hyperpressure for medical use might lead to what may be called a fashion. When reading a prospectus of such an (HBO) institution, one feels clearly that an attempt was made to make a business out of it. I think it was exactly the abandonment of the strictly rational indications that was the reason why this promising therapy was already abolished at its very start in the century. The same danger is menacing now. Providing a patient with concentrated oxygen seems to come up to the high value which the public is apt to ascribe to natural medicine. I received a large number of letters from different countries in which doctors as well as laymen asked me to put patients into the chamber for reasons which were quite irrational, and sometimes even ridiculous. So I think it is one of our responsibilities, all of us being deeply interested in the real value of hyperbaric oxygen drenching, to prevent an unscientific development of hyperpressure therapy.

Does this ring a bell? The previous paragraph comprises the exact words of Prof Ite Boerema in his final address of the First International Congress on Hyperbaric Medicine, 45 years ago.

This book is a re-edition of the Proceedings of this First International Congress, held in Amsterdam in 1963 and edited by I Boerema, WH Brummelkamp and NG Meijne. It has been reprinted now thanks to the efforts and funding of the Foundation of the International Congress on Hyperbaric Medicine, who endeavour to progressively reprint all Proceedings since the very start of clinical hyperbaric oxygen therapy in 1963.

The First Congress was strictly limited to invited clinicians and scientists, who were apparently hand-picked by Professor Boerema and his colleagues. The Proceedings cover a wide range of topics, from medical indications to tank building problems, to physiological and pharmacological problems. As hyperbaric oxygen started as a surgical specialty, it is not surprising that most of the discussed indications are surgical in nature: anaerobic infections, coronary infarction, cancer therapy, congenital cardiac surgery under pressure to name a few. There are no diving-related papers.

Although the era of the “Randomised Controlled Trial” had not yet arrived, one cannot but be impressed by the careful design and thoughtful evaluation and reporting of the many different experiments and case-cohort studies that are reported. It is clear that these pioneers in hyperbaric medicine had a scientific mindset that surpassed many of us younger “scientists”. While browsing through the book (it is unfortunately difficult to read it in one go, due to its volume) I was struck by multiple “déjà-vu” experiences. In fact, it seems that our current-day difficulties in providing unequivocal scientific proof for the efficacy of HBO therapy, the danger of institutions promoting HBO therapy for doubtful and even “non-sense” indications on a purely commercial motivation, standardisation of hyperbaric treatments, hyperbaric safety and security and their financial implications, were all present and addressed already 45 years ago.

The book itself is well-presented, in hardcover and bound. The format matches the other Proceedings of ICHM Congresses, published by Best. The cover, however, in glossy colour print, does not follow the same canvas-covered look of the ICHM series. This in my view is a bit of a pity, especially as this reprint is meant to be the first step in completing the series of ICHM Proceedings backwards.

As for the editing, I fell upon a few disturbing typographical errors in the Table of Contents (“coronary infection” instead of “infarction”; “hyperbalic chamber”), but fortunately the rest of the book seems to have been carefully controlled and corrected.

In order not to unnecessarily repeat scientific efforts and errors again and again, it is necessary to know and consider scientific data and reports even from the first years of hyperbaric medicine. Even if some of the reported uses of hyperbaric oxygen have now been abandoned or seem superfluous, many things can be learned from reading the observations and thoughts of our predecessors. This book, and any subsequent reprints of early International Congress Proceedings that may follow, have an incontestable historical and educational value and are well worth their place on any hyperbaric scientist’s bookshelf.

Peter Germonpré
Centre for Hyperbaric Oxygen Therapy, Brussels

Key words
Book reviews, history, meetings, hyperbaric oxygen
To the very depths: a memoir of Professor Peter B Bennett

Peter B Bennett

Hard cover, 248 pages
ISBN 978-1-930536-47-0
Flagstaff, AZ: Best Publishing Company; 2008
Price: US$21.95 plus postage
Copies can be ordered from Best Publishing Company, 2355 North Steves Boulevard, PO Box 30100, Flagstaff, AZ, USA 86003-0100
<www.bestpub.com>
Phone: +1-928-527-1055
Fax: +1-928-526-0370

I first met Peter Bennett in late 1964, when, as a second-year medical student, I and other undergraduate divers from Cambridge University spent time at the Royal Naval Physiological Laboratory, Alverstoke. I had not known that Peter, like me, was a ‘Pompey-ite’ (born and bred in Portsmouth, just across the harbour from Alverstoke). It was extraordinary to realise that we must have attended the same opera performances at the King’s Theatre as boys; life is certainly full of coincidence! We have met intermittently since at meetings and when I spent a short period at Duke in the late 1980s.

I have always taken a particular interest in Peter’s work because of my early involvement with field studies on diver performance and nitrogen narcosis under Professor Alan Baddeley’s tutelage, and still have my copy of Peter’s 1966 seminal work, The aetiology of compressed air intoxication and inert gas narcosis. This book and the 1969 first edition of Bennett and Elliott, Physiology and medicine of diving, were the catalysts for my enthusiasm for diving physiology and medicine over the past four decades, and I am sure that I am not the only physician in the world for whom this is the case. There is no question that he has had a very major influence on the field, and the bibliography of over 300 publications at the end of this book attest to this, as do the many international awards and recognitions that he has received. For anyone working in his areas of research, the bibliography will save much computer search time.

To the very depths chronicles Peter’s life from a dockyard worker’s son to the pinnacle of his career at Duke, and catalogues the development of his major research interests and the thinking that went behind it. The development of the Divers Alert Network (DAN) from its early days in a back room at the FG Hall Hyperbaric Laboratory at Duke University Medical Center to the present-day, large organization is covered extensively. Much of the end of the book is devoted to telling his side of the story regarding the internal strife within DAN during the period 2000 to 2003, and his enforced early ‘retirement’ from DAN. Anyone involved in diving during this period will be aware at least of the countless rumours regarding the battle for control of DAN between Bennett and members of the DAN Board. This book does little to help one understand what was really going on behind the scenes, though it provides a vigorous personal defence, coloured by a sense of resentment and anger. Earlier parts of the book that describe his initial years and later internal political problems at Duke smack somewhat of the same.

As a reader, what I want from an autobiography is to come away understanding the person better, their motives, beliefs, loves, hatreds and philosophy of life. I do not gain much of this here; Peter Bennett keeps his cards fairly tight to his chest and this is a disappointment. The author, like Eric Kindwall in his memoir a few years back, could have done with a good editor. Because a scientist or physician has much experience of writing scientific papers and monographs does not mean he writes well in a literary sense. There is a degree of repetition, and the cataloguing of awards becomes frankly boring.

An interesting read, in which Bennett the scientist is to the fore, Bennett the man seems somewhat hidden.

Michael Davis, Editor
Hyperbaric Medicine Unit, Christchurch

Key words
Book reviews, autobiography, DAN – Divers Alert Network, general interest
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 current financial member of the Society of at least two years' standing.
2. The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved facility. The list of approved facilities providing two-week courses is provided on the SPUMS website.
3. The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
4. The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, 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. Accompanying this written report should be a request to be considered for the SPUMS Diploma and supporting documentation for 1–4 above.

Additional information – prospective approval of projects is required

The candidate must contact the Education Officer in writing (e-mail is acceptable) to advise of their intended candidacy, 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, and that the candidate is the first author, where there are more than one.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of: Associate Professor David Smart, Education Officer, Associate Professor Mike Davis, Dr Simon Mitchell.

All enquiries should be addressed to the Education Officer:
Associate Professor David Smart
GPO Box 463
Hobart, Tasmania 7001
E-mail: <david.smart@dhhs.tas.gov.au>

Key words
Qualifications, underwater medicine, hyperbaric oxygen, research

Dr Carl Edmonds

The South Pacific Underwater Medicine Society and the European Underwater and Baromedical Society wish to congratulate Dr Edmonds on his award in June 2008 of the Medal of the Order of Australia (OAM). The citation reads “for service to subaquatic and hyperbaric medicine as a practitioner, researcher and educator, and to the advancement of diving safety.”


As always, Carl provides a twist to the tail!

HTNA 2008 Meeting SPUMS Prize

At the 2008 Hyperbaric Technicians and Nurses Association annual meeting, the SPUMS Prize for the best presentation by an HTNA member went to S/N Loo Mui from Singapore for her poster “Relationship between time to hyperbaric treatment and clinical outcomes of divers treated at the Naval Hyperbaric Centre, Singapore.”
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. ANZHM 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. 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.

4. 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.

5. 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.

6. Completion of a workbook documenting the details of clinical exposure attained during the training period.

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
38th Annual Scientific Meeting
24–30 May 2009

THEMES:

Diving, Flying and Space Exploration
Future synergies in Diving Accident Management
Ear Injuries and ENT Workshop

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

Keynote Speaker

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

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

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

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

SPUMS 2009 Convenor:
Associate Professor David Smart
Education Officer SPUMS
GPO Box 463 HOBART 7001
Tasmania, AUSTRALIA
E-mail: <dsmart@iinet.net.au>
Phone: +61-(0)3-6222-8193    Fax: +61-(0)3-6222-7268
Mobile: +61-(0)419-508577
FIRST ANNOUNCEMENT

Venue:
King’s College Conference Centre, University of Aberdeen

Hosts:
University of Aberdeen and Aberdeen Royal Infirmary Hyperbaric Medicine Unit.

Key topics will include:
- Health technology assessment and hyperbaric oxygen therapy
- Diving research and treatment of decompression illness
- Treatment of ORN and diabetic foot

Contact details:
EUBS 2009
c/o Environmental & Occupational Medicine
Liberty Safe Work Research Centre,
Foresterhill Road, Aberdeen, AB25 2ZP
Phone: +44-(0)1224-558188
Fax: +44-(0)1224-551826
E-mail: <hyperbaric@abdn.ac.uk>
<www.hyperchamber.com/EUBS2009>
EXECUTIVE COMMITTEE (as of January 2008)

**PRESIDENT**
Professor Alf O Brubakk  
NTNU, Dept Circulation & Imaging  
N-7089 Trondheim, Norway  
**Phone:** +47-(0)73-598904  
**Fax:** +47-73-(0)897940  
**E-mail:** <alf.o.brubakk@ntnu.no>

**VICE PRESIDENT**
Dr Peter Germonpré  
Centre for Hyperbaric Oxygen Therapy  
Military Hospital Brussels  
B-1120 Brussels, Belgium  
**Phone:** +32-(0)2-2644868  
**Fax:** +32-(0)2-2644861  
**E-mail:** <peter.germonpre@mil.be>

**IMMEDIATE PAST PRESIDENT**
Dr Noemi Bitterman  
Technion, Israel Institute of Technology  
Technion City  
Haifa 32000, Israel  
**Phone:** +972-(0)4-8294909  
**Fax:** +972-(0)4-8246631  
**E-mail:** <noemib@tx.technion.ac.il>

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

**HONORARY SECRETARY**
Dr Joerg Schmutz  
Foundation for Hyperbaric Medicine  
Kleinunhenterstrasse 177  
CH-4057 Basel, Switzerland  
**Phone:** +41-(0)61-631306  
**Fax:** +41-(0)6131006  
**E-mail:** <joerg.schmutz@hin.ch>

**MEMBER AT LARGE 2008**
Dr Peter Knessl  
Steinechtweg 18  
CH-4452 Itingen/BL  
Switzerland  
**Phone:** +41-(0)44-716-7105  
**E-mail:** <pknessl@bluewin.ch>

**MEMBER AT LARGE 2007**
Dr Phil Bryson  
DDRC, The Hyperbaric Medical Centre  
Tamar Science Park, Research Way  
Derriford, Plymouth  
Devon, PL6 8BU, United Kingdom  
**Phone:** +44-(0)1752-209999  
**Fax:** +44-(0)1752-209115  
**E-mail:** <phil.bryson@ddrc.org>

**MEMBER AT LARGE 2006**
Professor Maide Cimsit  
Department Underwater and Hyperbaric Medicine  
Istanbul Faculty of Medicine  
80620 Istanbul, Turkey  
**Phone:** +212-(0)5313544  
**E-mail:** <cimsit@istanbul.edu.tr>

**HONORARY TREASURER & MEMBERSHIP SECRETARY**
Ms Patricia Wooding  
16 Burselm Avenue  
Hainault, Ilford  
Essex, IG6 3EH, United Kingdom  
**Phone & Fax:** +44-(0)20-85001778  
**E-mail:** <patriciawooding@btinternet.com>

**NEWSLETTER EDITOR**
Dr Peter HJ Mueller  
PO Box 1225  
D-76753 Bellheim, Germany  
**Phone:** +49-(0)7272-74161  
**Fax:** +49-(0)7272-774511  
**E-mail:** <pete@ejuhm.de>
## EUBS Financial Statement 2007

### Sterling Account

#### Opening Bank Balance at 1st January 2007
£ 1,316.78

#### Petty Cash at 1st January 2007
£ 25.73

### Income

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membership Fees</td>
<td>£ 8,907.36</td>
</tr>
<tr>
<td>Paypay</td>
<td>£ 0.21</td>
</tr>
<tr>
<td>Journal Advertising</td>
<td>£ 187.00</td>
</tr>
</tbody>
</table>

### Expenditure

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Secretarial Fees</td>
<td>£ 600.00</td>
</tr>
<tr>
<td>Cardnet/Bank Charges</td>
<td>£ 675.57</td>
</tr>
<tr>
<td>Petty Cash for Postage/Stationery/Copying</td>
<td>£ 207.74</td>
</tr>
<tr>
<td>Telephone/Fax/Sundries</td>
<td>£ 82.46</td>
</tr>
<tr>
<td>Computer repair</td>
<td>£ 170.00</td>
</tr>
<tr>
<td>Travel/Hotel Expenses - Treasurer - Meeting 05</td>
<td>£ 856.50</td>
</tr>
<tr>
<td>Journal Expenses - September 06 Issue</td>
<td>£ 161.39</td>
</tr>
<tr>
<td>Transfer to Euro account</td>
<td>£ 1,000.00</td>
</tr>
</tbody>
</table>

#### Closing Bank Balance at 31st December 2007
£ 6,647.89

#### Petty Cash at 31st December 2007
£ 35.53

### Euro Account

#### Opening Bank Balance at 1st January 2007
€ 2,798.61

### Income

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Membership Fees</td>
<td>€ 1,734.92</td>
</tr>
<tr>
<td>P Kness</td>
<td>€ 794.01</td>
</tr>
<tr>
<td>Transfer from Sterling account</td>
<td>€ 1,409.90</td>
</tr>
</tbody>
</table>

### Expenditure

<table>
<thead>
<tr>
<th>Description</th>
<th>Amount</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bank Charges</td>
<td>€ 169.45</td>
</tr>
<tr>
<td>Journal expenses - Dec 06 Issue</td>
<td>€ 1,539.34</td>
</tr>
<tr>
<td>Journal expenses - Jun 07 Issue</td>
<td>€ 1,490.19</td>
</tr>
<tr>
<td>Web site fees</td>
<td>€ 372.32</td>
</tr>
<tr>
<td>Travel Grants</td>
<td>€ 2,050.00</td>
</tr>
</tbody>
</table>

### Closing Bank Balance at 31st December 2007
€ 1,116.14

### Exchange Rate at 31st December 2007
£1=€1.3864

### Effective Sterling Balance in Euro Account
£ 805.06

### Total Balance of both accounts as of 31st December 2007
£ 7,488.48

Ms P Wooding, EUBS Treasurer/Membership Secretary
Situations vacant

The Alfred Hospital, Australia
Registrar / Fellow in Diving and Hyperbaric Medicine

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

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

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

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>

The Hyperbaric Research Prize

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

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

Contact us for all your travel requirements within Australia and overseas.
Ask us about our low-cost air fares to all destinations.
The Australia and New Zealand Hyperbaric Medicine Group

Introductory Course in Diving and Hyperbaric Medicine

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

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

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

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

---

THE HISTORICAL DIVING SOCIETY  
CONFERENCE 2008

**Date:** Saturday 25 October 2008  
**Venue:** Merseyside Maritime Museum, Albert Quay, Liverpool

Speakers and subjects are:
- Christopher Swann: The Development of Commercial Helium Diving
- Dr Pavel Borovikov: The History of Diving in Russia
- Sven Erik Jørgensen: Danish Diving: From Bells to Hansen
- Don Hale: Crabb - was he the real James Bond?

Tickets cost £25, obtainable from The Historical Diving Society, 25 Gatton Road, Reigate, Surrey, RH2 0HB  
**Phone:** +44-(0)1737-249961  
**E-mail:** enquiries@thehds.com

---

The future of diving: 100 years of Haldane and beyond

International Symposium

**Dates:** 18 to 19 December 2008  
**Venue:** Trondheim, Norway

In 1908 Haldane and co-workers published their paper “The prevention of compressed-air illness”, that has formed the basis of modern decompression procedures. It is our belief that diving will be of increasing importance in the future. Climatic changes, lack of food and an increasing need for energy will force better use of our underwater resources. This movement will force a development of new and improved technologies for surviving, working and playing underwater, allowing us to dive deeper and stay longer. Still, however, physiology will be a limiting factor, increasing the need for better understanding of the effect of the underwater environment on man.

**For further information contact:** Alf O Brubakk  
**E-mail:** alf@ntnu.no

---

Diving medicine

The Royal Society of Medicine in association with the Diving Diseases Research Centre

**Dates:** 27-31 October 2008  
**Venue:** International Hall, Lansdowne Terrace, London

**Who should attend:** Doctors interested in recreational diving pre hospital care medicine

**Faculty:** Drs Elliott Singer, Churchill Medical Centre London; Denny Levett, UCL; Pieter Bothma, James Paget University Hospital, Gorleston-on-Sea; Sean Hopson, Devon; Steven Harrison, Whipps Cross University Hospital NHS Trust, London and Oliver Sykes (Course Leader), London

**Topics include:** Role and responsibilities of the diving doctor; Diving pathophysiology; Hyperbaric chamber familiarisation and orientation; Fitness to dive; Prevention of diving accidents; Management of chamber medical emergencies; Diving and disabilities.

**Fees:** RSM Fellow: £870, Non-Fellow: £950, Student Member: £500, Student Non-Member: £600

**For more information or a booking form contact:**  
**Phone:** +44-(0)207-290-3946 (Nicole Leida)  
**E-mail:** nicole.leida@rsm.ac.uk  
**Book online at:** <www.rsm.ac.uk/diary>
2008 ROYAL AUSTRALIAN NAVY MEDICAL OFFICERS’ UNDERWATER MEDICINE COURSE

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

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>

UNDERSEA & HYPERBARIC MEDICAL SOCIETY
Annual Scientific Meeting 2009
Preliminary notice

Dates: 25 to 27 June 2009
Venue: Crowne Plaza Resort
Los Cabos-Grand Faro
Blvd San Jose s/n, Zona Hoteler
San Jose del Cabo, 23400 Mexico

For further information:
E-mail: <uhms@uhms.org>
Website: <www.uhms.org>

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 course 2008
27 – 31 October

There are no further Medical Officer courses in 2008

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>

Asian Hyperbaric & Diving Medical Association
DMAC-EDTC Level I & IIa

Dates: 9 to 13 December 2008
Venue: Langkawi Island, Malaysia
Course fees: Level I US$700; Level IIa US$900

A course for experienced diving medical examiners and diving medicine physicians. This two-part course covers advanced aspects of diving medicine not usually detailed in recreational and military courses. The major emphasis will be on mixed-gas, deep and saturation diving operations.

Course Directors:
Professor David Elliott
Dr Jurg Wendling

Course Coordinator:
Dr Tony Lee

Sponsor: Hyperbaric health
For background information visit:
<http://www.dmac-diving.org/courses/>
Contact: <hyperbarichealth@gmail.com>
Fax: +61-(0)-3-9558-0216 or +605-242-8533

British Hyperbaric Association

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
Professor Richard Moon, Duke University

For further information and on-line registration go to the BHA2008 website:

For more information on the BHA go to:
<www.hyperbaric.org.uk>

DIVING HISTORICAL SOCIETY
AUSTRALIA, SE ASIA
P. O Box 347, Dingley Village, Victoria, 3172, Australia
Email: <deswill@dingley.net>
Website: <www.classdiver.org>
Instructions to authors
(revised August 2008)

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

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

Requirements for manuscripts

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

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

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

Illustrations, figures and tables should not be embedded in the wordprocessor document, only their position indicated. No captions or symbol definitions should appear in the body of the table or image. Table columns are preferred as tab-separated text rather than using the columns/tables options or other software and each submitted double-spaced as a separate file.

Illustrations and figures should be submitted as separate electronic files in TIFF, high resolution JPEG 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. 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:


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

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

Consent

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

Copyright

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

Full ‘Instructions to authors’ can be found on the EUBS and SPUMS websites and in Vol 38, June 2008 issue.
DIVING EMERGENCY SERVICES PHONE NUMBERS

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

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

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

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

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)

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

The DES numbers are generously supported by DAN

DAN Asia-Pacific DIVE ACCIDENT REPORTING PROJECT
This project is an ongoing investigation seeking to document all types and severities of diving-related accidents. Information, all of which is treated as being confidential in regard to identifying details, is utilised in reports on fatal and non-fatal cases.
Such reports can be used by interested people or organisations to increase diving safety through better awareness of critical factors.
Information may be sent (in confidence unless otherwise agreed) to:
DAN Research
Divers Alert Network Asia-Pacific
PO Box 384, Ashburton VIC 3147, Australia
Enquiries to: <research@danasiapacific.org>

DIVING INCIDENT MONITORING STUDY (DIMS)
DIMS is an ongoing study of diving incidents. An incident is any error or occurrence which could, or did, reduce the safety margin for a diver on a particular dive. Please report anonymously any incident occurring in your dive party. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver injury. Using this information to alter diver behaviour will make diving safer.
Diving Incident Report Forms (Recreational or Cave and Technical)
can be downloaded from the DAN-AP website: <www.danasiapacific.org>
They should be returned to:
DIMS, 30 Park Ave, Rosslyn Park, South Australia 5072, Australia.

DISCLAIMER
All opinions expressed in this publication are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policies of SPUMS or EUBS.
## CONTENTS

Diving and Hyperbaric Medicine Volume 38 No. 3 September 2008

<table>
<thead>
<tr>
<th>Page</th>
<th>Section</th>
</tr>
</thead>
<tbody>
<tr>
<td>121</td>
<td>The Editor's offering</td>
</tr>
<tr>
<td>122</td>
<td>The Presidents' pages</td>
</tr>
<tr>
<td>124</td>
<td>Extraglottic airway devices for use in diving medicine – part 3: the i-gel™</td>
</tr>
<tr>
<td>128</td>
<td>An underwater blood pressure measuring device</td>
</tr>
<tr>
<td>134</td>
<td>Personality as a predisposing factor for DCI: a pilot study</td>
</tr>
<tr>
<td>139</td>
<td>Hyperbaric oxygen therapy in the treatment of skin ulcers due to calcific uraemic arteriolopathy: experience from an Australian hyperbaric unit</td>
</tr>
<tr>
<td>145</td>
<td>Compressed breathing air – the potential for evil from within</td>
</tr>
<tr>
<td>152</td>
<td>Diving-related pulmonary oedema as an unusual presentation of alcoholic cardiomyopathy</td>
</tr>
<tr>
<td>155</td>
<td>Longitudinal field study of lung function in scuba divers – a progress report</td>
</tr>
<tr>
<td>156</td>
<td>Antimalarial drugs and diving – a brief review</td>
</tr>
<tr>
<td>157</td>
<td>Dengue fever and Japanese encephalitis – an update</td>
</tr>
<tr>
<td>158</td>
<td>Knowledge of underwater medicine in Milne Bay, Papua New Guinea [Abstract]</td>
</tr>
<tr>
<td>159</td>
<td>Basic life support in a diving bell and deck decompression chamber</td>
</tr>
<tr>
<td>163</td>
<td>New thoughts on the correction of presbyopia for divers</td>
</tr>
<tr>
<td>165</td>
<td>British Sub-Aqua Club Diving Incidents report 2007</td>
</tr>
<tr>
<td>167</td>
<td>Joint Committee on Quantitative Assessment of Research Citation Statistics</td>
</tr>
<tr>
<td>169</td>
<td>Clinical application of hyperbaric oxygen</td>
</tr>
<tr>
<td>168</td>
<td>Decompression dives</td>
</tr>
<tr>
<td>169</td>
<td>To the very depths: a memoir of Professor Peter B Bennett</td>
</tr>
<tr>
<td>171</td>
<td>Diploma of Diving and Hyperbaric Medicine requirements</td>
</tr>
<tr>
<td>172</td>
<td>ANZCA Certificate in Diving and Hyperbaric Medicine</td>
</tr>
<tr>
<td>173</td>
<td>SPUMS ASM 2009</td>
</tr>
<tr>
<td>174</td>
<td>EUBS 35th ASM 2009</td>
</tr>
<tr>
<td>175</td>
<td>EUBS Executive Committee</td>
</tr>
<tr>
<td>176</td>
<td>EUBS Financial Statement 2007</td>
</tr>
<tr>
<td>177</td>
<td>Situations vacant</td>
</tr>
<tr>
<td>178</td>
<td>Courses and meetings</td>
</tr>
<tr>
<td>180</td>
<td>Instructions to authors</td>
</tr>
</tbody>
</table>

---

Diving and Hyperbaric Medicine is indexed on SCIE and EMBASE

Printed by Snap Printing, 166 Burwood Road, Hawthorn, Victoria 3122