

South Pacific Underwater Medicine

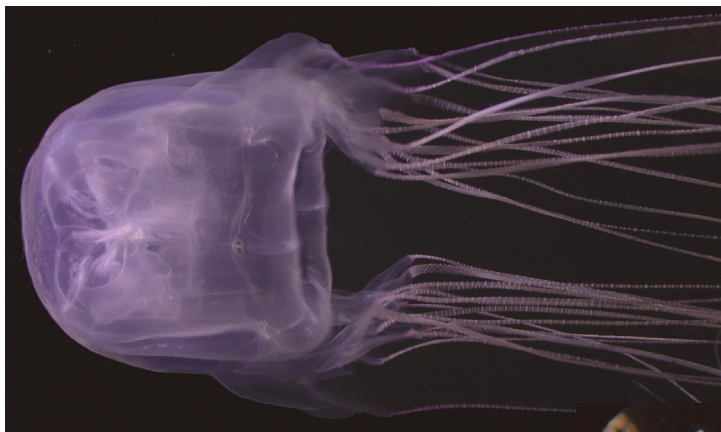
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SPUMS



Chironex fleckeri

Carukia barnesi



Venomous Australian jellyfish

Decompression modelling 102

Predicting treatment needs from nitrogen loading

Flying after recompression therapy: toss of a coin?

A free-ascent decompression accident

PURPOSES OF THE SOCIETY

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

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MEMBERSHIP

Membership is open to all medical practitioners.
Associate membership is open to all those who are not medical practitioners but are interested in the aims of the Society, and/or those engaged in research in underwater medicine and related subjects.
Membership application forms can be downloaded from the Society's web site at <<http://www.SPUMS.org.au>>

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The Society's financial year is January to December, the same as the Journal year.

The 2006 subscription will be Full Members A\$132.00 and Associate Members A\$66.00, including GST.
There will be an additional surcharge of \$8.00 for journal postage for all members living outside Australia.

The Editor's offering

Decompression sickness is described as a random event with a variable probability depending on the depth/time/breathing-gas history or 'decompression stress' associated with the initiating dive or dives. David Doolette's series of articles have described the modelling principles underlying decompression tables and dive computers and illustrated these in a report on a series of deep 'technical' cave dives. All such models are theoretical: we do not know how inert gas kinetics actually behave in the body.

Sayer and his Scottish colleagues assess whether hyperbaric treatment requirements in divers presenting with decompression illness can be linked to a simple measure of decompression stress, based on the pressure/time data stored in the presenting divers' dive computers. They calculated a 'nitrogen loading index' (NLI) for the incident dives and any preceding repetitive dives, and compared these with the no-stop square-wave profiles from the BSAC/RNPL 11 tables. Finally, they examined whether there were any relationships between the NLI (calculated in various ways) and the hyperbaric oxygen therapy given. This fairly simplistic approach did not demonstrate any relationships between the two. Nevertheless, the concept of linking exposure severity with the treatment given is an interesting one. The ability to download real-time data from divers' computers is improving all the time and may yet provide a useful tool in the management of decompression sickness. At present, apart from the presenting clinical status of the diver, there are no useful markers of severity and prognosis.

Continuing with decompression problems, the Plymouth group report what most hyperbaric physicians know intuitively: clinical advice regarding air travel after treatment for decompression illness is haphazard rather than having a rational basis. The recommendations of surveyed hyperbaric units are extraordinarily variable – from immediately to six weeks. That many chambers could not even provide these data due to limitations in staffing and/or record-keeping, and few followed up such patients, is cause for concern. In the absence of good data, Acott has provided a detailed physical and physiological rationale for a four-week delay to minimise recurrences.¹

The 'normal' lung is an amazing organ, delicate yet resilient, that takes a lot to damage it with environmental pressure changes. The lung tolerates crushing on breath hold to depths greater than 150 metres' sea water; gas-flow changes of 40- to 50-fold from resting minute ventilation to a maximum voluntary ventilation of more than 200 l.min⁻¹; large postural changes in intra-thoracic blood volume and rapid changes in trans-mural pressure with coughing and sneezing. In disease states, there is loss of elasticity, abnormal sheer forces develop between adjacent lung regions and air trapping from 'physiological' airway closure and/or obstruction are common. These must be risk factors for pressure-related pulmonary injury and yet no clear links

between pulmonary pathology and dysbaric injury have been established. Even in the healthy lung there are limits to these tolerances as illustrated in the case report by Sharkey. What is a constant surprise to the writer is why this type of injury does not happen more often.

Emergency ascent training may take a number of forms, e.g., buoyant free ascent as for submarine escape training, buddy-breathing ascent, controlled emergency swimming ascent and alternative-air breathing ascent. Diver training agencies differ in their recommendations, but apart from where military operations dictate, training in rapid ascents is no longer practised. Alternative-air-source ascent is widely considered the best and safest option, but even here confusion can arise, and Acott has drawn attention to the possibility that a first-stage regulator might not meet the needs of two panicked divers breathing simultaneously from the same air supply.²

Peter Fenner continues his invaluable series on marine 'nasties', focusing in this issue on Australian box jellyfish. Surf Life Saving Queensland maintains an excellent web site (www.marinestingers.com.au) with useful links, including to Dr Fenner's (<http://www.marine-medic.com.au>).

No sooner does the Society allow Robyn Walker off watch as President, than the Australian Defence Forces (ADF), recognising her considerable abilities, promote her to Commodore, with effect from 4 July, and transfer her to Defence Headquarters in Canberra. Commodore Walker is the first-ever, female star-ranking health services officer and, I understand, now the second-most-senior-ranking female officer in the ADF. Dr Walker's final report as SPUMS President can be read on page 97. Some aspects of Robyn's work for the Society will never reach the public eye. Suffice to say that she held the reins through some very difficult times and we have much to thank her for. We wish her well in her continued meteoric naval medical career.

It is with deepest regret I report the death of Gareth Jones earlier this year in a road accident in Thailand, where Gareth was working as a dive instructor. Many members will know Rees and Moira Jones, from Whangarei, New Zealand, and have met Gareth at past SPUMS ASMs. The Society extends its condolences to the Jones family in their loss.

References

- 1 Acott CJ. Flying after recompression treatment for decompression illness: why wait four weeks? *SPUMS J.* 2004; 34: 203-8.
- 2 Acott C. 457 equipment incident reports. *SPUMS J.* 2001; 31:182-95.

Michael Davis

The Journal is always interested to publish news of Society members. Please do let the Editor know of items. Front cover photos courtesy of Lisa-ann Gershwin and Peter Fenner

Editorial

The new SPUMS web site

Robyn Walker

Avid readers of the Journal and Annual Scientific Meeting (ASM) delegates will be aware that the SPUMS Executive Committee made a decision last year to upgrade the SPUMS web site. When originally developed, the current web site met our needs; however, in face of advancing technology this is no longer the case.

The web site is the face of the Society and the Internet is a valuable marketing tool. A 'clunky' web site does not encourage repeat visits and if not updated regularly the information rapidly becomes obsolete. An out-of-date web site does not encourage new members to join.

A shortlist of three companies was approached to compete for the SPUMS business and Squiz.Net was successful. This company has a breadth of experience in both corporate and Government circles and has produced a very professional web site.

The new web site will provide the capability for new members (both Australian and overseas) to join up over the Internet and current members will also be able to pay their

yearly renewal fee in this manner. A secure payment gateway (Securepay) will ensure financial security is of the highest level. Our membership database will be automatically updated, which is hoped will reduce the amount of secretarial support time we require. Members will be able to update their contact details on line as they change from time to time.

Web site management will become more automatic with prompts to the Committee for regular updating or archiving of material and the capacity to bulk e-mail the membership with ASM updates or Journal matters will be possible for the first time.

A search engine capability and a secure area for members only has been included. In the future this could allow the Journal to be posted electronically for those whose preference is for electronic delivery, thus reducing printing costs for the Society.

Many of the features allow for future development and the Committee will provide updates as these become available. We are currently awaiting the final e-commerce linkages to be completed and expect the web site to go live in the near future, probably by the time you receive this issue of the Journal. I encourage you to visit the Society on www.spums.org and to ensure your contact details remain current.

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Original articles

Analysing dive-computer profile integrations from incidents of suspected and actual decompression illness using cumulative nitrogen loading

Martin DJ Sayer, Elizabeth J Cook, Colin M Wilson and Jane Barrington

Key words

Decompression illness, computers diving, nitrogen, models, treatment

Abstract

(Sayer MDJ, Cook EJ, Wilson CM, Barrington J. Analysing dive-computer profile integrations from incidents of suspected and actual decompression illness using cumulative nitrogen loading. *SPUMS J.* 2005; 35: 59-66.)

The depth/time data derived from the dive computers of 48 divers presenting with actual or suspected decompression illness at the Dunstaffnage Hyperbaric Unit near Oban, on the west coast of Scotland, were integrated using pressure-dependent algorithms that generated totals of nitrogen loading. The profiles of dives that preceded the incident dives were also integrated and nitrogen penalties were added to the incident totals as dictated by surface-interval durations using half-life values of either two or four hours. The final matrix of totals was compared with the no-stop square-wave profiles for the BSAC/RNPL 11 tables using the published ascent and descent rates. The nitrogen loading index was derived when expressed as a proportion of the no-stop values at the same maximum or average profile depth indices. The calculation of the nitrogen loading index permitted direct comparison of multi-level, computer-controlled dive profiles with simple depth/time tables. The profiles were analysed by three sub-categories whereby the incident dive was (a) part of a multi-dive series, (b) had an ascent rate that generated a rapid ascent warning, and/or (c) was deeper than 35 metres' sea water. In general, the presenting group tended to exceed the no-stop table values when the analyses employed maximum depth of the incident dive, used a 4-hour half-life for calculating penalties from the preceding dive(s), and the incident dive was part of a multi-dive series and/or was deep. There were no significant relationships between any of the variables examined and the extent of hyperbaric oxygen treatment received.

Introduction

The majority of recreational divers presenting to hyperbaric treatment centres for recompression therapy rely on dive decompression computers as their primary method of decompression calculation.¹ Many dive decompression computers have a facility to store dive-profile information at varying levels of resolution and duration.¹⁻³

The ability to download dive-computer information from patients presenting to recompression chambers gives the treating physician an indication of the nature of the incident dive and the preceding dive history. This can be of assistance where the diver is confused or has no physical record of the dive history. Often, within the limitations of the respective computer logging regimes, the dive profile can show an exaggerated record of the patient's perception of the incident dive.² However, although the treating party gains a visual representation of the incident dive or dive series and is presented with an electronic record of the preceding dive history, unless there is a very obvious indicator of what has caused the incident (e.g., a rapid and uncontrolled ascent from depth) it can be difficult to relate the given profiles to the severity of presentation. The treatment of divers who present with dive computers that can be

interrogated must be one of the few areas of medicine whereby the physician can access such a detailed and accurate record of the incident(s) that has caused the illness. However, few studies have investigated these profiles in detail within the context of the eventual treatment regimes.

Diving activities that base their decompression calculation on dive computers tend to generate a different dive profile than diving operations at work or controlled by decompression tables. Because computers continuously recalculate the decompression schedule based on the ongoing depth changes, they allow for extended-duration diving to be undertaken if the majority of the dive is carried out in water shallower than the maximum depth reached. This contrasts with most decompression tables where a single, maximum depth value is used in the calculation.³⁻⁶ It could be assumed that regimes of single- or multi-day diving that undertake one or more multi-level, decompression algorithm-controlled dives at, over or close to the limit of not incurring decompression stops, are provocative in terms of contracting symptoms of decompression illness. At present, it is difficult to compare algorithm-generated decompression schedules with tables derived from extended development histories.

The objectives of this study are to derive a method that produces evaluations from dive-computer downloads of the likely severity of decompression illness that the presenting diver could experience. The development of a standardised index of exposure will allow for direct comparisons between computer-derived and table-derived dive profiles and decompression schedules. Finally, the exposure indices are compared with the type of treatment and the eventual outcome of treatment for a selection of patients.

This study is based on downloads obtained from the UWATEC Aladin™ series of dive computers from patients presenting to the Dunstaffnage Hyperbaric Unit near Oban from 1996 to 2002. The choice of these computers reflects only the popularity of that series of dive computers during the study period. They also have a relatively high level of data storage, retained the same software format over the total duration of the study and it was possible to interrogate the raw data sets. It is in no way a reflection of any relative effectiveness of this family of dive computers for controlling decompression schedules.

Methods

The UWATEC Aladin™ dive computers, when downloaded using the proprietary PC interface and software, give a varying amount of data depending on the model used and the information entered into the computer prior to the dive. However, the minimum download information given by these computers is a depth/time profile graphic based on the maximum achieved depth during 20-second time increments, visual representations of tissue saturation levels and some basic information on water temperature. In addition, a logbook is generated in electronic format that collates records of all the dives undertaken with that computer. If not downloaded regularly, it will store the last dives in detailed format only if the cumulative dive times are within a maximum of 180 minutes. Using independent additional interrogation software, the original logbook download can be presented differently in order to display rates of depth change, air consumption (where an integrated air-pressure monitor is employed), and tissue model saturation, and all the raw data can be transferred into standard spreadsheets for analysis.⁷

Data for this study were obtained from 48 dive-computer records obtained from some patients treated at the Dunstaffnage Hyperbaric Unit from July 1996 to May 2002. From those 48 computer downloads there were, in total, 127 dives recorded in detail or related directly to the incident dive. Downloaded dives were ignored if there was a break of more than 24 hours between them and the incident dive, or them and the first dive in the series resulting in the incident dive. Raw data from all the data logbooks were transformed into spreadsheet format. Where nitrox was used by the diver as a breathing gas the equivalent air depth (EAD) was calculated using the formula:

$$\text{EAD} = \frac{\text{Depth}_{\text{abs}} \times f\text{N}_2}{0.79} - 10$$

where $\text{Depth}_{\text{abs}}$ is the gauge depth in metres' sea water (msw) + 10, and $f\text{N}_2$ is the fraction of nitrogen in the mixture.² The raw data (both for air and equivalent air dives) for each dive were integrated using the partial pressures of nitrogen (ppN_2) calculated for every depth at the recorded 20-second intervals. The integration assumed a direct linear progression between each depth recording and standardised to values per minute using the trapezoid equation:

$$0.5 \times (\text{pN}_{n-1} + \text{pN}_n) / 3$$

where pN_n was the recorded ppN_2 value at time n and pN_{n-1} was the preceding ppN_2 value. Cumulative values of ppN_2 (ΣppN_2) were obtained for each downloaded dive through summation of all values standardised to a minute's duration.

In a series of dives that produced an incident of decompression illness, penalties were calculated by reducing by half the ΣppN_2 values for the previous dive in either 2-hour or 4-hour increments of the surface-interval time to produce 2-hour or 4-hour half-life penalties. Each temporal increment needed to be complete to generate the next half-life reduction and the resultant penalty was added to the next dive.

In order to compare the computer dives with a recognised decompression table, simulated dives were interrogated as above in accordance with the no-stop limits on the BSAC/RNPL 11 tables and employing the prescribed ascent and descent rates for those tables. This produced curvi-linear relationships of no-stop values for ppN_2 for either the maximum or average depth reached in a dive. Average depths were employed to compensate for dive profiles where the time at maximum depth was minimal compared with the total duration of the dive. Average depth for both the table-generated profiles and the dive-computer download profiles was a calculated mean of all the 20-second interval profile depths.

Using the methodologies detailed above, single values of ΣppN_2 were calculated for each of the dive series examined for each of the two half-lives employed. Initial analyses of the data compared resultant ΣppN_2 (with both 2-hour and 4-hour half-lives) for each casualty with the BSAC/RNPL 11 no-stop relationship using either maximum or average incident-dive depths. ΣppN_2 values for each casualty were classified as being:

- multi-dive series (where there was at least one dive preceding the incident dive by no more than 24 hours)
- rapid ascents (where the ascent rate of the incident dive exceeded the dive-computer guidance and generated an ascent warning on the download)
- deep dives (where the maximum depth of the incident dive was deeper than 35 msw).

Expressing the dive-computer ΣppN_2 values as a proportion of the BSAC/RNPL 11 no-stop limits for the same depth

(either maximum or average) generated indices termed here as the nitrogen loading index (NLI). This gave values of 1.0 to all no-stop profiles so that index values greater than 1.0 exceed no-stops for that decompression table. The NLI values were calculated for all the dive-computer downloads and were again analysed in the groups of multi-dive series, rapid ascents and deep (> 35 msw) dives.

The following primary recompression tables were used in the treatment of the 48 divers in this study:

- Unmodified Royal Navy Table 62⁸
- Modified Royal Navy Table 62 (with extensions at 18 msw and/or 9 msw)⁸
- Royal Navy Table 62 converted to US Navy Table 7.⁹

Where residual symptoms were still present following primary treatment, some patients were retreated with varying numbers of one of the following two secondary tables:

- Comex 12¹⁰
- Royal Navy Table 66.⁸

For each patient, the total therapy received in all primary and secondary treatments was calculated as ‘oxygen units of treatment’, which were the sums of the partial pressures of oxygen per minute breathed by the patient, assuming 100% delivery. The hyperbaric treatments, expressed in oxygen units, were then assessed against the maximum or average depths of the incident dive, the ΣppN_2 values and the NLI for the three incident groups.

Results

Without added penalties, the ΣppN_2 values ranged from 38 to 161. Use of the stated maxima for descent and ascent rates for the BSAC/RNPL 11 decompression tables results in a greater proportion of the total dive time being taken up with travelling to and from maximum depth as the maximum depth increases. In addition, there is a decreasing fraction of total dive time permitted at the maximum depth with increasing depth following the no-stop parameters. As a result, if the no-stop times are followed, the average dive depth values equate approximately to the maximum dive depths from 10 to 24 msw before tending toward an asymptotic value of between 32.1 and 34.2 msw. The relationship between maximum and average depths of a dive profile will differ markedly with profile and in the incident dives examined there were marked differences between rapid-ascent and staged decompression profiles. However, in the total dataset examined here, there was a positive linear relationship between the maximum and average depths of the incident dives whereby:

$$d_{avg} = (0.29 \times d_{max}) + 9.85$$

where d_{avg} is the average depth (msw) and d_{max} the maximum (msw), although the relationship was not strong ($r^2 = 0.376$).

Both maximum and average depths for the no-stop BSAC/RNPL 11 profiles were used to generate ΣppN_2 values for the table-generated profiles and produced negative curvi-

linear relationships of higher ΣppN_2 values at shallow compared with deeper depths (Figure 1).

The ΣppN_2 values for each incident were calculated: without penalty; with penalties added throughout the series of dives, where applicable, assuming a 50% reduction in the relative values from preceding dives after each 2-hour period of surface interval (2-hour half-life); and with penalties calculated with a 4-hour half-life. Twelve out of the 48 incident dives carried no penalty prior to the dive taking place. Of the other 36 incident dives the penalties preceding the incident dive ranged from 0.07 to 79.31% of the final cumulated total when calculated using a 2-hour half-life ($\mu = 24.6\%$ following arcsin transformation), and 4.00 to 86.05% when using a 4-hour half-life ($\mu = 35.4\%$ following arcsin transformation).

All three penalty categories of ΣppN_2 were plotted against the curvi-linear BSAC/RNPL 11 no-stop relationships for both maximum and average depths (Figures 2 and 3). If it is assumed that cumulative totals that occur above the no-stop relationships represent the potential for being outside the decompression limits of the table, then using the maximum depth attained during the incident dive explains 54.2% of the incidents (Figure 2). Adding 2-hour and 4-hour half-life derived penalties for previous diving activity increases the level of explanation to 70.8% and 72.9% respectively (Figure 2). Use of average depth of the incident dive reduced the strength of the trend and only 20.8%, 35.4% and 47.9% of incident dives had totals above no-stops (incident dive, incident dive with 2-hour half-life penalty, incident dive with 4-hour half-life penalty, respectively; Figure 3).

Figures 4 and 5 illustrate the 4-hour half-life data set subdivided by category of incident dive and analysed for either maximum or average depth of incident dive. The influences of analysis format and depth vary between groups

Figure 1
Cumulative partial pressures of nitrogen against maximum (open squares) and average (filled circles) depths (metres) for the no-stop depth profiles as prescribed by the BSAC/RNPL 11 decompression tables.

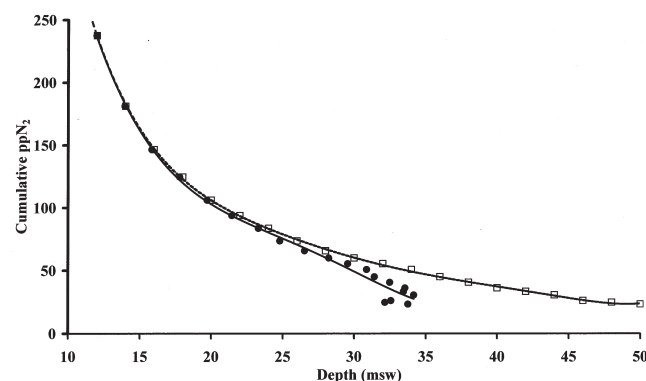


Figure 2

Cumulative partial pressure of nitrogen values plotted against the maximum depth (metres) of the incident dive, without penalty (o), with a 2-hour half-life for off-loading (●) and a 4-hour half-life for off-loading (hatched circles). The solid line represents the BSAC/RNPL 11 no-stop relationship for cumulative partial pressure of nitrogen against maximum depth.

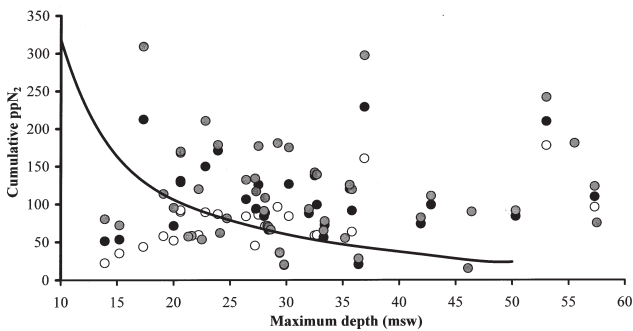
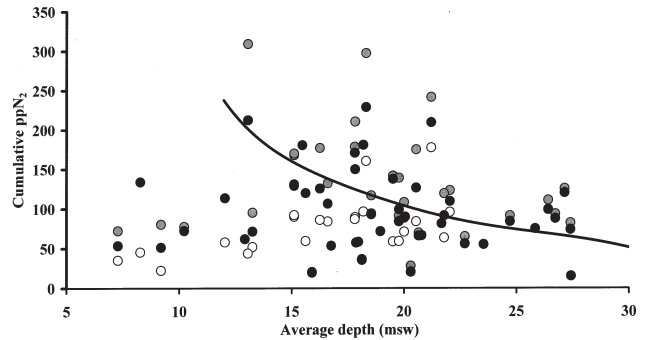


Figure 3

Cumulative partial pressure of nitrogen values plotted against the average depth (metres) of the incident dive, without penalty (o), with a 2-hour half-life for off-loading (●) and a 4-hour half-life for off-loading (hatched circles). The solid line represents the BSAC/RNPL 11 no-stop relationship for cumulative pressure root time against maximum depth.



and the only clear trend is that rapid ascents produce the lowest percentage of incident dives exceeding the no-stop values for the BSAC/RNPL 11 decompression tables (Table 1). The percentages for exceeding no-stop values in the multi-dive series and deep dive categories were much higher (61.9–90.9%) with most of the higher values obtained when analysed by maximum depth of incident dive (Table 1, Figures 4 and 5).

dive (NLI_{max}) ranged from 0.34 to 10.37 with a mean (\pm sd) value of 2.07 (\pm 1.77; $n = 48$). Using the average depth of the incident dive, the NLI values (NLI_{avg}) ranged from 0.16 to 2.67 with a mean (\pm sd) value of 1.10 (\pm 0.58; $n = 44$). The difference in sample number between the group using the maximum depth of the incident dive and that using the average depth was caused by four samples having average depths shallower than depths that could be computed on the BSAC/RNPL 11 decompression tables. The NLI_{max} and NLI_{avg} groups were tested for deviation away from the

The NLI derived using the maximum depth of the incident

Figure 4

Cumulative partial pressure of nitrogen values with a 4-hour half-life penalty from preceding dives plotted against the maximum depth (metres) of the incident dive for: (a) all dives; (b) multi-dive series; (c) rapid ascents; and (d) deep dives (>35 msw).

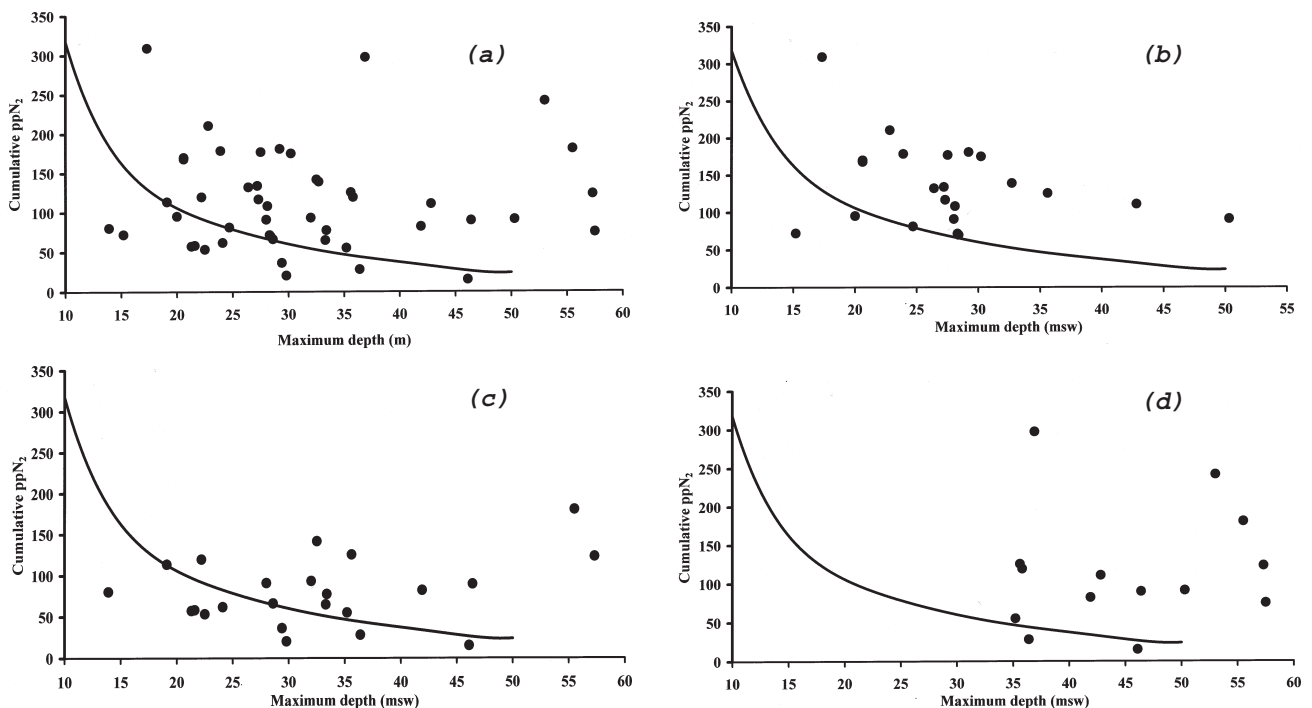


Table 1

Percentage (%) of incident dives exceeding the no-stop values for the BSAC/RNPL 11 decompression tables as derived from the integration of the computer profiles using cumulative partial pressure of nitrogen and either the maximum or average depths of the incident dive. All values carry a penalty from any previous dives as calculated assuming a 50% reduction in values after every four hours of surface interval. The data are presented for four categories of incident dive (a single incident dive could be in multiple categories).

	% incident dives using	
	Maximum depth	Average depth
All dives	72.9	47.9
Multiple dive series	90.9	61.9
Rapid ascents	52.2	26.1
Deep (>35 msw) dive	85.7	71.4

normalised no-stop value (1.00) using chi-squared analysis following tests for homogeneity of variance where the assumption was that the no-stop value was the expected variable. Taken as a single group, NLI_{max} levels were significantly greater than the no-stop values (χ^2 ; $P < 0.001$); NLI_{avg} levels were not significantly different from 1.00 (χ^2 ; $P > 0.05$).

The NLI values for both the maximum and average depths of the incident dives were divided into the three analysis categories: multi-dive series, rapid ascents and deep dives. Incident dives that had classified deep maximum depths produced the highest values (Figure 6). NLI_{max} levels for all analysis categories were significantly greater than the no-stop values (χ^2 ; $P < 0.05$) apart from the value for multi-dive series (χ^2 ; $P > 0.05$; Figure 6). NLI_{avg} values for multi-dives and deep dives were significantly greater than those for rapid ascents (Student's t-test; $P < 0.01$ in both cases; Figure 6). Deep dives produced significantly greater NLI_{avg} values compared with multi-dive series (Student's t-test; $P < 0.05$).

The total hyperbaric treatments employed on the cases detailed in the present study, as calculated in terms of oxygen units, ranged from 0 to 2240 (mean \pm sd, 882 ± 357 ; $n = 48$). These totals could result from no treatment (in one of the 48 cases), a single treatment or a series of treatments. When total oxygen units of treatment were compared separately against all incidents, incidents from multi-dive series, incidents from rapid ascents, and incidents from deep (>35 msw) dives using ΣppN_2 , there were no significant relationships ($P > 0.05$ in all cases). In addition, there were no significant relationships between resultant hyperbaric treatment and the maximum or average depth of the incident dive, the pre-incident dive penalties, or the NLI values for maximum or average depths of the incident dive ($P > 0.05$ in all cases).

Figure 5

Cumulative partial pressure of nitrogen values with a 4-hour half-life penalty from preceding dives plotted against the average depth (metres) of the incident dive for:

(a) all dives; (b) multiple dive series; (c) rapid ascents; and (d) deep dives (>35 msw).

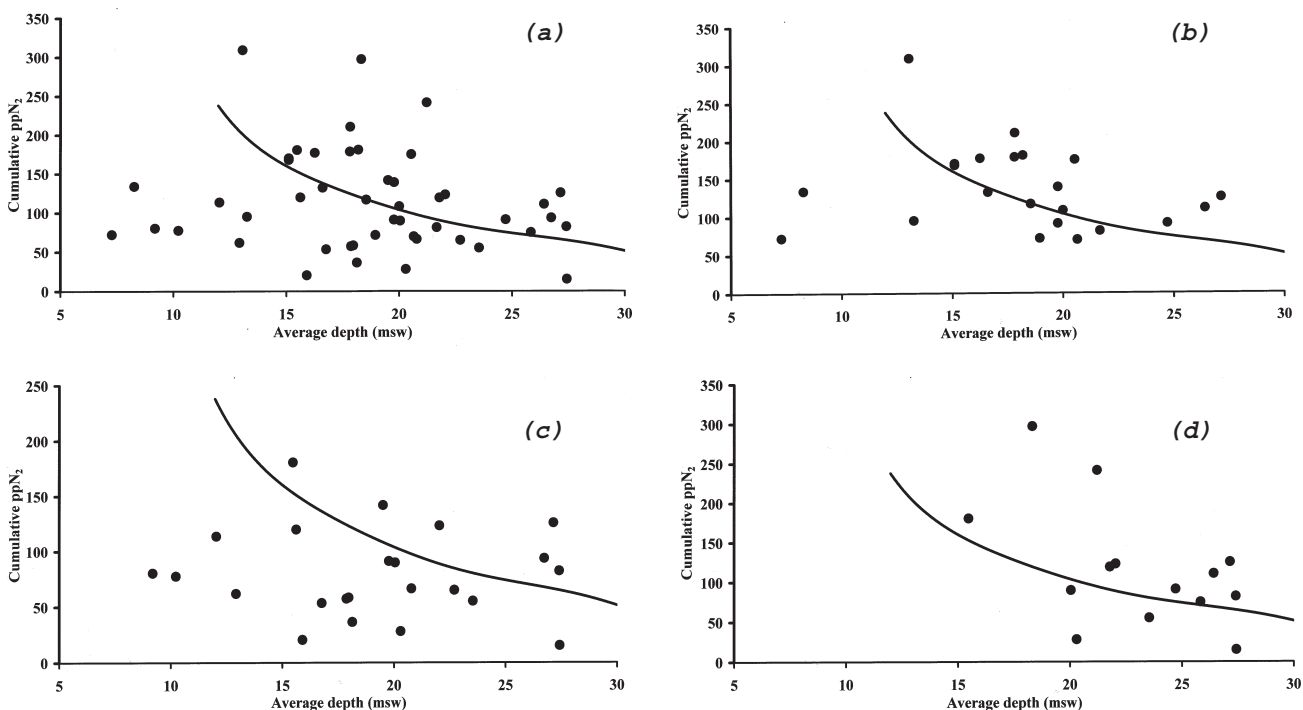
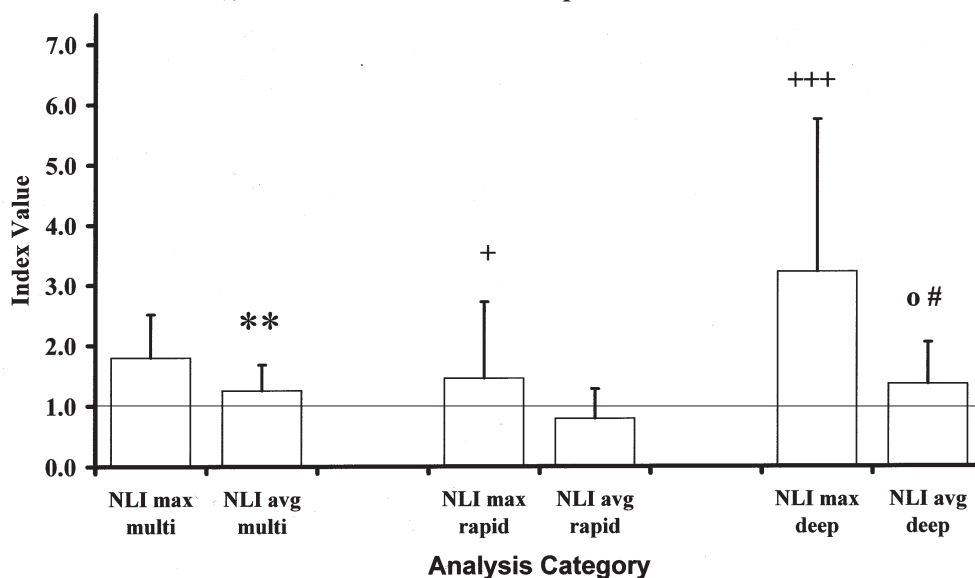


Figure 6

Nitrogen loading indices (NLI) derived for the maximum and average depths of the incident dives analysed against and between the categories of multi-dive series (multi), rapid ascents (rapid) and deep dives (deep). Significant values are indicated for deviation from no-stop levels (+), differences between rapid and deep categories (o), differences between multi and rapid categories (*), and differences between multi and deep categories (#). Number of symbols indicates the levels of significance (one = $P < 0.05$, two = $P < 0.01$, three = $P < 0.001$); horizontal line is the no-stop value where $NLI = 1.0$.



Discussion

This study presents a method of assessing multi-level, decompression computer-managed dives from divers presenting with actual or suspected decompression illness (DCI) and of comparing the outcomes of those dives with no-decompression dives derived from a set of long-established square-profile decompression tables. High rates of predicting DCI from cumulative nitrogen loading alone occur only when the incident dive was:

- the last of a multiple dive series
- relatively deep (a maximum depth greater than or equal to 35 msw)
- the calculations used the maximum depth of last dive
- if a surface-interval half-life of four hours was employed.

Even if the 'worst-case' factors were used to obtain estimates of the potential severity of the resultant decompression illness, these estimates did not relate to the duration or type of eventual hyperbaric oxygen treatment.

The present study has examined the profiles from the dive computers of divers who have presented with decompression illness. The overall objective of the study was to determine whether the type and/or duration of hyperbaric treatment could be informed following the integration of the profiles of the incident dives or series of dives. The methodology employed in the present study was designed to produce single indices of nitrogen loading. However, there are a number of limitations to the approach used. Although the modern versions of dive computers give indicative profiles of the dives undertaken, memory

restrictions yield relatively basic levels of data recording. For the series of computers used in the present study, records were limited to only the deepest depth reached during every 20-second period and only the last 180 minutes of profile information. The last 180 minutes of storage was again limited to complete dives only. So, if the last dives were long (and, in particular, where prolonged periods of staged decompression were employed) or the incident occurred as part of a long series of dives, some dives that may have affected the resultant indices could have been missed. However, the level of detail in the recording analysed in this study does satisfy the minimum levels of accuracy suitable to describe dive profiles for analysis of decompression data.^{11,12}

All of the analyses undertaken in the present study were made against the profile information of the final dive in the series. The assumption was that the last dive was the one that most influenced the actual incident. However, there were occasions when the onset of decompression illness could well have begun earlier in the series. Diving for three days or more without a break increases the risk of contracting DCI on subsequent dives irrespective of the predictions of the decompression model employed.¹³ Despite this, the only significant outcomes from the analyses were obtained when maximum depth of the final dive was used.

As an influencing factor, maximum depth will be a true descriptor of the dive profile only when the average depth is closer to the maximum. Related to this effect is the fact that nearly all the dive series where the final dive was

classified as being deep were correlated against the derived indices. Some of those correlations will be anomalies of the methodology employed in that the total values will continue to accumulate as the diver ascends irrespective of whether the diver is following a staged decompression profile or not. In addition, the methodology employed here is too simplistic to calculate the benefits of decompression that is intentionally staged at set depths and times, or where it is concomitant through a normally controlled, non-staged ascent. Similarly, the cumulative methods used in the present study will ignore the effects of rapid ascents and, in effect, the quick cessations in the pressure-affected values caused by rapid ascents will generate low cumulative index values. The model-independent methods of the present study also ignore the more generally accepted Haldanian theory of multi-compartmental approaches to decompression modelling and could have further examined the differences and rates of relative change between compartmental and ambient pressures.^{14,15}

The use of a single half-life value for calculating the effects of off-loading between dives in a series is simplistic but is based on the square-profile tables used to derive no-stop values in this study.¹⁶ In effect, this methodology is employing a single-compartment approach to determining decompression risk with the exponential off-loading rates for that single compartment of either two or four hours. Decompression algorithms based on Haldanian theory and the computations of Buhlmann can use between 6 and 16 tissue compartments with half-life times ranging from 2.5 to 640 minutes.^{1,17} In the present study, the only significant indications of decompression problems were obtained when a 4-hour (240-minute) half-life was employed. Although that value compares with the upper range of tissue half-lives used in many decompression computers, this ignores the subtlety of the multi-compartmental approach. However, it cannot be discounted that in multi-day, multi-level diving, the controlling compartments are more likely those attributed to the slower tissues.

The fact that there were quite a few profiles that, once analysed, did not exceed the no-stop values was partly related to the use of one of the least conservative decompression tables.¹⁶ The no-stop values for the BSAC/RNPL 11 table will yield much higher cumulative values for nitrogen that form the maximum no-stop line in the analyses reported here compared with other decompression tables. That line would be lower if other tables, such as those derived by the Defence and Civil Institute of Environmental Medicine (DCIEM), were used, and lower again if pre-dive penalties were employed for the assessment of multi-dive series. In addition, the indices derived as a proportion of the maximum no-stop line would increase if more conservative or multi-dive values were used.

The sample size for the study was relatively small ($n = 48$) and no additional breakdowns attributed to age, sex, experience, etc., were attempted. Dividing the main

population into sub-sets depending on the type of incident dives (rapid ascent, multi-day diving and/or deep diving) reduced the population sizes further ($n = 15-23$). The study was restricted in that entry to it was limited to a single type of dive computer and a single recompression treatment centre. In addition, the population analysed in this study was pre-selected in that the profiles were from divers with actual or suspected decompression illness. There are no comparisons with the profiles generated by divers who do not show signs or symptoms of decompression illness even though they may be diving at depth and/or multi-day diving. Therefore, neither the distributions shown here nor the scale of the generated indices can, at this time, be given as potential indicators of decompression illness.

In the present study recompression treatment time, recorded as cumulative oxygen units, was used as a proxy indicator of the severity of decompression illness that was treated, based on the assumption that more severe cases of decompression illness require more prolonged treatment. However, no trends at all were discernible with the duration of treatment(s). Again, the effects of rapid ascent are not represented well by the analysis technique. In addition, treatment time cannot contend with differences in patient self-assessments that may influence the treatment duration. Finally, there is evidence to suggest that changes in the treatment service over the duration of the present study may have influenced the efficacy of treatment where these changes were directly related to treatment time through employment of either extended primary treatments or multiple secondary treatments.¹⁸

This present study has demonstrated methods that can be used to compare multi-level, computer-controlled dive profiles against square-wave, empirically-tested, table-derived decompression schedules. By using the maximum depth of the last dive in any series combined with a 4-hour half-life for nitrogen off-loading between dives, a significant number of incidences of DCI, whereby the dives are part of a series or the last dive is deeper than 35 msw, can be explained using this approach. However, this is a relatively simplistic approach that fails to compute any beneficial effects of staged or unstaged decompression, or the negative effects of rapid ascent. There are a number of studies that have used probabilistic decompression modelling to attribute risk values to specific diving regimes and/or profiles.^{2,19-22} The use of similar approaches on profiles that have resulted in decompression illness, when compared with other diving groups, would inform future acceptable limits of probabilistic risk.

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Time to fly after hyperbaric chamber treatment for decompression illness: current recommendations

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Key words

Decompression illness, decompression sickness, treatment, flying, altitude, questionnaire

Abstract

(St Leger Dowse M, Barnes R, Smerdon G, Bryson P. Time to fly after hyperbaric chamber treatment for decompression illness: current recommendations. *SPUMS J.* 2005; 35: 67-70.)

Divers suffering decompression illness (DCI) increasingly undertake high altitude travel after hyperbaric treatment. Anecdotal evidence suggests hyperbaric medical professionals give widely differing advice regarding the safe time to fly after treatment (TFAT), resulting in possible health, socio-economic and insurance implications. Thirty-two chamber facilities were contacted to determine current trends in advice on TFAT and the rationale behind these trends. Twenty-three (72%) facilities responded of which five returned incomplete data, and 18 returned data fulfilling all the criteria. This study collates the current advice given by staff at these 18 chambers, and the basis on which it is given. Only one of the responding chambers had no relevant guidelines. Advised TFAT differed widely, varying from immediately to six weeks. Seven chambers gave advice based on Divers Alert Network (DAN) recommendations, two based on research, and the remaining chambers relied on local staff advice based on their own experience. Only six chambers followed up divers after treatment, leading to a paucity of data regarding complication and recurrence rates following 'return home'. Repeated contact with chambers revealed many units kept inadequate records, or did not have the staffing available to collate information for this study, limiting the success of this type of research. Guidelines on TFAT for DCI vary radically between chambers, and are rarely evidence based.

Introduction

Throughout the world, divers on holiday presenting with decompression illness (DCI) are treated in hyperbaric chambers. Once treatment concludes, part of the medical discharge process includes advice to the diver about when to seek further medical opinion, when it is considered safe to resume diving, and when it is safe to make the return flight to their home country. As there are increasing numbers of tourists who dive whilst on holiday, higher numbers of divers are being treated for DCI and more of these divers are embarking on short- and long-haul flights home after treatment.¹ It is therefore particularly important to know when it is considered 'safe' to fly again, in order to resume work and family life.

Few if any studies have been specifically designed to observe any relationship with recurrences or complications of DCI with altitude provocation after treatment. However, anecdotal evidence does suggest chambers give varying advice regarding TFAT. The aim of this study was to attempt, by means of a questionnaire sent to recompression chambers that treat divers, to collate current advice and the basis on which it is given.

Methods

Hyperbaric chambers routinely treating divers for DCI and located at busy, international, holiday diving destinations served by both short- and long-haul flights were identified.

A questionnaire (Appendix 1) investigating current trends in advice on high altitude travel after treatment was mailed and e-mailed to treatment chambers; data confidentiality was assured. Principal data gathered included chamber guidelines regarding TFAT, the basis of and rationale behind the guidelines (scientific, advice from other authorities, severity of DCI, success of treatment, number of treatments required, 'instinct'), follow-up procedures, and whether records were kept of divers who fly after treatment for DCI.

The initial point of contact at the chambers was the medical director of the unit. Chambers were re-contacted repeatedly by e-mail and/or telephone in order to improve the response rate; continued point of contact was either the medical director or other senior staff.

Statistical analysis was not used in this study. Data are presented as recorded by the chambers from the questionnaire. Data were evaluated from fixed-option responses, and from free-form solicited text that related to defining the origins and development of TFAT recommendations used by the chambers.

Results

Thirty-two chambers were contacted, of which 23 (72%) responded. Of these, five chambers, although expressing willingness to participate, were unable to provide data suitable for inclusion. Therefore, complete data for analysis were available from 18 (56%) of the chambers contacted.

Table 1
Recommendations, source and basis of advice given by 18 responding chamber facilities

Chamber	Recommendations	Basis of recommendations
1	72 hrs	Not given; prudence
2	Different for each case	DAN; "papers"
3	72 hrs to 7 days	DAN; clinical experience; instinct
4	72 hrs	DAN; success of treatment
7	6 weeks	"Science"; success of treatment; experience; instinct
8	2 weeks	Local staff opinion; altitude exposure post diving
9	3 weeks	"Science"
10	4 weeks	Local staff opinion
12	"immediately" to 4 weeks	Local staff opinions differed widely
16	5 days	Review of own data
17	3 days	No guidelines; instinct
26	"immediately" to 72 hrs	DAN; common sense
27	72 hrs	DAN
28	72 hrs; more if severe	Not given
29	72 hrs	Not given
30	72 hrs	DAN and HTNA presentation
31	72 hrs	DAN
32	72 hrs	Clinical experience; instinct; severity of DCI

Relevant guidelines were reported by 17 of these 18 chambers. The advised TFAT in these guidelines differed widely between chambers, varying from immediately to six weeks post cessation of treatment, with the range including: immediately, 24 hours, 72 hours, five to seven days, two, three, four and six weeks (Table 1). In one chamber different personnel quoted immediately, 24 hours and four weeks definitively. Seven chambers definitively quoted 72 hours as TFAT, with 13 chambers stating they generally had a TFAT policy of less than six days. Some chambers additionally have guidelines for high altitude land-travel, with one unit recommending no land-travel over 300 metres for 14 days after treatment. Reduced TFAT times for short-haul (not defined) internal flights as opposed to longer international flights were also intimated. Follow-up phone calls to request return of the questionnaire revealed not all the staff were initially aware of the presence or content of the guidelines within their unit.

Of the 18 chambers that did provide details of the rationale behind their TFAT recommendations, seven quoted Divers Alert Network (DAN) as the reference for their guidelines. Of this group, one also quoted "papers", (which they were unable to reference), and another chamber quoted "science" (a presentation at a Hyperbaric Technicians and Nurses Association meeting in Australia in 1998) as additional support. The remaining chambers based their guidelines variously as follows: the success of previous treatments, experience, instinct, prudence, common sense, review of own unit historical data, and current guidelines for altitude exposure post diving. One chamber stated that advising a delay prior to flying was "useful" as it provided time for a follow-up review of the patient. Six chambers reported following up patients after discharge, with times varying

from one month to three months. Only four chambers regularly kept records of divers flying home after treatment.

Discussion

Concern regarding flying after treatment for DCI stems from both the perceived theoretical risk and anecdotal evidence of relapse during flight. The mechanisms of relapse are postulated to be multi-factorial. Commercial aircraft are normally pressurised to approximately 2,400m, thus according to Boyle's law, any gas bubbles still present will expand in this reduced pressure. It is also postulated that there may be new bubble growth.^{2,3} Relative tissue ischaemia in this hypoxic environment may be more important in relapse of DCI, as bubbles are thought unlikely to remain in a treated case of DCI. This concept, however, remains unsubstantiated.

It can be seen that advice concerning TFAT currently varies widely between hyperbaric chambers. The advice is given with the intention of reducing the risk of complications that may arise due to the atmospheric changes involved in flying. This theory is derived mainly from work conducted on the relative risk of developing DCI during air travel after diving.⁴⁻⁸ The few published papers regarding the time to fly after treatment for DCI, although valuable and interesting, are based on little human research and are open to debate.⁹⁻¹¹

In 1989, the 39th Undersea and Hyperbaric Medical Society Workshop debated guidelines for recreational divers on pre-flight surface time following diving. The same workshop addressed the issue of flying after treatment of decompression sickness.¹² However, no formal definitive guidelines on TFAT resulted.

Butler, in 1992, asked

*“when was it safe for a diver who had sustained an episode of DCI to ascend to altitude?”*¹

He concluded that

“with the increasing popularity of recreational diving and the greater mobility of diving populations, flying after diving will continue to occur with greater frequency. Consequently, detailed follow-up studies of treated divers are now essential.”

Since that time a number of papers have been published theorising and attempting to address the issue of TFAT. These have included case reports, interrogation of retrospective data and, more recently, prospectively attempting to gather data from treating chambers.^{7,9-11} Recently, Acott, having extensively reviewed the current literature and debated the physics and physiology of flight, concluded that four weeks post treatment for DCI was a reasonable time to fly home.³

The wide variation in guidelines for TFAT amongst the chambers returning data for this study is not surprising as there appears to be little or no scientific evidence on which a decision-making process can be based. Some chambers understandably base their advice on theory, their clinical experience and anecdote. It may also be possible that some of the issues relating to TFAT are rooted in recommendations for the time to fly after diving, as cited by one responding chamber.

Staffing levels were often limited, and available resources and the standard of record keeping varied widely between chambers, illustrating that answers to this question from a questionnaire-based study will be limited in value. Five of the chambers contacted stated that their staff were too busy and/or information was not available in an accessible format to answer the questionnaire, but they supported the concept of the study. Thus, these factors were reflected in the response rate to the questionnaire, which was not improved by repeated contact with the chambers or by changing the format of the questionnaire to allow electronic submission.

Telephone contact produced a positive response towards the project, but due to time restraints of staff, full information was not readily available over the telephone. It was during telephone contact that it became evident some chambers did not always record treatments, outcome, and whether or not subsequent high altitude travel was involved. With only some chambers following patients after discharge, there is a paucity of data regarding complications and recurrence rates following return home.

With the advised TFAT guidelines differing so widely between chambers and appearing to be based on scientifically unfounded assumptions, there are significant implications in terms of personal socio-economic issues, together with insurance costs, that are completely unpredictable. All the chambers that responded to the

questionnaire were interested in participating in further research to establish an evidence-based policy for time to fly and high altitude travel after treatment for DCI. The matter of how elements such as short- and long-haul flights are factored into research and TFAT advice would need addressing. It would mean many chambers would have to commit significant amounts of time and energy to work together, implement administrative structures, and follow up treated divers in a disciplined fashion.

Chambers at long-haul holiday destinations are often the busiest with regard to treating divers, and potentially rich in data, but, conversely, the most under-resourced with regard to funding and staff, often relying on local volunteer diving personnel. Thirteen years on from Butler's review, there is clearly still a requirement for further research in this field in order to address these issues, or as one chamber was quoted as saying *“...to bring some order to this madness”*.

Acknowledgement

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Appendix 1

Questionnaire about recommendations for flying after recompression treatment for decompression illness (DCI). All questions pertained to the years 2000, 2001 and 2002.

- 1) Number of divers treated per calendar year:
 - 2) Number of 'holiday' divers requiring to fly or travel to high altitude after treatment:
- In relation to these 'holiday' divers, please answer the following questions:
- 3) Numbers of treated holiday divers with:
 - a) DCI musculoskeletal
 - b) DCI neurological
 - c) Other (please define)
 - 4) Initial tables used for treatment of holiday divers:
 - a) US Navy Table 6
 - b) US Navy Table 5
 - c) Comex 30
 - d) Other (please specify)
 - 5) How many required more than 2 treatments?
 - 6) How many had delayed presentation?
 - 7) How many had a flight to the chamber or long travel issues prior to first treatment?
 - 8) Does your chamber have guidelines on flying after treatment for holiday divers?
Yes / No
 - 9) If Yes to 8), what are your local guidelines?
 - 10) What are these guidelines based on?
 - a) Advice from Divers Alert Network
 - b) Advice from insurance companies
 - c) Advice from local chambers or staff
 - d) Scientific research, if so, please define
 - e) National guidelines
 - f) Other (please specify)
 - 11) If No to 8), what advice do you give holiday divers who need to fly after treatment?
 - 12) Is the advice given based on?
 - a) The severity of the DCI
 - b) The success of the treatment
 - c) The number of treatments required to produce resolution
 - d) The time of onset of DCI to initial treatment
 - e) Instinct; previous good/bad experience
 - f) Other (please specify)
 - 13) Have any holiday diver cases helped to formulate your management and recommendations of time to fly after treatment?
Yes / No
 - 14) If Yes, please define:
 - 15) Do you follow up your holiday divers once home?
Yes / No
 - 16) If Yes to 15), is this done by?
 - a) Letter
 - b) Telephone
 - c) E-mail
 - 17) If Yes to 15), over what time period after discharge?
 - a) 1 month
 - b) 2-3 months
 - c) 4-6 months
 - d) 1 year
 - e) Other (please define)
 - 18) Are records kept of holiday divers who fly after treatment?
Yes / No
 - 19) Are records kept of holiday diver complications and/or deterioration after discharge?
Yes / No
 - 20) Would you be willing to participate in the potential next phase of this project? This will consist of contacting chambers with a questionnaire that would allow us to prospectively follow holiday diver patients treated by that particular unit.
Yes / No

Review articles

Gas-content versus bubble decompression models

David J Doolette

Key words

Decompression models, diving theory, review article

Abstract

(Doolette DJ. Gas-content versus bubble decompression models. *SPUMS J.* 2005; 35: 71-5.)

Decompression models predict the probability of decompression sickness from the characteristics of a dive. The first step in this procedure is to calculate an index of decompression stress from the depth/time/breathing-gas history. Gas-content models and bubble models are two major classes of decompression models that differ in this method of calculating decompression stress. Calculation of decompression stress typically involves simulating the amount of gas (in units of pressure) that dissolves in theoretical 'tissue' compartments during a dive. For gas-content models, the decompression stress is simply any positive value of supersaturation (tissue gas pressure – ambient pressure). For bubble models, the decompression stress is the simulated number or volume of bubbles formed as the result of any supersaturation. These two model classes result in a different shape of decompression, with bubble models typically beginning decompression stops deeper. There is as yet no scientific evidence supporting one format of decompression over the other. Gas-content models are the most widely used method of decompression calculation although bubble models have gained recent popularity with technical divers.

Introduction

Decompression sickness (DCS) is a disease caused by bubble formation in body tissues from excess dissolved gas upon reduction in ambient pressure (decompression). Bert (1878) first made the association between nitrogen in compressed-air breathing, bubbles, and DCS.¹ Haldane and colleagues (1908) developed the first practical decompression model and produced the first decompression schedules. These minimised the risk of DCS by controlling the depth and duration of compression and the decompression rate.²

Decompression models link the probability of decompression sickness (pDCS) to an index of decompression stress calculated from the depth/time/breathing-gas history of a dive. Decompression models developed and tested through experimental dives can then be used to predict the outcome of future, similar dives and therefore be used to produce decompression schedules. A previous paper provided an overview of decompression model structure, examined the probabilistic and deterministic functions used to link decompression stress to outcome, and compared the development and testing of probabilistic and deterministic decompression models.³

This paper provides a brief overview of the biophysical component of decompression models, which is the method of calculating decompression stress from the depth/time/breathing-gas history of a dive. In particular this paper will compare gas-content models and bubble models – two major classes of decompression models that differ in this biophysical component.

Decompression stress

DCS probably results from bubbles formed in body tissues, so a natural choice for measuring decompression stress would be estimation of the number and size of those bubbles. The actual bubbles that cause DCS have not been measured, not least because their size, number, and location have not been identified. Some intravascular bubbles can be detected by ultrasonic methods leading to a useful but indirect measure of decompression stress.⁴ Therefore, in decompression models, decompression stress is not a measured quantity, but rather a theoretical index calculated from the characteristics of the dive thought to influence pDCS, typically the depth/time/breathing-gas history. Decompression stress is typically a calculated index of bubble number or volume (bubble models), or of the excess inert gas that drives bubble growth (content models).

Uptake and washout of inert gas

Calculation of the uptake and washout of inert gas based on depth/time/breathing-gas history is common to both model classes. Breathing gas must be delivered at ambient pressure. With the increase in ambient pressure encountered in underwater diving, the inert gas component of the breathing mixture is absorbed into tissues during a dive, approaching equilibrium with the partial pressure of the inspired gas. Excess inert gas is eliminated from tissues both during and after ascent. The dominant route of inert gas into and out of the blood is via the lungs. Alveolar partial pressure and arterial tension (concentration/solubility, units of pressure) of the inert gases commonly

used as breathing gas diluents (nitrogen and helium) equilibrate rapidly. Therefore, over a time course relevant to DCS, the inert gas kinetics can be reduced to a model of exchange between the blood and the tissues. The main factor that determines tissue uptake and washout of gas is the rate at which gas is carried in the blood perfusing the tissue, although these kinetics are modified by diffusion processes.⁵

Nevertheless, the most common structural model of gas uptake and washout is the single exponential tissue compartment where the rate-limiting process is usually considered blood perfusion. In this context a compartment is represented by a single, time-varying concentration. Underlying this notion is the assumption that, owing to rapid diffusion, equilibration of inert-gas concentration gradients across the tissue region represented by the compartment is much faster than transport in and out of the compartment. In this model, the arterial to tissue inert-gas-tension difference declines mono-exponentially according to a half-time notionally determined by tissue:blood perfusion ($\text{ml} \cdot 100\text{ml}^{-1} \cdot \text{min}^{-1}$) and the blood:tissue partition coefficient of the gas. Figure 1 shows mono-exponential uptake and washout of an inert gas from one such compartment. Several (typically five to sixteen) parallel perfusion-limited compartments with different half-times are used to accommodate different rates of gas uptake and washout across the relevant body tissues.

Bubble formation

If the sum of inert and metabolic-tissue gas tensions (P_{tis}) exceeds ambient pressure (P_{amb}) during or after decompression, gases can leave solution forming bubbles in tissues and blood. In Figure 1 the maximum supersaturation ($P_{\text{ss}} = P_{\text{tis}} - P_{\text{amb}}$) for this particular compartment is indicated.

The pressure inside a bubble (P_{bub}) is the sum of the external pressures applied to the bubble including ambient pressure, pressure due to surface tension, and any mechanical compression from the tissue. Ignoring the latter factor for simplicity:

$$P_{\text{bub}} = P_{\text{amb}} + 2st/R_{\text{bub}} \quad \text{Equation 1}$$

where st is surface tension and R_{bub} is bubble radius. P_{bub} exceeds ambient pressure for small bubbles but approaches ambient pressure for mechanically stable, large (e.g., ultrasonically detectable) bubbles. Therefore, for a bubble to form:

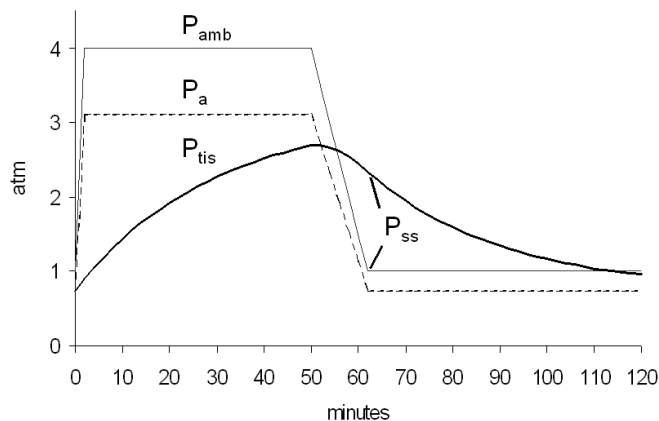
$$P_{\text{tis}} > P_{\text{amb}} + 2st/R_{\text{bub}} \quad \text{Equation 2}$$

or equally:

$$P_{\text{tis}} - P_{\text{amb}} = P_{\text{ss}} > 2st/R_{\text{bub}} \quad \text{Equation 3}$$

The extent of supersaturation determines the probability (or the rate) not only of bubble formation, but also bubble growth. If the partial pressure of gases inside a bubble exceeds the tissue gas tensions the bubble will shrink; conversely, bubbles of sufficient size can grow, acquiring gas by diffusion from adjacent, supersaturated tissue. The

Figure 1
Exponential approach of tissue gas pressure (P_{tis}) to arterial gas pressure (P_{a}) with changing ambient pressure (P_{amb}) during a 30 metres' sea water dive. The maximum supersaturation (P_{ss}) is indicated.



tissue metabolic gases' tensions and the bubble metabolic gases' partial pressures can be considered to be equal, so for a single inert gas, the conditions for bubble growth are also determined by Equation 3.

Gas-content models

Since supersaturation determines the probability of bubble formation and represents the tissue gas content available for bubble growth it has been used as an index of decompression stress. Deterministic content models prescribe a schedule's ascent rate and decompression stops according to ascent rules that limit supersaturation without directly calculating any bubble index. A widely used format for ascent rules is:

$$P_{\text{tis_inert}} < z \cdot P_{\text{amb}} + w \quad \text{Equation 4}$$

where $P_{\text{tis_inert}}$ is the tissue inert-gas tension and z and w are experimentally derived constants.⁶ Equation 4 can be solved for supersaturation ($P_{\text{ss}} = P_{\text{tis}} - P_{\text{amb}}$) and Figure 2 illustrates how increasing supersaturation is allowed at greater depths. However, Equation 4 is more useful in the form:

$$P_{\text{amb_tol}} = (P_{\text{tis_inert}} - w)/z \quad \text{Equation 5}$$

where $P_{\text{amb_tol}}$ is the minimum tolerated ambient pressure. To calculate decompression according to a content model, $P_{\text{tis_inert}}$ and $P_{\text{amb_tol}}$ are calculated for each compartment according to the preceding depth/time/breathing-gas history. Decompression stops may be required so that the P_{amb} is never lower than the maximum value of $P_{\text{amb_tol}}$.

An example bubble model

There are two general classes of bubble decompression models. Although there are overlapping aspects, one class focuses on the dynamics of gas transfer between bubbles and the surrounding tissue and is typified by the bubble volume model, which is central to current US Navy

Figure 2

Ascent rules expressed as allowed supersaturation in atmospheres absolute at different depths during decompression expressed in atmospheres gauge. In content models, different half-time compartments typically have a different rule (e.g., HT=5, HT=20). In bubble models allowed supersaturation is independent of depth (bubble).

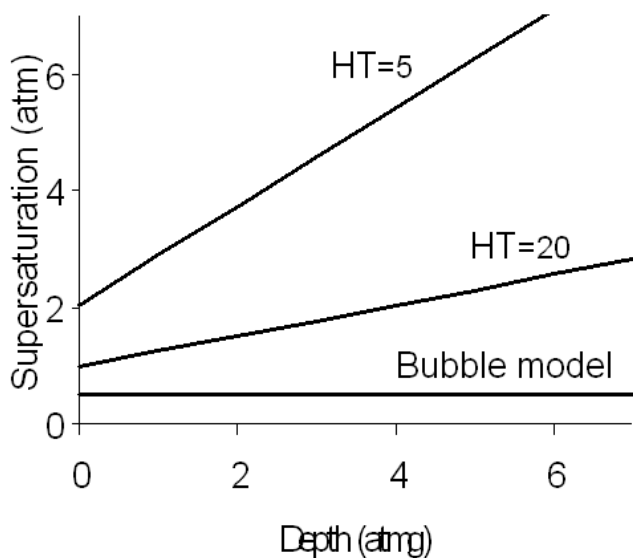
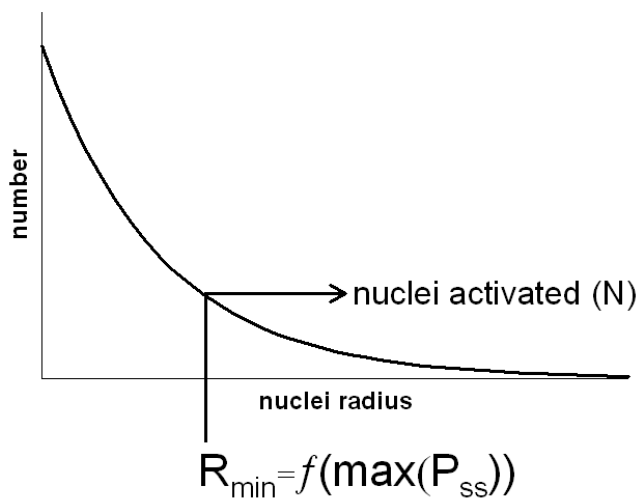


Figure 3

Exponential distribution of gas nuclei radii. The number of gas nuclei activated into growth as bubbles (N) is all nuclei of R_{min} and larger. R_{min} is a function of the maximum supersaturation (P_{ss}).



decompression research.⁷ The second class, to be outlined here, focuses on the number of bubbles that form during decompression and is typified by the variable permeability model derived from observations of bubble formation in gelatine, derivatives of which are seeing increasing use amongst technical divers.⁸

Equation 3 describes the inverse relationship between bubble size and the supersaturation required to form that bubble. Bubbles can form *de novo* from chance clusters of dissolved gas in physical systems supersaturated at more than 10.1 Mpa (100 bar). The extent of supersaturation required for bubble formation is not well defined *in vivo*, but bubbles form after relatively trivial decompression; for instance, in humans, bubbles are detected in the venous blood by ultrasonic Doppler shift after decompression from prolonged air breathing at 3.6 metres' sea water (137 kPa) to the surface (101 kPa).⁹ It seems more likely, therefore, that *in vivo* bubbles result from accumulation of gas into or around pre-existing gas nuclei (theoretical 'proto-bubbles'). One possible form of gas nucleus is a small bubble coated with surface-active agents that counteract surface tension, rendering the bubble relatively stable.

In the varying permeability model this surface-active coating makes available a population of stable gas nuclei some of which are sufficiently large that they can be activated into growing bubbles by supersaturation of the extent encountered in normal diving. In this model, the surface-active coating has the additional property of

maintaining the pressure inside the gas nuclei equal to P_{tis} . For any particular sized gas nucleus from the population before a dive, the supersaturation subsequently required for growth is described by an equation similar to Equation 3, except that the right-hand side has additional terms that account for the difference in opposing forces of surface tension and the surface active agents, and for compression of the gas nucleus during descent. Ignoring these additional terms for simplicity, Equation 3 can be rearranged to give:

$$R_{min} = 2st/P_{ss} \quad \text{Equation 6}$$

where R_{min} is the radius of smallest gas nucleus that will be activated by any particular level of supersaturation.

By assuming a negative exponential distribution of radii for the population of gas nuclei, and substituting Equation 6 into that exponential equation, the number of gas nuclei activated into growing bubbles can be calculated for a maximum supersaturation encountered during decompression (Figure 3). For completeness, but with no further explanation, the model name refers to the assumption that the surface-active coating becomes impermeable to gas diffusion, and therefore the behaviour of the gas nuclei changes with compression beyond approximately 912 kPa.

In the simplest form of the deterministic varying-permeability model, decompression can be controlled by a maximum-allowed number of bubbles and therefore a maximum-allowed supersaturation. Unlike in content models, this maximum-allowed supersaturation is constant throughout the decompression, as illustrated in Figure 2. Alternatively, decompression is controlled by a maximum-allowed index of bubble gas volume calculated by multiplying the excess number of bubbles (total number minus an always-safe number) by the integral of supersaturation and time, out to some long cut-off time

after decompression. Additionally, this gas volume index can be subject to expansion according to Boyle's law with decompression.

Decompression models in recreational diving

Most recreational diving is conducted according to the prescription of gas-content decompression models. The differences among various content models are the number (typically five to sixteen) of parallel compartments used, the range of half-times covered (1 to 1000 minutes), and the experimental data (if any) used to derive the ascent rules. Well-known decompression tables based on content models are the DSAT recreational dive planner, the US Navy 1957 standard air tables, and the DCIEM standard air tables.¹⁰⁻¹² The latter two differ in some specifics from the above description.

To the best of this author's knowledge, at the time of writing, all diver-carried electronic decompression computers (dive computers) use a real-time gas-content model. Many of these dive computer models are based on the ZH-L16 gas-content model, as is much of the user-controllable decompression software used by technical divers.¹³ Some newer dive computers have branding that implies a bubble decompression model. The models are proprietary information but, in fact, appear to be content models with user-controlled or dynamic modification of half-times, z or w parameters to result in longer decompression if the preceding dive history is notionally compatible with bubble formation (e.g., rapid ascent, repetitive diving). Some computers also prescribe, *ad hoc*, short decompression stops deeper than specified by the model. New computer models capable of real-time bubble-model calculations are likely to appear as processing power increases.

The use of bubble models by the recreational diving community to date is limited to the user-controlled decompression-planning software used primarily by technical divers. This software is based on the varying permeability model or a derivative called the reduced gradient bubble model.^{8,14}

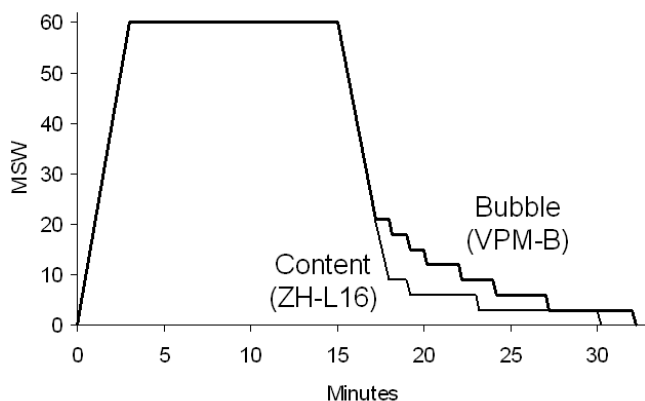
Bubble models typically prescribe deeper decompression stops than content models. Figure 4 illustrates the decompression schedule prescribed by the ZH-L16 content model and then how approximately the same amount of decompression time for the same dive is redistributed amongst a greater number of stops using a version of the varying permeability model (VPM-B). Theoretical analysis using a probabilistic bubble model suggests that such redistribution of decompression time to deeper stops can result in a lower risk of DCS (Gerth WA, personal communication, 2004) but this has yet to be objectively tested.

Conclusions

The previous paper in this series examined the functions that link calculated decompression stress to an observed outcome, either pDCS (probabilistic models) or usually adequate versus potentially inadequate decompression (deterministic models). The present paper provides an overview of how the measured depth/time/breathing-gas history of a dive is used to calculate decompression stress based on gas uptake and washout and bubble formation. This model component is the 'black box' between the measured dive history and outcome; none of gas uptake and washout, bubble formation and growth, or decompression stress is a measured value. These models are therefore useful only in so far as they can describe experimental decompression data and make predictions for dives similar to these experimental dives.

Figure 4

Comparison of decompression schedules following a 60 metres sea water dive prescribed by the ZH-L16 content model and by a variable permeability model derivative (VPM-B) with parameters set to provide an approximately equal duration of decompression. Note there appears to be no specific testing of the VPM-B model.



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- This paper is based on a talk given by Dr Doolette at the SPUMS Annual Scientific Meeting 2004 in Noumea.**

International Marine Contractors Association publishes 2003 safety statistics

The latest safety statistics published by the International Marine Contractors Association (IMCA) are based on figures supplied by 311 IMCA contractor members covering approximately 200 million hours worked overall around the world during 2003 (an increase of some 2% over the previous year). "Although only a lagging indicator of health, safety and environmental performance, safety statistics are nevertheless seen as providing a useful insight into the performance of a company in this area," explains IMCA's Chief Executive, Hugh Williams. "The purpose of the statistics is to record the safety performance of IMCA contractor members each year and to enable members to benchmark their performance. We also compare them with the figures published by organisations such as IADC (International Association of Drilling Contractors), OGP (International Association of Oil & Gas Producers) and IAGC (International Association of Geophysical Contractors)."

"We have seen an interesting development since the IMCA Safety, Environment & Legislation (SEL) Core Committee developed leading indicators (of health, safety and environmental performance), which can be promoted to clients and adopted by members, in order to get away from the high reliance on lagging indicators, for example lost-time injuries, as the arbiter of safety. Interestingly, the pleasing number of companies that supplied leading indicators for our 2003 survey would seem to have reaped the benefit of this commitment to safety, as their performance is generally better than the average."

Further information on leading performance indicators is available...in information note IMCA SEL 05/03. The 2003 statistics show that fatalities increased, with five reported in 2003 (one offshore), as opposed to three in 2002. Despite the various initiatives to improve safety, the offshore fatal accident rate (FAR) increased from 4.83 fatalities to 5.96 per 100,000,000 offshore working hours. There were 372 lost-time injuries reported (184 offshore) that resulted in at least one day off work.

"All participating members providing figures to the exercise reported their offshore data, where over 67 million hours were worked, compared with about 62 million hours in 2002," explains Hugh Williams. "The offshore lost-time injury frequency rate (LTIFR - Offshore LTIFR is calculated by multiplying the lost-time injuries offshore by a million and dividing by the number of offshore hours worked based on a 12-hour day) has continued to show an improvement over the last four years, from 4.25 in 2000 to 2.96 in 2002 and to 2.74 [in 2003]. This demonstrates that very definite benefits are being derived from safety initiatives."

"If we are to eliminate injuries, damage or near miss incidents, it is imperative that we focus on at-risk acts and unsafe conditions, which have not yet caused loss or harm but have the potential to," says Hugh Williams. "This is why safety guidance lies at the core of IMCA's work, and why we have published over 170 safety-related guidance notes."

Dangerous Australian box jellyfish

Peter J Fenner

Key words

Marine animals, jellyfish, envenomation, review article

Abstract

(Fenner PJ. Dangerous Australian box jellyfish. *SPUMS J.* 2005; 35: 76-83.)

Each year in the tropical waters of Australia, just south of the Tropic of Capricorn on the east coast and extending northwards and then westwards to Exmouth in Western Australia, serious envenomation can occur from two major species of box jellyfish. *Chironex fleckeri*, a multi-tentacled box jellyfish (chirodripid) has caused 68 fatal stings since 1883. Injuries vary from life-threatening stings affecting the conscious state, the breathing or the circulation of the victim, to smaller stings that cause severe local skin pain. *Carukia barnesi*, commonly known as the 'Irukandji', is a single-tentacled box jellyfish (carybdeid) causing stings that produce severe, rarely life-threatening systemic symptoms. The number of stings from this species varies considerably from year to year from a few to several hundred. However, in some years potentially life-threatening stings present, suggesting there may be two main 'groups' of Irukandji – the first, *Carukia barnesi*, causing the classical syndrome with its severe, unpleasant but non-life-threatening systemic symptoms; and the other, a different species or group, causing a severe syndrome responsible for two human fatalities in 2003. This article describes the symptoms caused by each species and the first-aid and medical treatment of the stings.

Introduction

Tropical Australian beaches and the adjacent waters including the Great Barrier Reef are amongst some of the best in the world for both scenery and opportunities for great recreational water activities, including reef diving and snorkelling for coral and fish viewing. However, in the hot summer months potentially lethal box jellyfish share these waters. These box jellyfish fall into two groups or genera:

- Chirodripids – box-shaped jellyfish with up to fifteen tentacles in each corner. The most common of these being *Chironex fleckeri*, which can cause immediate, severe, burning pain and depending on the extent of the sting may cause life-threatening respiratory or cardiac arrest within minutes.
- Carybdeids – box-shaped jellyfish with a single tentacle in each corner. There are many types of carybdeids throughout Australia, but in summer months it is the Irukandji group of jellyfish whose sting can cause severe systemic symptoms that completely prostrate their victim, and in some areas may cause life-threatening symptoms and even death. Species of Irukandji are present from coastal waters and deeper waters right out to and including the Great Barrier Reef.

Chironex fleckeri

Although the first death from a jellyfish sting in Australia was in 1883, the causative jellyfish was not discovered until 1955. It was then that Hugo Flecker, a radiologist in Cairns (Figure 1), caught several large, box-shaped jellyfish in the waters near Tully, North Queensland, where a young child died after being stung. Southcott confirmed that this

jellyfish was a new species and genus and in 1956 named it *Chironex fleckeri* (Figure 2) after Flecker.¹

There have been 68 deaths in Australia from *Chironex* since the first was described in 1883.¹⁻⁴ *Chironex* stings cause instant, severe skin pain, similar to being branded with burning irons. In large stings (more than 50% of the area of one limb) impaired consciousness, breathing and circulation may occur leading to death within three minutes.² The severity of sting depends on the area of envenomation (also depending on the width and the length of tentacles) and the intimacy of skin contact, which may be partially reduced due to the presence of hair. The latter may be a reason why relatively hairless victims such as children, who also have a greater surface-area-to-mass ratio, suffer more serious stings. Children account for 53 of the 68 deaths from *Chironex*.² In *Chironex* stings, tentacles avulsed from the animal by the struggling victim in the water adhere firmly to the skin, anchored by vast numbers of discharged nematocyst tubules buried in the victim's skin.

Tentacle contact causes severe, localised skin pain, with weal formation and blistering occurring within six hours (Figure 3). A 'ladder' pattern may be seen glistening in the sun on the stung area (Figure 4), caused by remaining discharged nematocysts on the skin surface.³ This ladder pattern matches the arrangement of 'batteries' of nematocysts on the jellyfish tentacle (Figure 5). Usually, in the next 24 hours the skin becomes necrotic (Figure 3) and tends to scar for life (Figure 6).

Ice or cold packs alleviate minor skin pain but they are of less benefit after major envenomation because of deep skin

Figure 1
Hugo Flecker
(courtesy John Pearn)



Figure 2
Chironex fleckeri, the box jellyfish

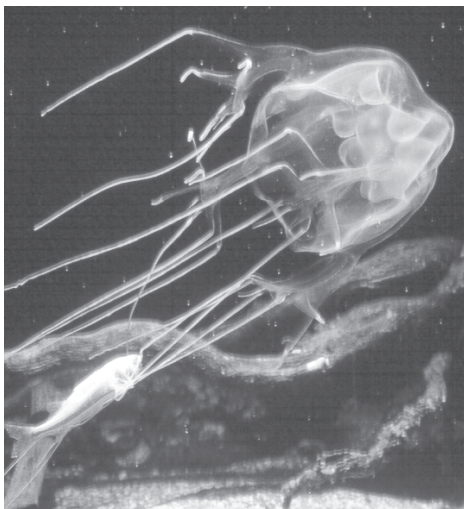


Figure 3
Chironex sting:
typical skin pattern



Figure 4
Chironex sting : typical skin 'ladder' pattern



Figure 5
The arrangement of 'batteries' of nematocysts on a
Chironex tentacle

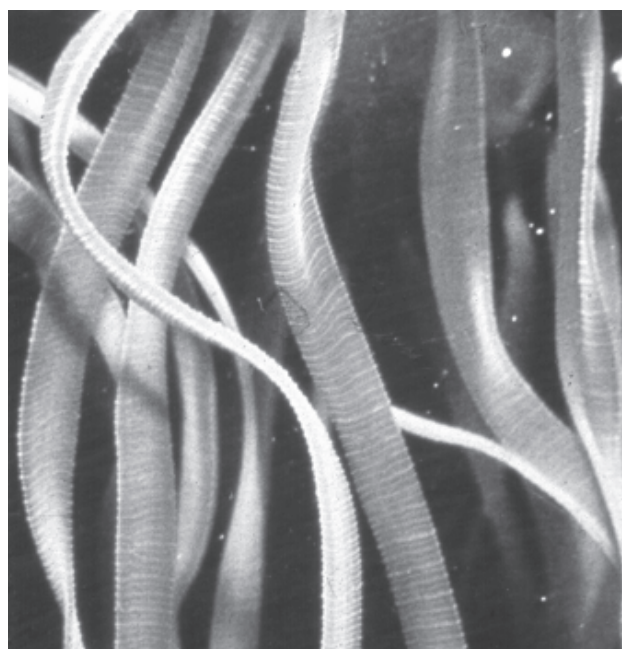
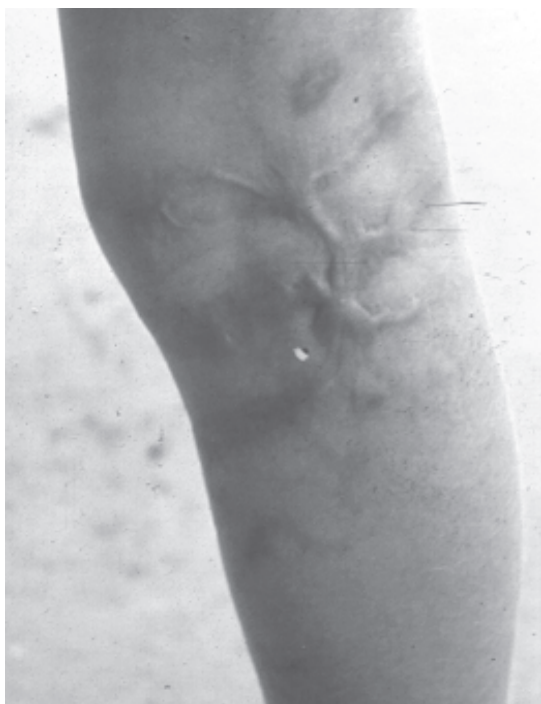


Figure 6
Scarring of the skin after a serious *Chironex* sting

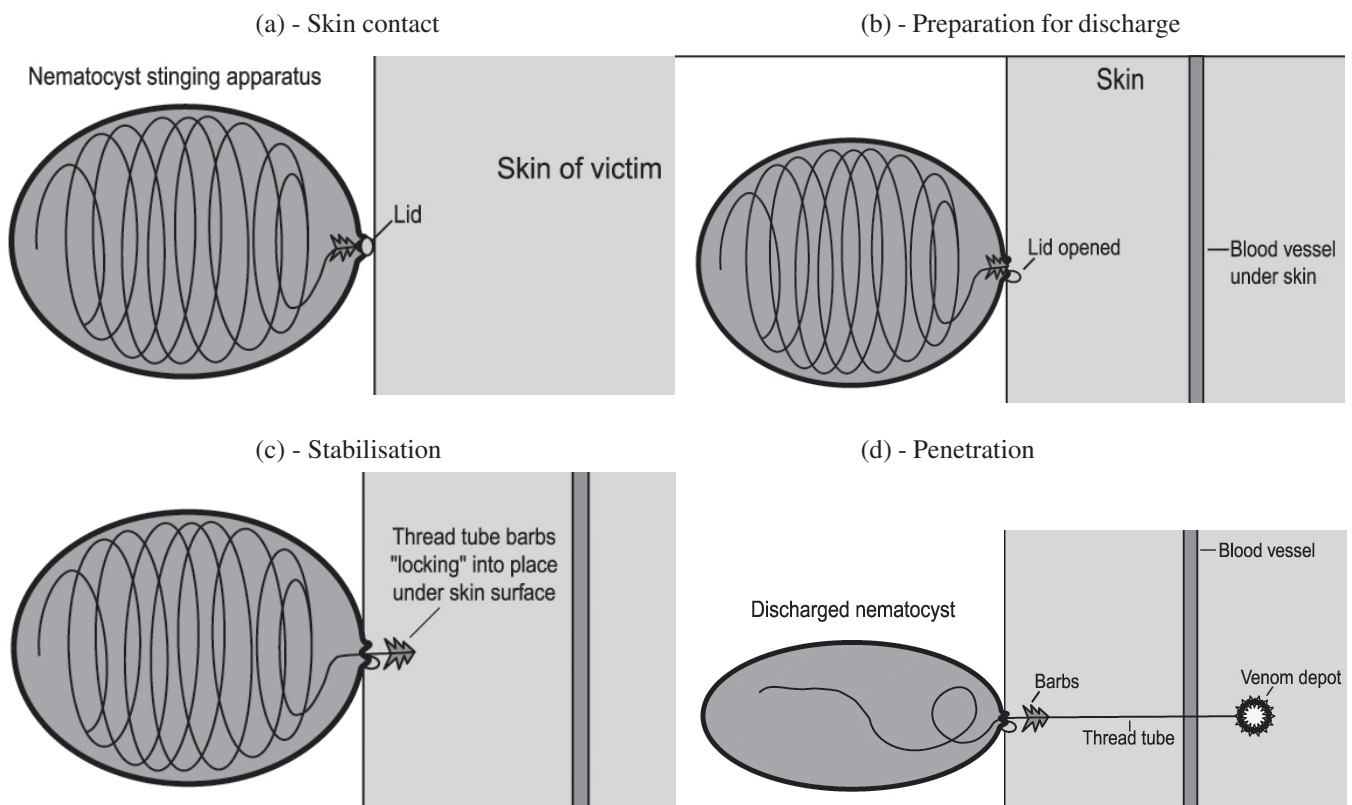


damage and severe pain. Although some victims are taken to hospital, only 1–2% are admitted (Fenner, unpublished data from sting database) with exact numbers of stings unknown and unobtainable.

Chironex envenomation is probably the most rapid envenomation process known, with unconsciousness, and respiratory or cardiac arrest occurring within minutes, often on the beach before appropriate first-aid treatments are commenced. The victim may be screaming with pain one moment but suddenly become quiet and 'co-operative' due to rapid unconsciousness where the need for resuscitation may be imminent.²⁻⁴

Method of jellyfish envenomation

Injection of the venom occurs from a specialised cell called the nematocyst. Millions of nematocysts are present in the tentacles of a jellyfish. Each nematocyst has a thread tube that is tightly coiled inside a capsule. On the outside of this nematocyst is a 'lid' with a 'trigger'. When this trigger touches the integument of a potential victim, it is stimulated by both the taste (chemical) and touch (tactile) of the potential prey ((a) - Skin contact). This causes the lid of the nematocyst to open ((b) - Preparation for discharge). The thread tube then everts itself in just a few thousandths of a second, driving through the skin of its victim with tremendous force and speed. The first part of the discharging thread tube has a set of 'spines' that 'lock' into the epidermis ((c) - Stabilisation). The thread tube extends into the dermis and venom from the inside of the cell then passes down the tube and is deposited into the dermis ((d) - Penetration). As the thread everts, venom is present on the outside of the thread tube and when this tube punctures microscopic blood vessels, venom is introduced directly into the blood stream, accounting for the rapid onset of symptoms in massive *Chironex* box jellyfish envenomation.



FIRST AID FOR *CHIRONEX* ENVENOMATION

- Retrieve the victim from the water and restrain if necessary (activity causes an increased heart rate and faster absorption of venom from muscle contraction).
- Send others for an ambulance and/or medical help.
- Assess the conscious state and treat airway, breathing and circulation.
- Pour vinegar liberally over the stung area for at least 30 seconds to inactivate remaining stinging cells of adherent tentacles on the skin.
- Use oxygen, if available, for severe envenomation; Entonox (50% nitrous oxide, 50% oxygen) for severe pain. Entonox is contra-indicated in scuba divers who have recently been diving.
- In Queensland, the ambulance service has *Chironex* antivenom available. If the victim is unconscious, or if there is evidence of life-threatening cardiac or

respiratory decompensation, three ampoules are given intramuscularly by ambulance officers, or one ampoule intravenously by paramedics.⁵

Vinegar

Pour vinegar liberally over the area of the sting for at least 30 seconds to stop further nematocyst discharge. Do not rub the stung area either before or after the application of vinegar. After vinegar use, it is unnecessary to waste time removing the tentacles. However, if vinegar is not available for a major sting, pick off the bulk of the adherent tentacles as quickly and as gently as possible without compromising resuscitation. The skin of the rescuer's fingers is too thick to permit substantial penetration by nematocysts although a faint prickling sensation may be felt on the fingers during handling of tentacles that have not been treated with vinegar. Wash hands carefully to remove these nematocysts,

and subsequently apply vinegar for at least 30 seconds at the earliest opportunity. There is currently no effective substitute for vinegar in inactivating unfired chirodroid nematocysts. However, if vinegar is not available Coca Cola™ has been shown to be of limited benefit as an alternative.⁶ Vinegar does NOT stop the skin pain.

MEDICAL MANAGEMENT FOR *CHIRONEX* ENVENOMATION

- If the victim is unconscious, or in life-threatening cardiac or respiratory decompensation, or demonstrating severe arrhythmias, antivenom can be given intravenously (the ampoule of 20,000 units is diluted 1:10). In a life-threatening situation three (or more) ampoules may be given intravenously and consecutively if the clinical response is inadequate.
- For persistent life-threatening cardiac decompensation or arrhythmia after antivenom use, consider giving verapamil intravenously (0.1 mg.kg^{-1} , up to 5 mg adult dose) while continuing cardiopulmonary resuscitation.⁷ Questions have been raised regarding the efficacy and safety of verapamil but in critical cases it should be considered.^{8,9} Inotropic support may be required for persisting hypotension, but calcium is contra-indicated.
- Cardiopulmonary resuscitation, employing oxygen-enriched air (preferably 100% oxygen) should continue until after consideration of further therapy with more antivenom (at least six ampoules total dose if available) and consideration of more verapamil and inotropes. Intermittent positive pressure ventilation will assist in the control of pulmonary oedema.
- In non-life-threatening stings involving cosmetically sensitive areas (e.g., face or neck, especially in females), consider giving one ampoule of intravenous antivenom (administered as above) for potential cosmetic benefits.

Pressure bandaging

First aid using the pressure-immobilisation bandaging technique (PIB) has been advocated for major *Chironex* box jellyfish stings.^{2,4} It was a technique originally developed to delay absorption of venom in snakebite.⁹ However, it has now been shown *in vitro* that pressure can cause further discharge of nematocysts remaining on the skin, even after vinegar application, and PIB has been condemned by some groups, who have suggested that it will cause further envenomation of the victim.¹¹ However, this has never been shown in clinical use in major stings where PIB was used after initial treatment with vinegar.^{3,5}

The principle behind the use of PIB in major *Chironex* envenomation is that after resuscitation and vinegar application, this technique would 'buy time', as it does in snake envenomation. This enables rescuers to get the victim to hospital for intravenous access, antivenom and advanced resuscitation facilities. Although the Australian Resuscitation Council does not now recommend PIB,¹² the

outcome of its use awaits controlled evaluation and it could well be useful in certain cases.

The Irukandji syndrome

In earlier days, the organism responsible for causing the delayed, severe systemic symptoms in swimmers in tropical waters in summer months in Australia was unknown. In 1952, Flecker named this set of severe systemic symptoms the 'Irukandji syndrome' after the Aboriginal tribe inhabiting the region around Palm Cove, north of Cairns, where many of the stings occurred.¹³

Figure 7
Carukia barnesi, the 'original' Irukandji

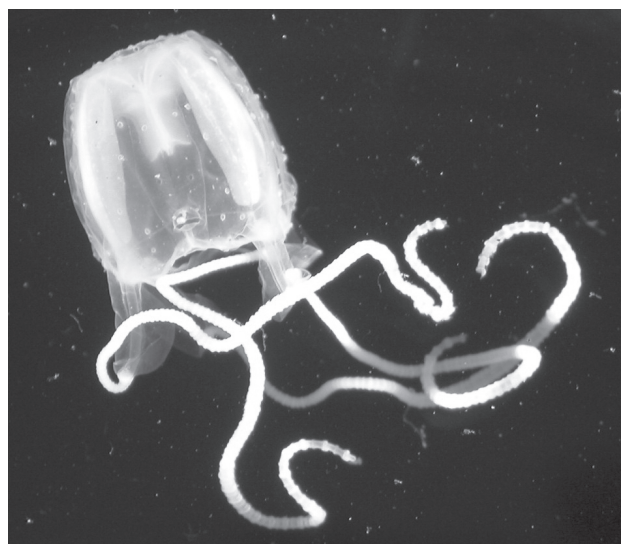
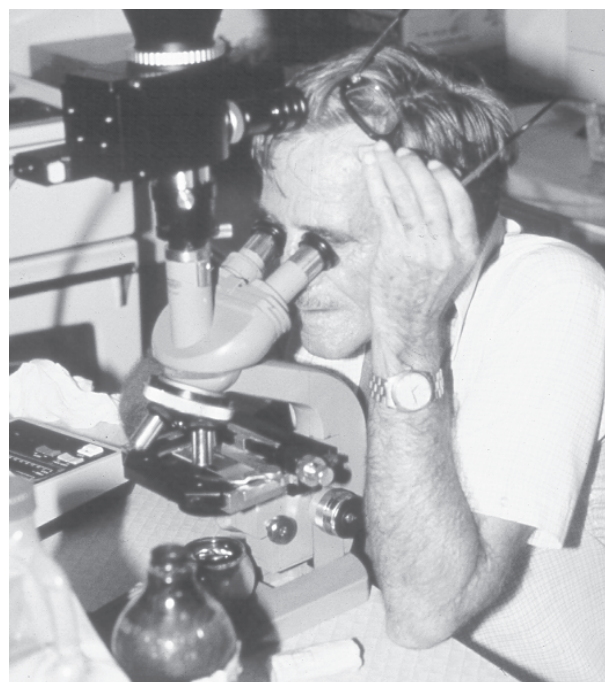


Figure 8
Jack Barnes



THE IRUKANDJI (*Carukia barnesi*)

Jack Barnes caught two small carybdeids (box jellyfish) in shallow waters off Palm Cove in 1963 after some very careful detective work.¹⁴ After stinging himself, his son and a friend, all of whom developed the full Irukandji syndrome, Barnes knew he had the true Irukandji. Southcott identified this jellyfish as an unknown species and genus and named it *Carukia barnesi* (Figure 7), after its intrepid discoverer (Figure 8).¹⁵ It remains the only jellyfish to date proven to cause this syndrome in tropical Australian waters. *Carukia barnesi* has a transparent bell 10–12 mm in diameter, making it almost impossible to see in the water.¹⁶ This small carybdeid jellyfish has one tentacle in each corner, each being 50–70 mm long when contracted, but extending to some 600–700 mm when the Irukandji is ‘trawling’ for its prey of small fish.²

IRUKANDJI SPECIES

The slang (colloquial) name of Irukandji was previously used only for *Carukia barnesi*; however, recent research now suggests the syndrome is caused by at least 10 small carybdeids similar to the original *C. barnesi* and at least two larger carybdeid jellyfish (approximately 60–70 mm bell diameter at maturity; personal communication, Gershwin L-A, James Cook University, 2005). Consequently, the name Irukandji is now used loosely to describe any jellyfish causing the Irukandji syndrome.

THE ‘CLASSICAL’ IRUKANDJI SYNDROME

Using original descriptions, the Irukandji syndrome can be defined as:

The severe systemic symptoms developing some 30 minutes (20–40 minutes) after a sting from a small box jellyfish.

The initial envenomation causes only minor skin pain with a faint red mark, which may be missed. Close inspection may reveal localised piloerection and sweating but these symptoms are usually totally missed. The initial envenomation is followed approximately half an hour later by a classical sequence of symptoms, including:

- Local pain
- Low back pain – severe boring pains in the sacral area.
- Muscle pain or ‘cramps’ in all limbs, the abdominal and chest-wall muscles. The pain is severe, unbearable and comes on in waves (similar to labour pains), never fading completely.
- Chest pain or tightness – spasm of the intercostal muscles (with no bronchospasm demonstrated); however, cardiac muscle pain and damage must be present, especially in more severe cases, as a rise in cardiac enzyme levels can be measured.

Catecholamine excess

Many of the signs and symptoms associated with the Irukandji syndrome resemble those of an adrenal medullary tumour (phaeochromocytoma), or a funnel-web spider or scorpion envenomation, with excessive release of catecholamines into the bloodstream.

- Sweating – may be localised or generalised. It may be localised to the site of the sting, or a totally unrelated small area on the body. Often, it is generalised, profuse and drenching.
- Piloerection – localised or generalised, at the original site of envenomation, or an area totally unrelated.
- Anxiety and ‘wretchedness’ – the victim is over-anxious, feels absolutely dreadful and often describes a feeling of impending doom.
- Restlessness – the victim does not keep still, moves continuously, trying unsuccessfully to get comfortable.
- Headache – severe frontal, or global and incapacitating.
- Nausea – often with severe, intractable vomiting.
- Increased respiratory rate – rapid ‘sighing’ respiration.
- Tremor – a fine tremor, or fasciculation of the small muscles of the limbs.
- Pallor, or peripheral cyanosis – due to intense peripheral vasoconstriction.
- Oliguria – from both reduced renal perfusion and fluid loss from the sweating and/or vomiting.
- Tachycardia – fast and occasionally irregular, with ventricular extra-systoles.

Barnes was a meticulous record keeper and rarely, if ever, noted hypertension other than that associated with the pain of envenomation. He never described any life-threatening symptoms, although he was suspicious that a diving death in Townsville was connected to Irukandji envenomation.¹⁷

In 1998, a report of 60 Irukandji stings from Cairns also showed that the Irukandji syndrome, although unpleasant, had no life-threatening systemic effects.¹⁸ The majority of patients were discharged after several hours’ monitoring in a specialised observation ward, without the need for admission.

SEVERE IRUKANDJI SYNDROME

In the Whitsunday region and on the outer Great Barrier Reef (GBR) symptoms have been more severe than those in the Cairns area, with severe hypertension and pulmonary oedema being reported.^{19–21} The 1998–99 season was also notable for the number of victims with severe toxic heart failure who needed admission to intensive care facilities for more complex treatment and investigations than usual. There have been other severe and unusual symptoms reported since, leading to the conclusion that there may be more than one species of jellyfish causing the Irukandji syndrome.^{22,23}

Systemic symptoms in severe cases

The skin pain seems to be sharper, and the severe and even bizarre systemic symptoms occur faster than in the classical syndrome, often starting some 5–20 minutes after the initial sting. The two recognised clinical sequelae of pain and catecholamine excess occur together with severe hypertension, which may reach levels as high as 300/180 mmHg in previously normotensive victims, and acute pulmonary oedema, which often develops some 15–18 hours post envenomation, occasionally less. Echocardiography shows marked global dilatation and left ventricular dysfunction, with a demonstrable rise in cardiac troponin levels, indicating an acute toxic myocarditis.^{21,22}

Rarely, some people develop burning, neurasthenic pain in the lower limbs or in the jaw, priapism and acute angioedema within minutes of the initial sting and often this is accompanied by an audible wheeze.²²

Skin scrapings to identify the species of Irukandji as 'classical' or 'severe' may be useful clinically.²⁴ However, it appears that the method described, using a scalpel blade, may cause marked scarring, and it may be better to revert to the suggested method by Currie et al of using sticky tape.²⁵

FATALITIES IN IRUKANDJI SYNDROME

Death from an Irukandji syndrome was first reported in 2002 in the Whitsunday Islands.²⁶ The victim quickly developed the severe Irukandji syndrome with marked hypertension within 15 minutes and, despite reaching medical aid, had an intra-cerebral haemorrhage, dying a few hours later from massive brain damage. A second death occurred in 2002 after an Irukandji jellyfish sting at the outer GBR, off Port Douglas. Again the victim suffered the severe syndrome with a blood pressure of 250/130 mmHg before having a major intra-cerebral bleed (personal communication, victim's partner).

The Irukandji syndrome has probably been responsible for other deaths in the past, mistakenly attributed to causes such as heart attack, cerebrovascular accidents, drowning or even diving deaths. Further severe, life-threatening Irukandji envenomation cases have been reported very recently from Western Australia.²⁷

DISTRIBUTION

The distribution of Irukandji stings has been confirmed to be from the Agnes Water area in Central Queensland, northwards around the north Australian coast, and then as far south as Broome in Western Australia. However, occasional cases of Irukandji syndrome have now been reported in Queensland as far south as Moore Park, Bundaberg. Although Irukandji stings often occur in deep water, including the offshore islands of North Queensland and the outer GBR, swarms may be brought to the surface at

coastal swimming beaches by underwater currents. Multiple stings may occur in summer months in these shallow waters.²²

Irukandji sting numbers and intensities vary considerably each year. In some seasons there are 100–200 reported cases, whereas in others there may be very few. This may reflect variations in the ecology of species of jellyfish causing the Irukandji syndrome, and/or any unknown environmental, geophysical and geographical factors influencing their ecology.

FIRST-AID TREATMENT FOR IRUKANDJI SYNDROME

There is currently no specific first-aid treatment for Irukandji stings. Recommendations are as follows.

- Remain with the victim, reassure them and encourage rest (muscle activity increases the heart rate and absorption and systemic dissipation of the venom).
- Douse the area with vinegar – although there are no visible, adherent tentacles, skin scrapings of the area show that, as with all jellyfish stings, there are remaining, unfired nematocysts. Vinegar prevents further discharge of nematocysts. It does not stop pain or inactivate the injected venom.
- Use sublingual nitrate spray, if available, to reduce the life-threatening hypertension in severe Irukandji syndrome.²⁸
- Transport the victim to hospital by ambulance. The ambulance carries Entonox, a mixture of 50% oxygen and 50% nitrous oxide, which gives some pain relief, but further treatment in hospital is necessary. Entonox is contra-indicated if the stings occurred during scuba diving because of the risk of inducing decompression sickness with nitrous oxide.

MEDICAL MANAGEMENT OF IRUKANDJI SYNDROME

There is no antivenom available, treatment being symptomatic.²² Intravenous magnesium sulphate has proved to be very effective in the treatment of the signs and symptoms of Irukandji envenomation, greatly reducing pain and hypertension, and reducing all effects of the syndrome.²⁹ It is given either as boluses of 10 to 20 mmol magnesium sulphate, or as an infusion after an initial bolus of 30 mmol. It has few, if any, side effects in this dose. Respiratory support may be required until the severe symptoms are ameliorated.

DIFFERENTIAL DIAGNOSIS

Myocardial infarction

Cases of Irukandji envenomation with chest pain, particularly if pulmonary oedema develops, have previously been misdiagnosed as acute myocardial infarction with developing heart failure.²⁰ This may be reinforced by a history of swimming (exertion), especially if the history of

a mild sting is not elicited, or is forgotten by the victim. The situation was further confused when only the basic creatinine phosphokinase (CK) measurement was available, before it could be split into muscle and heart fractions (CK-MB) and long before cardiac troponins could be measured. Now, raised levels of troponins and/or CK-MB are taken as a measure of cardiac damage, or potential damage.

Decompression sickness

The Irukandji syndrome in a diver can also resemble decompression sickness, and may present a difficult differential diagnostic problem.¹⁶ There have now been a number of cases around the GBR when the Diving Emergency Service (DES) has been contacted by phone when, a short time after surfacing, a diver suddenly develops severe low back and chest pain and trouble breathing, and is distressed and restless. A high index of suspicion and careful questioning are needed. A history of a minor sting, frequently on the back of the neck, when surfacing (a small mark, often difficult to see) and/or careful differentiation of the symptoms are necessary. All this questioning may be conducted over a radiotelephone from a dive boat on the reef to the DES phone base in Adelaide – no easy task.

VENOM RESEARCH

Venom from *Carukia barnesi* caught in the Cairns region has been analysed and proven to be a presynaptic neuronal sodium channel agonist, strongly stimulating noradrenaline release, causing many of the clinical features of the Irukandji syndrome.³⁰ However, it is essential that other Irukandji species are caught from both the Whitsunday area and northern GBR and their venom compared *in vivo* and *in vitro* to that of *C. barnesi*. With severe hypertension and pulmonary oedema exhibited by Irukandji victims in these areas, there is obviously a potentially lethal factor in the Whitsunday Irukandji venom and that from the GBR where the second victim was stung. If antivenom can be produced it must be manipulated so that it neutralises these lethal factors, as well as the unpleasant classical effects.

Conclusions

Public awareness of prevention and treatment of dangerous, severe, and occasionally fatal *Chironex fleckeri* and Irukandji stings in tropical Australian waters in the summer months is lacking.³¹ Sufficient funding is currently not available for the further research essential to develop public awareness and formulate preventive strategies. Development of effective first-aid treatment regimes for the severe systemic symptoms associated with Irukandji envenomation, including an effective antivenom against all species of Irukandji, is essential.

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Case report

Pulmonary barotrauma and arterial gas embolism from free-ascent training: case report

Sarah E Sharkey

Key words

Pulmonary barotrauma, cerebral arterial gas embolism, decompression illness, ascent, treatment, case reports

Abstract

(Sharkey SE. Pulmonary barotrauma and arterial gas embolism from free-ascent training: case report. *SPUMS J.* 2005; 35: 84-8.)

A 29-year-old military dive trainee suffered a pulmonary barotrauma and arterial gas embolism during free-ascent training. The diver presented acutely with a pneumothorax, subcutaneous emphysema, and evidence of cerebral and spinal involvement. Clinical reports of cases with this particular combination of conditions are rare. Some factors affecting incidence, pathology and outcome are discussed.

Introduction

In the military and civilian environments, ascent training is conducted in order to provide a means by which a diver or submariner can reach the surface in an emergency when alternative breathing equipment is not available. In addition, some elements of the military conduct free ascents during clandestine operations.

This incident involved a military diver who was undergoing free-ascent training at the Submarine Escape Training Facility (SETF) at *HMAS Stirling*. SETF is a six-floor building and contains a 20-metre fresh-water (mfw) tower built for the purpose of providing ascent training to submariners. The water column is well-lit and warm (approximately 34°C). The tower is fitted with recompression chambers (RCCs) at the base and surface of the water column. The RCCs and medical team are available at immediate notice during operations in the tower.

Case report

A 29-year-old, male member of the Australian Defence Force (ADF), who was a qualified but inexperienced self-contained underwater breathing apparatus (scuba) air diver, was undergoing free-ascent training at SETF. Diver X had completed a Navy scuba air diving course 10 years previously but had not dived extensively since. He considered himself to be inexperienced. He had a diving medical seven months prior to this training and met the ADF diving medical standards. In particular, inspiratory and expiratory chest X-ray (CXR) and pulmonary function tests (PFTs) were all within normal limits. During the assessment at this presentation the patient reported a previously undeclared history that he had suffered asthma and pneumonia as a pre-school child. He could not recall

any requirement for treatment. He suffers some seasonal hay fever but is free of other illness or injury. He was a smoker between the ages of 14 and 20 years.

Diver X had performed a shallow scuba air dive within seven hours of the incident (38 minutes' duration at four metres' depth).

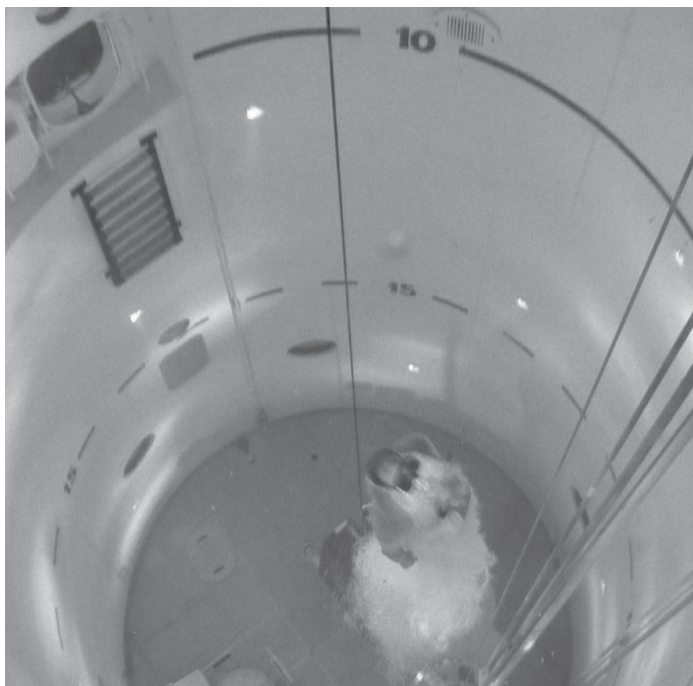
Free-ascent training was conducted wearing wetsuits and each diver was paired with a supervisor (Figure 1). The supervisor wore an open-circuit scuba air set from which both he and the diver breathed at depth. Ascent was conducted with the divers holding each other, ascending on a jackstay with the trainee's regulator out of his mouth but available. The diver was required to exhale continuously, maintain a posture that extends his airway and swim to the surface at a rate not exceeding 3 sec.m⁻¹.

Within minutes of successfully completing an ascent from 9 mfw and without exiting the water, a second ascent was conducted from 18 mfw. During the 18 mfw ascent Diver X was observed by the accompanying supervisor to be ascending too rapidly and was slowed down. He later described (and was observed from the surface by a diving supervisor) gulping during the ascent at approximately 5 mfw depth.

On reaching the surface, Diver X exited the water via a ladder and was immediately observed to be unable to declare himself well (Figure 2). Within seconds he collapsed and complained of difficulty breathing, generalised tingling and right-sided pleuritic chest pain. He remained conscious with a Glasgow coma scale score of 14–15, but became increasingly agitated and disorientated over several minutes. Initial assessment revealed a right-sided pneumothorax and rapidly progressive neurological

Figure 1

An unaccompanied supervisor demonstrating a free ascent from 18 metres' depth at the Submarine Escape Training Facility at HMAS Stirling

**Figure 2**

The exit ladder from the water tower with RCC at immediate notice. An instructor is providing a demonstration during Submarine Escape Training.



deficits in both lower limbs including hypertonia, spasms, weakness (graded 1–3/5 in both legs) and incoordination. A presumptive diagnosis of pulmonary barotrauma (Pbt) and cerebral arterial gas embolism (CAGE) with right-sided pneumothorax was made.

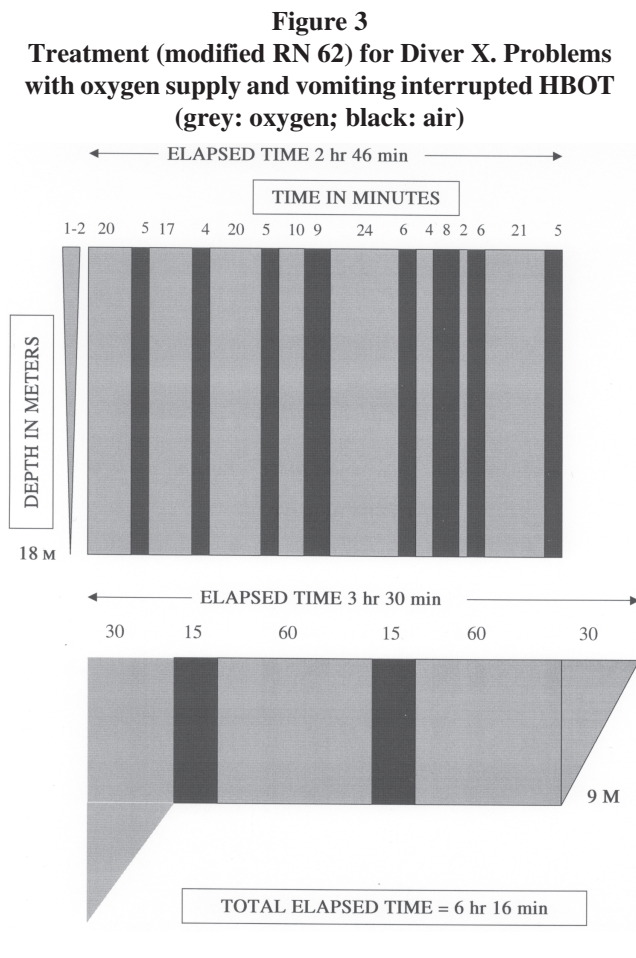
He was recompressed to 283 kPa within five minutes of collapse, and a Royal Navy treatment table 62 (US Navy 6) was commenced. On reaching 283 kPa and commencing 100% oxygen by mask, his dyspnoea improved and he became well-orientated but continued to complain of significant right-sided pleuritic chest pain and bilateral leg weakness, worse on the right side. Intravenous (IV) fluids were administered and he was aggressively hydrated. He remained haemodynamically normal throughout the treatment.

At 30 minutes into the treatment table, examination by the medical officer revealed persistent right-sided pneumothorax, subcutaneous emphysema over the right anterior chest wall from clavicle to nipple, and neurological deficits limited to the patient's lower limbs. Neurological examination revealed a hypertonic right leg with weakness graded at 1–2/5 in his right leg and 4–5/5 in his left leg. Sensory loss was patchy and limited to the lateral aspects of the right thigh and right leg. Knee jerks were hyper-reactive on both sides with absent ankle jerks and equivocal plantar reflexes. An intercostal catheter with underwater seal drainage was inserted to relieve the pneumothorax.

Delivery of oxygen during the recompression was both interrupted and delayed due to a combination of a built-in breathing system failure during the first two oxygen-breathing periods, and the diver's nausea, vomiting and anxiety during insertion of the chest tube. The treatment profile is detailed at Figure 3. It is estimated that at least 81 minutes of effective 100% oxygen was delivered at 283 kPa.

Prior to leaving 283 kPa, power, sensation and coordination had returned to normal and remained so at 202 kPa and on completion of the treatment table. He received no further recompression treatment on the basis that the risks of complications with a chest tube *in situ* were considered to outweigh any potential clinical benefit given his full neurological recovery on clinical examination. CXR the following morning confirmed small residual pneumothorax and subcutaneous emphysema that persisted for several days clinically. The intercostal catheter was removed uneventfully 48 hours after insertion.

Post-injury management included respiratory assessment with CXR, PFTs and high-resolution computerised tomography (CT) of the chest, all of which were repeated at two months post injury. The initial CT showed some residual surgical emphysema but was otherwise normal. PFTs performed at five days post incident and then at two months demonstrated progressive improvement in lung volumes, ventilatory capacity and transfer factor consistent with



resolution of the small residual pneumothorax, and excluded any on-going respiratory disease. CT was normal at two months. Review by a respiratory physician concluded that he had no respiratory disease or structural abnormalities. Within one week of the accident the diver was back running and has since been returned to full duties but with permanent exclusion from diving.

Discussion

PBt of ascent and CAGE are widely recognised potential complications of compressed-air diving and free-ascent training. The risks of their occurring during submarine escape training and other forms of ascent training have resulted in the discontinuation and/or significant modification of these activities.¹ Although a rare event, PBt with arterial gas embolism (AGE) is one of the more frequent causes of death in scuba diving and arguably may be the leading cause of death in the recreational diving community.¹ Mortality rates in the range of five to ten per cent have been reported even with the institution of appropriate treatment.^{1,2}

The two main contributing factors in the incidence of PBt are considered to be pulmonary pathology (e.g., asthma, adhesions and bullous disease) and inadequate technique resulting in air trapping during ascent. PBt and secondary

AGE occur more frequently in novice or inexperienced divers.³ However, most cases of PBt reported in the literature describe the use of apparently appropriate exhalation techniques and an absence of detectable lung pathology on post-injury medical screening.^{1,4}

Clinical evidence of pulmonary damage in cases of AGE is rare (3%). Sequelae of PBt include pulmonary tissue damage, pneumothorax, mediastinal and subcutaneous emphysema, and AGE. These may occur singly or in combination. The simultaneous occurrence of these conditions is reported as low.⁵ PBt is the result of pulmonary tissue being damaged by a transmural pressure change that causes over-stretching of the lung tissue.⁶ Pressure differentials of as little as 70 mmHg across lung tissue can cause tearing.⁷ Benton et al report a case of PBt and CAGE from a depth of 1m during helicopter escape training.⁸ Others have reported PBt and CAGE from 5 m and less.⁹

Pneumothorax associated with PBt occurs due to air entering the pleural cavity via either rupture of the visceral pleura or by air tracking through the mediastinum and via the parietal pleura. Subcutaneous emphysema results from gas tracking along tissue planes surrounding the airways and blood vessels, into the hilar regions and then into the mediastinum and neck. Subcutaneous emphysema is invariably associated with the existence of a pneumomediastinum.¹

The neurological signs in Diver X were unusual in that they could represent a combination of cerebral and spinal pathology or a cerebral or mid-brain lesion alone. The acute presentation and rapid deterioration precluded any more than a primary survey and brief neurological assessment prior to recompression of this patient.

Diver X completed a shallow no-decompression dive within seven hours of this ascent that was well within the guidelines of the Royal Australian Navy dive tables. The 'gas burden' from this dive profile, in addition to the surface interval, is small. It is generally reported that the 'submarine escape training presentation' type of PBt and AGE (amongst which this would be included given the lack of nitrogen tissue load and access to immediate recompression) does not produce a combination of decompression sickness (DCS) and AGE pathology.¹ In addition, spinal cord DCS is not thought to be principally embolic, since AGE is more likely to affect the more vascular grey matter of the cord rather than the white, and the effect of venous gas emboli could not account for the rapid presentation in cases like this.¹⁰

Neuman describes a syndrome of 'biphasic DCS' that occurs in the setting of an AGE and a trivial gas burden and may explain the presentation in this case.¹ It is suggested that AGE can precipitate DCS in divers who are well within the US Navy no-stop limits and who would not otherwise be expected to suffer DCS.¹ These cases are reported as severe and refractory to treatment.

MEDICAL SCREENING

PFTs are used in divers to detect pulmonary pathology and thus minimise exposure to risks such as PBt and AGE. An association between pre-morbid low forced vital capacity (FVC) and PBt has been reported in submarine escape trainees.¹¹

Consistent with Australian/New Zealand Standard 2299.1 and other diver medical screening recommendations, the ADF medical screening for diving requires spirometry to be performed.^{12,13} This is despite a lack of evidence that the incidence of PBt among divers has been reduced through this screening.¹⁴ Forced expiratory volume in one second (FEV₁) and FVC values within the predicted normal range do not exclude an individual from developing PBt.¹³ There is some limited evidence that mid-expiratory flow rates, derived from a single-breath flow-volume loop, may be more predictive of PBt.¹⁵ However, numbers in that study were small and definitive recommendations cannot be made without further research.¹⁶ In Diver X, simple spirometry, not a flow-volume loop, was conducted prior to diving. His post-morbid results, which include mid-expiratory flow rates, were all within normal limits.

Recommendations regarding diving with a history of asthma remain controversial due to the lack of controlled clinical data available. Current ADF policy is to exclude from diving members with evidence of demonstrable bronchial hyper-responsiveness. Although many experts consider that an absence of symptoms in the last five years is a reliable indicator of the absence of significant bronchial hyper-responsiveness, it has also been demonstrated that a proportion of individuals with demonstrable bronchial hyper-responsiveness do not have a clinical diagnosis of asthma.¹⁷ ADF diving candidates who declare a childhood history of asthma but have been asymptomatic since are generally referred for provocation testing.

TREATMENT

Immediate treatment of PBt occurring with AGE includes resuscitation, hyperbaric oxygen therapy (HBOT), intravenous fluids and consideration of adjunctive treatments such as lignocaine. It is widely accepted that the early application of HBOT plays an important role in the management and treatment of AGE and that delays to recompression significantly increase morbidity and mortality.¹⁸ Delays in recompression exceeding four hours reduce the chances of complete resolution by 50 per cent.¹⁹ Gorman concluded that if the delay to recompression following AGE is less than five minutes then the morbidity in survivors is almost zero although five percent may still die.²

Similar injuries occurring in the open-water environment are reported as having poorer outcomes and this is largely explained by the increased complications of near drowning, delays in recompression and resuscitation, and additional

nitrogen gas burden. The excellent outcome in this case can be attributed to the extremely controlled environment in which the training was being conducted and the immediate access to recompression facilities.

Conclusions

This case is a rare presentation of PBt and AGE. The following factors could be considered as potential contributors: gulping during the ascent (poor technique, relative inexperience and anxiety); microstructural pulmonary pathology; and possible bronchial hyper-responsiveness (childhood history of asthma). Mitigation of PBt/AGE in this context is addressed through the conduct of best practice and evidence-based medical screening, the prompt availability of trained medical support and recompression, and delivery of high-quality diver training. It cannot be concluded that additional medical screening would have helped to prevent this case and it is not possible to determine the cause precisely. The excellent clinical outcome is most likely attributable to immediate access to recompression facilities and medical care, and to favourable physiological characteristics of the patient.

Acknowledgments

The professionalism and skill of the medical team contributed importantly to the excellent patient outcome; specifically the team members were CPOMEDSM Benjamin Stock, CPL Matthew OShea, LSMEDU Toni Robinson and ABMEDU William Purcell.

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Divers Alert Network Project Dive Exploration

(edited from a news item in *Diver*, June 2005)

Liveboard diving, at just one case of decompression illness (DCI) per 10,000 dives, is providing the lowest incidence of DCI cases, according to a continuing research programme being carried out by Divers Alert Network (DAN). For whatever reason, the highest ratio has been for divers visiting Britain's Scapa Flow, at 30 DCI cases per 10,000 dives.

DAN, the international diving medical research and advisory body, is seeking the help of sport divers all over the world to research the causes of DCI. Rather than using scientific theory to work towards a diving decompression model, its Project Dive Exploration (PDE) aims simply to record which dive profiles worked without problem for divers and which did not. When enough data has been gathered, DAN hopes to produce tables and computer models which will give an idea of the DCI risks for different dive profiles, based on experience. Such a model would probably be regarded as complementing rather than replacing existing decompression programmes.

DAN developed its PDE data collection methodology and software from 1995-97, and began collecting data in 1999. So far, some 105,000 dives, whether safe or involving injury,

have been logged and analysed. It is from these preliminary figures that the Scapa and liveboard data are derived. DAN plans to obtain and work through more than a million responses before it begins to establish any sort of definitive model for divers to use. The scale of the programme makes PDE the "most extensive study of recreational diving ever conducted." For each dive they do, participating divers fill out a downloadable form, upload their dive computer's dive record (or otherwise record the profile if they used a table), and provide details of their physiology and health. A 48-hour post-dive report is included. Here the diver lists any medical signs and symptoms, or recompression treatment received.

If a chamber is involved, DAN will contact it for medical details, and find out whether flying after diving was involved. It has been estimated to take 30-60 minutes for a diver to send to DAN details relating to, say, 20 dives on a liveboard trip. DAN is conducting PDE "wherever diving occurs throughout the world." Divers can obtain relevant information on how to participate from dive centres, shops or aboard charter boats, or visit DAN's website, www.diversalertnetwork.org, or the South-East Asia Pacific offices at www.danseap.org. SPUMS members are encouraged to join the PDE programme themselves as well as encouraging divers they see in the course of their work to do the same.

The diving doctor's diary

Hooked on oxygen...hypochondriasis perhaps?

Sandy Inglis

Key words

Decompression illness, case reports, medical conditions and problems

Abstract

(Inglis S. Hooked on oxygen...hypochondriasis perhaps? *SPUMS J.* 2005; 35: 89-91.)

A young man presented to hospital on 15 occasions over eight months. Five of these were with presumed decompression illness (DCI) and for four he received recompression therapy. Each time, he presented with joint pains and non-specific symptoms, but never had objective neurological signs of DCI. His other presentations were for a wide range of complaints and multiple specialties were involved in his management. These presentations followed a characteristic pattern, always at night and mostly after midnight, and in most instances he was discharged without specific treatment, although he received normobaric or hyperbaric oxygen on almost every occasion. His diving-related presentations were initially plausible but on the last occasion he used an alias and, when challenged, discharged himself. What was driving this young man? The psychiatric differential diagnosis is wide, or was he just hooked on oxygen?

Introduction

Decompression illness (DCI) may present clinically in many forms, and previous case series and anecdotal reports have illustrated that some presenting divers do not have DCI.¹ This report presents the case of a young man with multiple presentations to hospital over a short period, some of which were for presumed DCI. The discussion will concentrate on the likely differential diagnosis.

Case report

A 19-year-old man presented to hospital on 15 occasions over eight months. Five of these were with presumed decompression illness (DCI) and on four he received recompression therapy. His other presentations were for a wide range of complaints including chest and abdominal pain, alleged trauma, collapse, nausea and even following a fictitious road traffic accident (Table 1). He underwent

multiple investigations, including blood and urine tests, chest X-rays and CT scans of the head and abdomen. Multiple specialties were involved in his management including Emergency and General Medicine, Hyperbaric Medicine, Psychiatry, Urology, Radiology, Neurology and even Neurosurgery. The presentations followed a characteristic pattern; always at night and mostly after midnight. At no stage did he have objective clinical or investigational findings. In most instances he was discharged without specific treatment, although he received normobaric or hyperbaric oxygen on almost every occasion.

DIVING-RELATED PRESENTATIONS

First presentation (second overall)

SS presented at 2022 hrs complaining of pain in his right knee and shoulder commencing about two hours following diving. He also felt dizzy, and had a headache and blurred vision. There was no chest pain, shortness of breath or skin itching. He was unemployed, but currently undertaking a Work and Income New Zealand-funded dive leader scuba course and this was his thirteenth dive. His only previous medical history was of bilateral inguinal hernia repair as a child. He was on no regular medications and had no allergies.

His first dive was reportedly to a maximum depth of 22.5 metres' sea water (msw) for 15 minutes. After a surface interval of 75 minutes, his second dive was to 20 msw for 15 minutes with an ascent over 2.5 minutes. He did not do a safety stop on either dive. These dives are within the no-stop times on the DCIEM decompression tables.

Table 1

Summary of 15 presentations, several related to scuba diving, by a 19-year-old male to the emergency department over an eight-month period

Attendance number	Presenting complaint	Diagnosis	Admissions to hospital
1, 6, 9, 11, 15	Alleged trauma	Uncertain	2
7, 8	Chest pain	Uncertain	2
12, 13	Abdominal pain	Uncertain	1
10	Nausea, shaking	Intoxicated	0
2, 3, 4, 5, 14	Joint pains, etc	?DCI	3

On examination he was alert and orientated, and had a strong body odour. Cardiovascular, respiratory, ENT and neurological examinations were all normal. Right shoulder and knee examination was also normal with no exacerbation of pain or limitation of range with movement. There was a patchy, erythematous rash on his chest.

A presumptive diagnosis of mild, stable musculoskeletal and neurological DCI was made. Initial investigations included a full blood count and biochemistry and a chest X-ray, all of which were normal.

Hyperbaric oxygen therapy (HBOT) comprised a Royal Navy treatment table 62 (RN 62), which produced moderate relief of his joint pains. The following day he received a further short HBOT (18.60.30 treatment table) and was discharged with minimal residual pain. That evening he presented to the emergency department with worsening shoulder pain, and received further HBOT the following day, with no further relief of his shoulder pain. He failed to present for review on the following day.

Second presentation (third overall)

Six weeks later he presented again, following what he said were his first dives since the previous admission, complaining of pain and paraesthesiae in his left shoulder, a headache and nausea. He had no chest pain, shortness of breath or itching. He reported that his symptoms had commenced after a hot shower, five hours after his last dive.

His first dive was a multi-level dive with a maximum depth of 33 msw for a total dive time of 45 minutes, including a safety stop of 10 minutes at 5 msw. Following a surface interval of 2.5 hours, his second dive was to 26 msw for a total dive time of 30 minutes, including a safety stop of 10 minutes at 5 msw. Three hours later he did a third dive to 18 msw for 25 minutes, including a safety stop of 10 minutes at 5 msw. His buddy apparently became caught in a net and SS claimed he had to cut him free. He had been somewhat anxious about this but insisted that he had still done his safety stop. His dive buddy did not accompany him to hospital and he did not know how to contact him.

He was alert and orientated, and clinical examination was normal, except that he reported subjective decreased sensation in his left hand. Chest X-ray was normal. The diagnosis was again presumed to be mild, stable musculoskeletal and neurological DCI following, this time, a provocative (alleged) dive profile.

He underwent an RN 62 and then an 18.60.30 HBOT on the following day. He admitted to some improvement following these but had ongoing paraesthesiae in his left hand. He was discharged from care but was readmitted to hospital the same evening complaining of sharp chest pain. A chest X-ray, electrocardiogram and blood tests were all normal. A further HBOT was given the following day, but he again failed to present for review subsequently.

Third presentation (fourteenth overall)

His third dive-related presentation was ten weeks later, when he complained of a throbbing headache, nausea, generalised muscle aches and pruritus. He had no chest pain or shortness of breath. He said he was 'unwell' following three scuba dives a day over a three-day period, but was extremely vague about his dive profiles and said that he had left his dive computer at home. His dive computer was not presented for analysis at any of his presentations, despite requests for him to do so.

On examination he was alert and orientated, and the rest of his examination was normal. At this point, although he had presented under an alias, he was recognised by another member of the emergency department staff as SS. The hyperbaric medicine doctor commented that he was "smelly and dirty, so highly unlikely to have been in the water yesterday". It transpired that SS had a Court appearance that day. No investigations were done, and he was referred urgently for psychiatric assessment but failed to present until eventually seen by the Drug and Alcohol Clinic a year later.

Discussion

The differential diagnosis in this young man is broad and remains elusive. It includes DCI, hypochondriasis, Munchausen's syndrome and factitious disorder. Other specific diagnoses include psychiatric disorders such as depression, anxiety disorder and malingering. Also, he may have had renal colic, been assaulted and even been involved in a road traffic accident, but there was never any substantiating evidence to confirm these. He had episodes of 'atypical' chest pain and may have been abusing drugs, especially alcohol.

It is still conceivable that the patient had DCI. It was confirmed that he was indeed doing a diving course at the time of his first dive-related presentation and he was precise about his diving profiles on this and the subsequent dive-related presentation. He only ever presented with symptoms, his clinical examination on each occasion being unremarkable. He appeared to only partially respond to HBOT, which was not consistent with his mild symptoms and early presentation. Following both these presentations he failed to present for review. His final dive-related presentation was a farce with him presenting under an alias with similar symptoms to before. It was never substantiated that he had actually been diving on any of these occasions. This is difficult to do, of course, without breaching patient confidentiality.

Hypochondriasis is defined as "*a preoccupation with bodily functions and fears of acquiring or having a serious disease based on misinterpretation of physical symptoms.*"² Hypochondriacs have no real illness, but are overly obsessed with normal bodily functions and become preoccupied with ideas or fears of having a serious illness. Appropriate medical

investigation and reassurance do not relieve these ideas. These ideas are not delusional and are not restricted to concern about appearance. Patients often seem highly invested in their own suffering. The person with hypochondriasis feels real distress, so the symptoms should not be denied or challenged by others. They cause distress that is clinically important or impairs work, social or personal functioning. Symptoms generally last six months or longer and this is a chronic illness that usually develops in middle age or later. Sufferers cannot shake the idea that something is seriously wrong with them and seek many tests and much reassurance from their doctor. Although some of these features describe SS, he did not appear overly obsessed about normal bodily functions and appeared reassured after his investigations or treatment.

Munchausen's syndrome is defined as "repeated fabrication of physical illness – usually acute, dramatic, and convincing – by a person who wanders from hospital to hospital for treatment."³ Patients with Munchausen's may simulate many physical disorders and may have prominent histrionic personality features but are usually intelligent and resourceful. They are sophisticated regarding medical practices and often have an early history of emotional and physical abuse. They have problems with their identity, intense feelings, inadequate impulse control, a deficient sense of reality, brief psychotic episodes, and unstable interpersonal relationships. 'Munchausen's by proxy' is seen in parents or care givers who present with a fabricated illness in their child. SS appeared neither intelligent nor sophisticated.

Patients with factitious disorder may consciously produce the manifestations of a disease and present themselves for medical care, but they sabotage therapy with self-induced or self-perpetuated disease. They tend to simulate only one disease and only during major psychosocial stress. They do not tend to wander from one hospital or physician to another and they can usually be treated successfully.³ SS simulated many diseases, did not appear to sabotage therapy and was not treated successfully.

Conclusions

SS had 15 presentations over eight months, was referred to no fewer than seven different specialties, some multiple times, and was admitted to the emergency observation ward five times and to a general medical ward twice. His triage code was invariably 'four' (to be seen within one hour) but

on two occasions it was 'two' (to be seen within 10 minutes).⁴ Presentations were predominantly after midnight (nine out of fifteen). He had extensive investigations, including CT scanning, which were all essentially normal, and apart from the first two diving-related presentations received no definitive treatments.

No diagnosis clearly fits with this young man's pattern of behaviour. It would seem that most of his symptoms were fabricated. He was a student on a diving course and certainly his diving symptomatology could have been learnt on that course. What was driving him will probably remain a mystery. What was definite was that he kept a lot of medical people busy and much time, effort and money was spent investigating and treating him. Finally, was he just hooked on oxygen?

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This case report was presented by Dr Inglis at the SPUMS ASM 2004 in Noumea.

East Somerset Research Consortium

Dr Peter Glanvill (Glanvill P, St Leger Dowse M, Bryson P. A longitudinal cohort study of UK divers with asthma: diving habits and asthma health issues. *SPUMS J.* 2005; 35: 18-22.) advises that he has joined a small general practice consortium in order to pursue further diving

medicine research in the primary health setting. The group meets regularly for brainstorming on a variety of topics.

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The world as it is

An estimate of the risk of fatal shark attack whilst diving in Western Australia

Peter Buzzacott

Key words

Marine animals, deaths, epidemiology

Abstract

(Buzzacott P. An estimate of the risk of fatal shark attack whilst diving in Western Australia. *SPUMS J.* 2005; 35: 92-4.) Many divers reassure themselves that they are more likely to perish by bee sting or lightning strike than by shark attack. Methods of estimating exposure to risk amongst scuba divers are described. The risk of a fatal shark attack whilst scuba diving in Western Australia is estimated to be less than one in three million dives.

Introduction

Though the absolute risk of a fatal shark attack whilst diving in Western Australia (WA) cannot be calculated with certainty, the likelihood is assured to be less than if there were twice as many attacks as reported, and the number of dives made in WA fewer than is conservatively estimated. By this method the likelihood can be given as "less than..." an estimate of the risk in the worst imaginable case.

Number of shark attacks

A review of the Western Australian newspapers indexed by Factiva found just one report of a diver being attacked by a shark during the last decade. The Taronga Park Zoo Australian Shark Attack File records one fatal attack on a scuba diver during the last ten years, and one on a diver using surface-supplied air (hookah) during the decade prior to that (personal communication, West J, 3 December 2004). In the interests of estimating the risk conservatively, we may consider it unlikely that there are more than two fatal shark attacks upon divers per decade in WA, and that only one is reported.

Number of divers

In 2000 the Australian Bureau of Statistics (ABS) conducted the Participation in Sport survey and found that 30,700 Western Australian divers had made dives during the previous year.¹ In 2002, however, the next ABS Participation in Sport survey found only 17,200 Western Australian divers had made dives during the previous year.² Differing methodology probably accounts for the estimated number of divers almost halving in two years. In 2000 people were asked, in four quarterly surveys, to choose sports in which they had participated from a list, whereas in 2002, during autumn only, they recalled sports from memory.

Methods of estimating numbers of dives

In the Orkney Islands dive-boat operators record the number of boat dives made, whilst shore dives are reported by local dive centres.³ With just two dive centres in the Orkneys it is easier to count dives there than it is in WA. At L'Esartit on the Mediterranean coast personal interviews were conducted at popular dive sites by researchers.⁴ This is a similar method to both a study conducted since 1996 in Japan, and to a study completed on Saba, a Caribbean island.^{5,6} With 10,000 km of coastline this method would be difficult to use in WA. Studies in the United Kingdom cited membership of the British Sub-Aqua Club and the Scottish Sub-Aqua Club when estimating exposure, though there are reservations about the completeness of this strategy.^{7,8} It is not known what proportion of divers in WA belong to dive clubs.

Possibly the most reliable method for determining exposure within a defined area is to count air-fills supplied. This method was used in the US military community on Okinawa Island, Japan, where treatment records of the hyperbaric chamber were compared with the number of air-fills recorded.⁹ A similar method was employed in the Northern Arabian Gulf to measure the frequency of decompression sickness within a population of commercial divers making scuba dives.¹⁰ In Victoria air-fills were used to determine the number of recreational dives made during one year, in a monthly telephone survey of 46 dive centres.¹¹ Likewise, air-fills supplied by dive centres were counted during a 14-month survey of dive centres in British Columbia.¹²

Research into the number of dives made by divers in WA alone has not been identified. In a survey of charter-boat divers in the Florida Keys, just 54% reported making fewer than 25 dives within the previous year.¹³ In Great Britain, a prospective survey of 100 asthmatic divers recorded 12,697 dives over five years, averaging just over 25 dives each per

year.¹⁴ Surveys of British Sub-Aqua Club members in 1986 and 1990 found mean annual diving rates of 22 and 33 dives per year respectively. In Geneva, a survey of 215 dive-club members found a mean of 28 dives made during the preceding six months, while a survey of 29 Australian dive clubs, four of which were in WA, found 346 divers averaged 39 dives per diver per year overall.^{15,16} This latter statistic is higher than found elsewhere and may reflect the high number of diving professionals within these clubs.

Diving exposure in Western Australia

The Recreational Diving Taskforce was appointed in WA in 1999, and tasked with analysing the accident and injury data for Western Australian scuba divers.¹⁷ The taskforce reported "...there is no mechanism to collate information on dive activity, e.g. tank fills, as part of a statewide database on recreational diving in Western Australia," (p.11) before conceding "It has not been possible to quantify nor qualify the degree of exposure to risk in the industry" (p.12). The aforementioned British Columbia study managed a participation rate of 65%.

Taking the estimate of 30,700 divers in 2000 as the most accurate to date, and allowing for an annual population growth of 1.4%, we might reasonably expect there to be 32,500 divers in WA today.¹⁸

Unless the mean number of dives made annually by Western Australians significantly differs from the populations surveyed in the above studies, then it is likely the average number of dives made in WA each year by Western Australians is no fewer than 15 to 20 per diver.¹³⁻¹⁶ If 32,500 divers make no fewer than 17 dives per year in WA then local divers account for at least 550,000 dives per year.

Between March 2000 and March 2003 there were, on average, 1.45 million visitors to Western Australia per year, not including interstate and day visitors.¹⁹ If the proportion of this visiting population who dive is approximately equal to the proportion of Western Australians who dive, which in 2001 was 31,100 divers out of 1,826,676 Western Australians within Australia on the night of the last census, then 25,000 of the visitors to WA each year made dives during the preceding year.

A survey of visitors to the southern half of the state between 1999 and 2002 found that, whilst on holiday in Australia/WA, 48% of international visitors had engaged in a sport or activity such as scuba diving, golf and/or fishing during their visit.²⁰ When domestic visitors were added the overall sports participation rate fell to 32%. If we accept that 25,000 visitors per year are divers and, on average, at least 32% will take either one single-dive or one double-dive trip during their stay, then a further 12,000 or more dives are made in WA each year by visiting divers.

At last count there were 50 dive centres operating in WA,

certifying more than 9,000 people each year and generating at least an additional 40,000 dives. Therefore, the estimated number of dives made in WA each year is no fewer than 550,000 by locals, 40,000 by trainee divers and staff, and at least 12,000 by visitors, totalling no fewer than 600,000 dives per year in WA.

Results

If the number of fatal shark attacks on divers in WA is estimated as above at less than 0.2 per year, and no fewer than 600,000 dives are estimated to be made in WA each year, then the risk of a fatal shark attack while diving in WA is less than one in three million dives (1:3,000,000).

Discussion

It cannot be known for certain how many divers are killed by sharks in WA each decade. It is possible, though unlikely, that fatal attacks remain unreported in either the media or the Australian Shark Attack Files. The 2001 census did not account for Western Australians who were overseas for the night of the census, so the population of WA may be slightly higher than the denominator used to estimate popularity of diving amongst residents. The number of international visitors to WA is calculated by surveying visitors at airports as they are leaving, missing international visitors travelling by other means. The number of interstate visitors is generated by telephone survey. The proportion of visitors who dive may differ significantly from the proportion of Western Australians who dive.

There were 8,900 Professional Association of Diving Instructors (PADI) certifications issued in WA during 2003 (personal communication, Oliver D, 31 January 2005). The total annual number of certifications is likely to be higher than this as other training providers certify divers in WA.

If a risk of shark attack as low as this fails to reassure, then nervous divers might consider the adage: if you dive with a buddy, your own risk of an attack feels immediately halved.

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Peter Buzzacott, BA, is a PADI Master Instructor. He is currently researching Western Australian scuba diving injuries at the School of Population Health, University of Western Australia, for a Master of Public Health Degree.

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Extract from Editorial, *Diver Magazine*, April 2005

I couldn't believe the figure when I first heard it. Unforgivably, I thought Pat Oates, the Secretary of the Scuba Industries Trade Association (SITA), must at least have added a stray nought, so that when she said "nearly 400,000", she actually meant "nearly 40,000". Even 40,000 seemed such a lot. But I should not have doubted her – when she said 400,000, she meant it.

I refer to the number of names on the petition which SITA, UK diving's trade body, launched in 2003 to test feeling on discriminatory airline baggage allowances. The latest figure is 409,925 and it's still growing. SITA has been helped in reaching this dizzy number in part by enlisting the help of PADI and gaining access to its membership lists...[*section deleted*]... SITA has had its critics, but three cheers to the diving trade for standing up and taking action. It is now contacting the airlines to seek real concessions for you, so if you haven't signed the petition already, visit <www.sita.org.uk> and download a form.

More than 400,000 names should be enough to light bulbs in the dimmest airline marketing exec's brain. That's a lot of bums on seats, with or without a few extra kilos of dive gear.

Steve Weinman

Editor's note: This is an international effort to allow concessions for diving equipment carried on international air flights to diving destinations. Many SPUMS members have been charged for excess baggage on flights to ASMs or on other diving trips. Readers are invited to support this effort.

SPUMS notices and news

South Pacific Underwater Medicine Society Diploma of Diving and Hyperbaric Medicine

Requirements for candidates

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

- 1 The candidate must be medically qualified, and be a financial member of the Society of at least two years' standing.
- 2 The candidate must supply evidence of satisfactory completion of an examined two-week full-time course in Diving and Hyperbaric Medicine at an approved Hyperbaric Medicine Unit.
- 3 The candidate must have completed the equivalent (as determined by the Education Officer) of at least six months' full-time clinical training in an approved Hyperbaric Medicine Unit.
- 4 The candidate must submit a written proposal for research in a relevant area of underwater or hyperbaric medicine, and in a standard format, for approval by the Academic Board before commencing their research project.
- 5 The candidate must produce, to the satisfaction of the Academic Board, a written report on the approved research project, in the form of a scientific paper suitable for publication.

Additional information

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

All research reports must clearly test a hypothesis. Original basic or clinical research is acceptable. Case series reports may be acceptable if thoroughly documented, subject to quantitative analysis, and the subject is extensively researched and discussed in detail. Reports of a single case are insufficient. Review articles may be acceptable if the world literature is thoroughly analysed and discussed, and the subject has not recently been similarly reviewed. Previously published material will not be considered.

It is expected that all research will be conducted in accordance with the joint NHMRC/AVCC statement and

guidelines on research practice (available at <http://www.health.gov.au/nhmrc/research/general/nhmrcavc.htm>) or the equivalent requirement of the country in which the research is conducted. All research involving humans or animals must be accompanied by documented evidence of approval by an appropriate research ethics committee. It is expected that the research project and the written report will be primarily the work of the candidate.

The Academic Board reserves the right to modify any of these requirements from time to time. The Academic Board consists of:

Dr Chris Acott, Education Officer, Professor Des Gorman and Associate Professor Mike Davis.

All enquiries should be addressed to the Education Officer:

Dr Chris Acott,
30 Park Avenue
Rosslyn Park
South Australia 5072
Australia

E-mail: <cacott@optusnet.com.au>

Key words

Qualifications, underwater medicine, hyperbaric oxygen, research

Minutes of the SPUMS Committee Meeting held in Sydney on 30 October 2004

Opened: 1020 hr

Present: Drs R Walker (President), C Meehan (Secretary), M Davis (Editor), A Patterson (Treasurer), D Walker (Committee Member), D Smart (ANZHMG Representative)

Apologies: Drs G Williams (Immediate Past-President), C Acott (Education Officer), S Mitchell, C Lee (Committee Members)

1 Minutes of the previous meeting (May/June 2004)

Moved that the minutes be accepted as a true record, after minor corrections.

Proposed Dr M Davis, seconded Dr A Patterson, carried.

2 Matters arising from the minutes

2.1 Improving our Internet cost effectiveness

Dr R Walker gave an update on the new SPUMS website being developed by SQUIZ Pty Ltd. Dr Walker will attend a training session in November. One other committee member may need to do the same.

2.2 There was some discussion regarding the SPUMS Journal CD and ways to improve it. Giving the CD to new members as part of a joining package was also discussed. This would require an increase in membership

fee and this would have to be proposed at the AGM by the Treasurer. The earliest this could be effective would be 2006.

2.3 It is planned to publish the constitution and to put the diving doctors list on the website. The constitution is to be scanned and retyped.

3 Update from the Editor, Dr M Davis

A journal report was presented.

Sarah Webb has officially resigned from the *New Zealand Medical Journal*. Dr Mike Davis has suggested that SPUMS withdraws from that office. Dr Davis will continue the SPUMS editorial work on the computer at the HBU. Dr Davis sought permission from the Committee to form an editorial board and noted that this may have a financial cost. The Treasurer, Dr A Patterson suggested that Dr Davis comes back to the Committee with a proposal outlining costs. Dr Davis would also like to increase the Diving Doctor's Diary section in the Journal.

There are still outstanding papers for the last SPUMS ASM in Noumea. Dr R Walker will write to the speakers. There will be a template for speakers outlining the requirements for the meeting.

4 Update from the Education Officer, Dr C Acott

This was given by Dr R Walker.

Dr Meehan will see if Dr J Knight has any of the original Mac back-up files. There are some data that need to be verified.

5 Annual Scientific Meetings

5.1 2004 ASM Noumea final figures

There was possibly a small loss incurred at the last ASM. Final figures are not yet available.

5.2 2005 ASM, Coco Palm Resort and Spa, Maldives update

As the island seems to have adequate oxygen delivery equipment readily available (and which is well maintained) SPUMS will not need to take our own equipment to the Maldives. There was discussion about the need to upgrade the SPUMS projector. The upgrade will be delayed for now, as a back up is available at the resort. There is no requirement to upgrade the SPUMS computer for this ASM.

There have been several changes in the quotes provided by Allways, partly as a result of exchange-rate variations with the US dollar, and also as a result of changes in SPUMS requirements. Clarification of the changes from the original quote provided in December 2003 has been requested. There is an ongoing need to improve the transparency of the process of managing the ASM.

There was discussion regarding the practice of Allways Dive Expeditions to include a list of participants and their full contact details with the travel documents. A list of participants can be included with the travel documents but contact details should be used only with the attendee's permission.

There was discussion regarding on-line registration for future conferences. There was also a discussion regarding graphing the ASM attendance rate to help with future planning. Dr Meehan will ask Steve Goble to provide the data for this.

There was also some discussion with regard to exchange-rate variations. It was suggested that, in future, costs linked to a different currency could be quoted in that currency. This would solve the problem of having to adjust the quote if the exchange rate changed.

5.3 Venues for future ASMs to be discussed

The Marshall Islands have been recommended, as has been returning to PNG. Mabul Island, Sabah, was also discussed again, although the safety of this region is still in question. Taveuni, Fiji, was also mentioned as a possible future venue. Due to the reducing numbers attending the conference alternative options for the ASM format will be explored.

6 Treasurer's report

There is a substantial cost involved in upgrading the SPUMS website. In view of this there may need to be an increase in the SPUMS membership fees. There was discussion regarding an additional joining fee to cover the cost of the proposed SPUMS Journal CD.

7 Correspondence - Nil

8 Other business

8.1 Report from Dr D Smart regarding Australian Standards Committee 017

8.2 Report from Dr C Meehan regarding the Queensland Industry Code of Practice

8.3 There is to be a certificate signed by the President and the Education Officer to accompany the SPUMS HTNA prize.

8.4 Review of the Editor's contract

The contract needs updating. At present the remuneration increases annually according to the CPI. The total remuneration package needs to be discussed at the face-to-face meeting held at the end of every year and adjustments made as deemed appropriate by the Committee. If more hours are required, there needs to be a formal business proposal presented to the Committee.

8.5 Discussion regarding committee positions which will be up for nomination at the next AGM. Notice of the AGM and a call for motions will need to go into the December 2004 journal, due to early timing of the AGM. The positions of Editor, Education Officer, Public Officer and the ANZHMG Representative are not elected. The Treasurer was elected at the last AGM and so has two further years to serve. There will be a call for nominations at the 2005 AGM for the President, Secretary and three committee members. Dr M Davis will print the nomination forms.

8.6 Dr D Smart will format a manual of corporate memory to assist the new members of the Committee.

Closed: 1730 hr approximately

Minutes of the Annual General Meeting of SPUMS held at Coco Palm Resort and Spa, Baa Atoll, Maldives, on Wednesday 27 April 2005

Opened: 1820 hr

Present: All members attending the Annual Scientific Meeting

Apologies: Graham McGeoch, Guy Williams

1 Minutes of the previous meeting

Unratified minutes of the previous meeting will be posted on the meeting notice board and appeared in the SPUMS Journal 2004; 34: 159-64.

Motion that the minutes be taken as read and is an accurate record.

Proposed Dr M Davis, seconded Dr V Haller, carried.

2 Matters arising from the minutes

Nil

3 Annual reports

3.1 President's report

3.2 Past-Presidents Committee report

3.3 Secretary's report

3.4 Education Officer's report

4 Annual financial statement and Treasurer's report

These were read by Dr R Walker.

Motion that the financial statements be accepted and the subscription fees will remain the same.

Proposed Dr S Paton, seconded Dr V Haller, carried.

5 Election of office bearers

The following nominations were received:

President: Dr C Acott

Secretary: Dr S Sharkey

Committee Members: Drs C Lee, D Vote, G Williams

Proposed Dr R Walker, seconded Dr M Davis, carried.

6 Appointment of the Auditor: Mr David Porter

Proposed Dr V Haller, seconded Dr C Lee, carried.

7 Business of which notice has been given

Nil

Closed: 1838 hr

President's report 2005

It seems that no time has passed since we last convened in Noumea. At last we have been blessed with fantastic weather, a fabulous resort and great diving. It seems the Society has been troubled by natural disasters (a volcano eruption soon

after we left Rabaul, the earthquake and tsunami of 26 December 2004) and terrorist events (9/11 and in the region of South-East Asia more generally). It has become increasingly difficult to find a safe venue and attract the large numbers to our conferences that we did in the 1990s.

However, one of the aims of the Society is to promote and facilitate the study of all aspects of underwater and hyperbaric medicine and the Committee is determined to persevere. We must take heed of the decreasing numbers attending the ASM and look at the format and content as well as the destinations (and make it financially affordable). Any suggestions in this regard are welcomed.

A final decision for next year's ASM has not been made, but Fiji is once again the most likely choice. A decision will be made shortly.

As foreshadowed last year, the SPUMS website has undergone a complete overhaul. Whilst delays (mostly on my part) have prevented the completion of this project, I expect we will go live shortly after our return from the Maldives. This will allow payment of fees via the Internet, group e-mail, members-only pages, etc. I believe this will make the face of our Society more professional.

I would expect to provide details of next year's ASM via this means, so if members wish to be kept up to date please ensure we have your current e-mail address.

The Journal, which is the backbone of the Society, continues to develop in leaps and bounds under Mike Davis's stewardship. I believe the Journal fulfils a unique role as a practical, relevant and dedicated diving medicine journal that other journals in this field do not match. I encourage all members to consider publishing in the Journal in preference to other more generalised publications.

The Society remains in a prosperous financial position, which has further improved with the efforts of our Treasurer Andrew Patterson (who is, I might say, very ably assisted by his wife Jenny).

Our long-standing Secretary of 12 years (Cathy Meehan) is standing down from the Committee this year. Cathy has given many hours of her time over the years to the Society and her corporate memory will be sorely missed. Cathy has also convened this year's meeting, which I am sure you will agree has been an outstanding success. Thank you, Cathy.

Simon Mitchell and Douglas Walker have also not sought renomination this year on the Committee and will step down. I thank them for their thoughtful contributions.

The Committee will continue to focus on membership and will be looking at additional Society membership categories for consideration at the next Annual General Meeting. This may include a retired member's category.

I acknowledge that there is a lot more that the SPUMS Committee could achieve but as we are a voluntary organisation and as most of the Committee hold down full-time positions we can never achieve all we would like. I have been President for six years and this is too long. New ideas, and fresh enthusiasm and direction are needed to ensure the Society remains relevant and at the forefront of diving medicine. The last six years have been characterised by many ups and downs but I hope that overall the Society has progressed. I have had much support from the Society and have made many friends. I will continue to participate in the Society but am pleased to hand over the reins to Chris Acott. I wish him well.

Robyn Walker

Secretary's report 2005

It is with great sadness but even greater happiness that I am making this, my last Secretary's report. I attended my first SPUMS conference in 1992. It was held in Port Douglas, in the far north of Queensland, and as I lived in Cairns, less than an hour away by road, it was a very easy and non-threatening start. Or so I thought!

It was at this, my first SPUMS conference, that John Williamson and Des Gorman approached me and asked if I would be willing to consider accepting a nomination to the position of Secretary of SPUMS at the next AGM. Not quite knowing what this involved, and feeling very honoured, I said "Yes". However, after spending the week in a twin share with Dr Sue Paton, who was then the Treasurer, and seeing the piles of paperwork she had brought with her and the intensity of her commitment, I wondered if there was any way of retracting my yes and getting out of it.

In 1993 at the AGM in Palau, I became Secretary and have been so for the last 12 years. Although it has been a very interesting and fruitful experience, I am sure that many will agree with me that I am more than ready to pass on the task to a younger and fresher candidate who can bring new enthusiasm to the position.

The past 12 years have flown past through a kaleidoscope of ASM venues and events, Palau 1993, Rabaul PNG (followed by the volcano eruption), Fiji, The Maldives, New Zealand, Palau again, Layang Layang (followed by the kidnappings by Philippines rebels of the divers on Sipadan), then the millennium ASM in Fiji followed by the coup, Madang in PNG, Vanuatu, Palau again, New Caledonia, and now The Maldives again, preceded sadly by the tragic tsunami. It becomes difficult to find an ASM venue that is safe from political unrest or risk of natural disaster. There was a time when our President was reluctant to take us to PNG, considering it unsafe and remote, but now even PNG is looking more and more like one of our safest and easiest destinations.

When first taking over the reins as Secretary back in 1992, all communication was done by facsimile and telephone. I spent hours feeding sheets of paper through the fax machine repeating each transmission nine times for the nine other committee members. What joy it was when we purchased the latest-model facsimile, which had a memory and automatically stored and then dialled all numbers on your list. Soon I heard of this new form of communication called e-mail, and encouraged the Committee to experiment with this exciting way of communication. For many years the Committee communicated half by e-mail and half by fax. Eventually, when all embraced this new modality, communication became a cinch.

Somewhere around this time I was also Secretary of our local dive club. Our first venture into cyberspace was putting some information about SPUMS on the Secretary's page of the Nautilus Dive Club website. Our own SPUMS site was developed a few years later, and we have been using this site since. After embarking early on an exciting voyage we then slowed down. We have been aware for a few years now that our website is behind the times and so it is with great pleasure that we look forward to launching the new SPUMS website with its increased functionality and dependability. We are hoping that this will encourage growth in our membership and greatly improve our service to our members.

So without wasting any more of your time, I would like to thank the past and present members of the Committee for their hard work, and for putting up with me for the last 12 years. In particular, I would like to thank Robyn Walker, the exiting President, who has successfully steered SPUMS through some difficult periods. I would also like to thank all the members of SPUMS for their involvement with the Society, and particularly all the past and present attendees at the SPUMS Annual Scientific Meetings for making the meetings so interesting and enjoyable.

I look forward to attending a SPUMS meeting as a totally free agent, although wonder what I will do with all that spare time. Well, the bar sounds good, as does walking on the beach in the moonlight.

Cathy Meehan

Treasurer's report 2005

As can be seen from the Financial Statements for the 2004 financial year, the Society is once again in a strong financial position.

While the deficiency for the year of some \$13,000 is disappointing, it is largely accounted for by the cost of setting up the Society's new website and by the writing off of \$5,889, a sum that has been carried as Accounts receivable since 2003. The history of this item remains unclear. It was first presented in the Financial Statements for the year

ending December 2003 and is thought to be outstanding membership subscriptions. Unfortunately, records are insufficient to provide a clear picture. Mr. Porter, the Society's auditor, has advised that the sum should be written off.

For the first time the funds remaining in a New Zealand bank account (BNZ Achiever Savings) are being recorded in the Financial Statements. This money derives from the residue of the account held by a separate NZ Chapter, now defunct, of the Society. I have elected to maintain this account because of the convenience it offers in supporting the work of the Editor of the SPUMS Journal. Although no large balance is kept in it at any time, I believe it is important to account for it in order to maintain proper standards of governance.

I am pleased to be able to report that after taking advice from an unpaid expert, who spent considerable time researching records of credit card transactions (such as

annual subscriptions and ASM registrations) reaching back over four years to the commencement of GST, the Australian Taxation Office returned over \$4,000 in GST owing to the Society.

Because a number of refunds were made to members following the 2004 ASM, the final overall costs for that meeting exceeded the income. I do not expect the 2005 ASM to improve on that situation. There will be further planned expenditure on the new website. However, I anticipate that the fully functional website will reduce the overall costs of administering the Society. Therefore, I recommend that subscription rates remain unchanged for the coming year, 2006.

Andrew Patterson

**THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
BALANCE SHEET
AS AT 31 DECEMBER 2004**

	2004	2003
MEMBERS' FUNDS		
Balance at 1 January 2004	133,237	114,856
Funds provided by NZ Chapter	4,384	-
Surplus/(Deficiency) for year	<u>(13,134)</u>	<u>18,381</u>
	<u>\$124,487</u>	<u>\$133,237</u>
represented by:		
NON-CURRENT ASSETS		
SPUMS website	20,000	-
Less, provision for amortization	(6,667)	-
	<u>13,333</u>	-
CURRENT ASSETS		
ANZ Bank ASM Account	-	6,447
ANZ Access Cheque Account	46	13,632
ANZ VZ Plus	100,590	96,400
ANZ SPUMS Annual Scientific Meeting	6,949	-
BNZ Achiever Savings	2,307	-
Accounts receivable	-	5,889
Prepaid 2004 ASM expenses	-	10,333
GST recoverable	1,262	536
	<u>111,154</u>	<u>133,237</u>
NET ASSETS	<u>\$124,487</u>	<u>\$133,237</u>

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale, NSW 2103
Dated: 17 April 2005

**THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY STATEMENT OF INCOME AND
EXPENDITURE FOR THE YEAR ENDED 31 DECEMBER 2004**

	2004	2003
INCOME		
Subscriptions and registrations	91,799	100,264
Interest	4,273	3,579
Advertising and Journal sales	323	316
ASM 2003	37,275	-
ASM 2004	37,370	-
ASM 2005	3,100	-
Sundry income	807	1,102
	<u>137,672</u>	<u>142,536</u>
EXPENSES		
Amortization of website	6,667	-
ASM costs	36,110	39,406
ASM registrations previous year	10,333	-
Bad debt written off	5,889	1,456
Secretarial wages	13,868	14,527
Stationery, printing, postage	1,332	2,122
Journal	37,907	27,645
Committee expenses	5,509	5,766
Computer equipment	2,778	747
Mail forwarding	456	-
Miscellaneous/Subscriptions	231	386
Bank charges	4,415	4,799
Audit	2,000	1,800
Editor's honorarium	17,350	19,335
Insurance	<u>5,961</u>	<u>6,166</u>
	<u>150,806</u>	<u>124,155</u>
SURPLUS/(DEFICIENCY) FOR YEAR	<u>\$(13,134)</u>	<u>\$18,381</u>

These are the accounts referred to in the report of D S PORTER, Chartered Accountant, Mona Vale, NSW 2103.
Dated: 17 April 2005

Audit report to the members of the South Pacific Underwater Medicine Society

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of The South Pacific Underwater Medicine Society for the year ended 31 December 2004 report that the accompanying Income and Expenditure and Balance Sheet have been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

Dated: 17 April 2005

David S Porter
Chartered Accountant
Suite 7, Ground Floor, 20 Bungan Road
Mona Vale, New South Wales 2103

**THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY MOVEMENTS ON BANK BALANCES
FOR THE YEAR ENDED 31 DECEMBER 2004**

	2004	2003
OPENING BALANCES		
ANZ bank – ASM account	6,447	9,212
– Access cheque account	13,632	8,085
– Plus	<u>96,400</u>	<u>92,825</u>
	116,479	110,122
add, RECEIPTS	<u>137,666</u>	<u>142,536</u>
	254,145	252,658
less, PAYMENTS	<u>144,253</u>	<u>136,179</u>
CLOSING BALANCES		
ANZ bank – ASM account	-	13,632
– Access cheque account	46	6,447
– VZ Plus	100,590	96,400
– SPUMS Annual Scientific Meeting	6,949	-
BNZ Achiever Savings	<u>2,307</u>	-
	<u>109,892</u>	<u>116,479</u>

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

The Australian and New Zealand College of Anaesthetists Special Interest Group in Diving and Hyperbaric Medicine

The Australian and New Zealand College of Anaesthetists now awards a Certificate in Diving and Hyperbaric Medicine to specialists, in recognition of the expertise and specialist knowledge required for practice in this field of medicine. The Certificate represents the significant achievement of recognition of diving and hyperbaric medicine as a 'subspecialty' by a well-established, learned College, and is an important step toward raising the profile of diving and hyperbaric medicine throughout the medical community. The SPUMS Diploma is regarded as the basic qualification for entry into the training for the ANZCA Certificate, which is intended for those who are working in hospital-based hyperbaric units, providing hyperbaric oxygen therapy for a variety of medical conditions as well as diving-related disorders. Details of the training requirements for the ANZCA Certificate in Diving and Hyperbaric Medicine are now posted on the ANZCA website, <www.anzca.edu.au>, under education and training.

The first examination was held in September 2004, four candidates presented, of whom three passed. It is anticipated that the exam will be held annually, according to demand. At present there has not been a date set for the next

examination. In order to be eligible to sit the exam, trainees must have completed their SPUMS Diploma, and a minimum of 12 of the 18 months' FTE clinical experience in an accredited unit. There are currently three accredited units in Australia, at Royal Hobart Hospital, Fremantle Hospital, and Prince of Wales Hospital Sydney. Hyperbaric units wishing to be accredited for training should contact the Australian and New Zealand College of Anaesthetists for details and an application form.

On behalf of all the members of the SIG, I would like to congratulate Dr Bob Wong on his unceasing efforts to achieve recognition of diving and hyperbaric medicine within the ANZCA, culminating in the Certificate we have today.

The ANZCA/ASA SIG in Diving and Hyperbaric Medicine will hold its Annual General Meeting at the Hyperbaric Nurses and Technicians Meeting in Melbourne in August 2005. Notice of the meeting and an agenda will be circulated to all members prior to this meeting.

Margaret Walker, Chairperson

SPUMS Annual Scientific Meeting 2003

Negative neurofunctional effects of frequency, depth and environment in recreational scuba diving: the Geneva “memory dive” study [Abstract]

Slosman DO, De Ribaupierre S, Chicherio C, Ludwig C, Montandon ML, Allaoua M, Genton L, Pichard C, Grousset A, Mayer E, Annoni JM, De Ribaupierre A

Division of Nuclear Medicine, Geneva University Hospital, Geneva, Switzerland. <slosman@medecine.unige.ch>

Abstract

Objectives: To explore relationships between scuba diving activity, brain, and behaviour, and more specifically between global cerebral blood flow (CBF) or cognitive performance and total, annual, or last 6 months' frequencies, for standard dives or dives performed below 40 m, in cold water or warm sea geographical environments.

Methods: A prospective cohort study was used to examine divers from diving clubs around Lac Lemman and Geneva University Hospital. The subjects were 215 healthy recreational divers (diving with self-contained underwater breathing apparatus). Main outcome measures were: measurement of global CBF by (133)Xe SPECT (single photon emission computed tomography); psychometric and neuropsychological tests to assess perceptual-motor abilities, spatial discrimination, attentional resources, executive functioning, and memory; evaluation of scuba diving activity by questionnaire, focusing on number and maximum depth of dives and geographical site of the diving activity (cold water vs warm water); and body composition analyses (BMI).

Results: (1) A negative influence of depth of dives on CBF and its combined effect with BMI and age was found. (2) A specific diving environment (more than 80% of dives in lakes) had a negative effect on CBF. (3) Depth and number of dives had a negative influence on cognitive performance (speed, flexibility and inhibition processing in attentional tasks). (4) A negative effect of a specific diving environment on cognitive performance (flexibility and inhibition components) was found.

Conclusions: Scuba diving may have long-term negative neurofunctional effects when performed in extreme conditions, namely cold water, with more than 100 dives per year, and maximal depth below 40 m.

Reprinted with kind permission from: Slosman DO, De Ribaupierre S, Chicherio C, Ludwig C, Montandon ML, Allaoua M et al. Negative neurofunctional effects of frequency, depth and environment in recreational scuba diving: the Geneva “memory dive” study. *Br J Sports Med.* 2004; 38: 108-14.

Key words

Scuba diving, deep diving, cold, cerebral blood flow, morbidity, abstracts, reprinted from

This paper was presented by Professor Slosman at the SPUMS Annual Scientific Meeting 2003 in Palau.

Diving-related fatalities document resource

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

These documents have been the basis for the series of reports previously printed in this Journal as Project Stickybeak.

These documents will be available free of charge to bona fide researchers attending the library in person, subject to the stipulation that the researcher signs an agreement that no identifying details are to be made public.

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

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

Articles reprinted from other journals

Hyperbaric oxygen treatment and survival from necrotizing soft tissue infection [Abstract]

David Wilkinson and David Doolette

Abstract

Hypothesis: Necrotizing soft tissue infection (NSTI) refers to a spectrum of infective diseases characterized by necrosis of the deep soft tissues. Features of manifestation and medical management have been analyzed for association with outcome. The use of hyperbaric oxygen (HBO₂) therapy has been recommended as an adjunctive treatment but remains controversial.

Design: Retrospective cohort study.

Setting: A major tertiary hospital.

Patients: All patients admitted with a diagnosis of NSTI across a 5-year period.

Intervention: Features of manifestation and medical management were analyzed for their association with survival to hospital discharge. Long-term survival was analyzed for the intervention of HBO₂ therapy.

Main Outcome Measures: Primary outcome was survival to hospital discharge. Secondary outcomes were limb salvage and long-term survival after hospital discharge.

Results: Forty-four patients were reviewed, with 6 deaths (14%). Survival was less likely in those with increased age, renal dysfunction, and idiopathic etiology of infection and in those not receiving HBO₂ therapy. Logistic regression determined the strongest association with survival was the intervention of HBO₂ therapy ($P = 0.02$). Hyperbaric oxygen therapy increased survival with an odds ratio of 8.9 (95% confidence interval, 1.3 – 58.0) and a number needed to treat of 3. For NSTI involving an extremity, HBO₂ therapy significantly reduced the incidence of amputation ($P = 0.05$). Survival analysis revealed an improved long-term outcome for the HBO₂ group ($P = 0.002$).

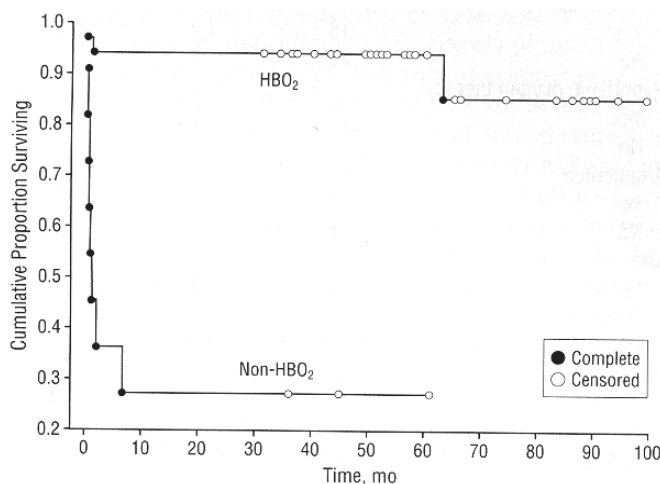
Conclusion: Hyperbaric oxygen therapy was associated with improved survival and limb salvage and should be considered in the setting of NSTI.

Reprinted with kind permission from Wilkinson D, Doolette D. Hyperbaric oxygen treatment and survival from necrotizing soft tissue infection. *Arch Surg.* 2004; 139: 1339-45.

Key words

Hyperbaric oxygen, necrotising infections, gas gangrene, infectious diseases, abstracts, reprinted from

Figure (from Wilkinson and Doolette)
Kaplan-Meier survival curve for hyperbaric oxygen (HBO₂) and non-HBO₂ groups commencing from the time of hospital admission with the diagnosis of necrotizing soft tissue infection.



Commentary

There are no published prospective randomised clinical trials (RCTs) of hyperbaric oxygen therapy (HBOT) for necrotising soft-tissue infections, though many case series, some quite large, have been published. There is also a good theoretical and experimental basis for HBOT in these infections. Case series, such as this one from the Royal Adelaide Hospital, and animal studies suggest that it is not so much the nature of the organism (e.g., *Clostridium sp.*) *per se* but rather the microbial–host interaction and the synergy between antibiotics and HBOT that are important in treating these devastating infections.

This is the first report of which the Editor is aware that has looked at the long-term outcome from such infections, and the evidence is compelling. It would be a brave Ethical Committee that sanctioned an RCT in a hospital with hyperbaric facilities with a non-HBOT treatment arm in a condition that carries a mortality of 30–70% in the absence of HBOT.

Michael Davis

Editor, SPUMS Journal

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Risk of decompression illness among 230 divers in relation to the presence and size of patent foramen ovale [Abstract]

Torti SR, Billinger M, Schwerzmann M, Vogel R, Zbinden R, Windecker S, Seiler C

Background: The risk of developing decompression illness (DCI) in divers with a patent foramen ovale (PFO) has not been directly determined so far; neither has it been assessed in relation to the PFO's size.

Methods: In 230 scuba divers (age 39 ± 8 years), contrast trans-oesophageal echocardiography (TEE) was performed for the detection and size grading (0–3) of PFO. Prior to TEE, the study individuals answered a detailed questionnaire about their health status and about their diving habits and accidents. For inclusion into the study, ≥ 200 dives and strict adherence to decompression tables were required.

Results: Sixty-three divers (27%) had a PFO. Overall, the absolute risk of suffering a DCI event was 2.5 per 10^4 dives. There were 18 divers (29%) with, and 10 divers (6%) without, PFO who had experienced ≥ 1 major DCI events ($P = 0.016$). In the group with PFO, the incidence per 10^4 dives of a major DCI, a DCI lasting longer than 24 hr and of being treated in a decompression chamber amounted to 5.1 (median 0, interquartile range [IQR] 0–10.0), 1.9 (median 0, IQR 0–4.0) and 3.6 (median 0, IQR 0–9.8), respectively and was 4.8–12.9-fold higher than in the group without PFO ($P = 0.001$). The risk of suffering a major DCI, of a DCI lasting longer than 24 hr and of being treated by recompression increased with rising PFO size.

Conclusion: The presence of a PFO is related to a low absolute risk of suffering five major DCI events per 10^4 dives, the odds of which is five times as high as in divers without PFO. The risk of suffering a major DCI parallels PFO size.

Reprinted with kind permission from: Torti SR, Billinger M, Schwerzmann M, Vogel R, Zbinden R et al. Risk of decompression illness among 230 divers in relation to the presence and size of patent foramen ovale. *Eur Heart J.* 2004; 25: 1014-20.

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Key words

Patent foramen ovale, decompression illness, cardiovascular, reprinted from

Commentary

Paul Langton

This seminal study retrospectively examines the association of decompression illness (DCI; decompression sickness and/or arterial gas embolism) with patent foramen ovale (PFO) in a group of 230 experienced divers. Torti et al used a standardised trans-oesophageal echocardiography (TOE, or TEE) technique with echo contrast and Valsalva manoeuvre to detect PFO in their cohort of divers. In addition to detection of PFO, the functional size was arbitrarily graded from one (smallest) to three (largest). The study was adequately powered to detect differences between groups.

Torti et al demonstrated that the odds of suffering DCI were significantly increased in the presence of a PFO. Although unclear from the abstract, the four- to six-fold increase was restricted to subjects with larger shunts (grade 2 or 3). Subjects with a small PFO (grade 1; defined as “*crossover of a few single bubbles*”) had no more DCI than subjects without PFO. The principal findings are consistent with and extend previous observations of Wilmschurst and others.

The take-home messages are:

- The incidence of major DCI is very low, with an average of 2.5 per 10,000 dives.
- The larger functional PFO shunts are, the greater is the incidence of DCI.

The authors recommend that divers with a grade 2 or 3 PFO refrain from diving altogether. This advice appears to be based on the “*uncertain neurological hazards of asymptomatic ischaemic brain lesions...which have been found to be related to PFO in divers.*”¹ Given the very low demonstrated prevalence of DCI such advice would seem premature. The paper makes no mention of the incidence of recurrent DCI and its distribution between divers with and without PFO. This additional information would provide useful guidance for our advice to patients after an episode of DCI. The potential for percutaneous device closure of PFO is briefly mentioned. Retrospective series have demonstrated the safety of this technique; prospective

randomised trials are under way and hard outcome data are eagerly awaited.

Limitations

The need for TOE in contrast to trans-thoracic echocardiography (TTE) remains unclear and continues to be debated; TTE is more accessible and less invasive. Whilst it is clear that TOE offers greater sensitivity in detecting PFO, it appears to do so at the cost of specificity.²⁻⁴ Indeed, this fact is alluded to in the reference used by the authors in their paper.⁵ In addition, the ability to adequately perform Valsalva manoeuvre is compromised by the trans-oesophageal probe position. Regrettably no TTE information in the current series is available. Our own practice is to perform TTE at the same time as TOE; if a PFO is demonstrated on TTE, the more invasive TOE is not required. Indeed, our anecdotal rate of large PFO shunts on TOE is similar to the rate detected with TTE.

The authors do not state from where the divers were recruited. They were experienced divers (minimum of 200 dives each, with average exposure of approximately 450 dives) with self-stated “*strict adherence to decompression tables*”. The population may therefore not be representative of the general recreational-diving community. The findings are, however, likely to be broadly applicable. Larger, confirmatory studies in recreational-diving cohorts will be of interest.

Additional comments

PFO was initially proposed as a cause of cryptogenic stroke in young subjects. Progressive stretching of the fossa ovalis may occur during normal ageing and may lead to an increased likelihood of paradoxical embolism in later life. This effect would be expected to be magnified by hypertension and/or atrial fibrillation, both of which lead to atrial dilatation. Recently, PFO has been found to be more predictive of recurrent CVA in an older population.⁶

Of interest was the observation of a greater prevalence of migraine in subjects with a PFO. This is an area of increasing interest in the specialty of Neurology. There have been a number of recent retrospective reports documenting a reduction in migraine after percutaneous PFO closure.⁷ Prospective randomised studies are awaited.

Analogy to asthma

This study confirms that PFO is simply and reliably detected. Divers with a PFO have a statistically significantly increased risk of a clinically relevant, albeit rare, outcome. There is no routine recommendation to screen prospective divers for (large) PFO or to advise them of the availability of echocardiography screening. It is interesting to compare

this level of science with contemporary diving practice in relationship to ‘asthma’. A number of expert bodies recommend provocative testing for hypertonic saline responsiveness. Despite many theoretical concerns, there are few or no outcome data to show that the disease detected, be it truly asthma or not, carries any statistically significant or clinically relevant risk. Indeed, retrospective series show a poor predictive value, with strongly positive test results in a number of experienced divers who have had no DCI. Is it time to reconsider our advice to divers?

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Co-ordinated investigation into the possible long term health effects of diving at work. Examination of the long term health impact of diving: The ELTHI diving study [Abstract]

Macdiarmid JI, Ross JAS, Taylor CL, Watt SJ, Adie W, Osman LM, Godden D, Murray AD, Crawford JR, Lawson A

Departments of Radiology and Environmental and Occupational Medicine, University of Aberdeen, and Departments of Medicine and Therapeutics and Psychology, University of St Andrews, Highland & Island Health Research Institute and Arnold School of Public Health, University of South Carolina

Abstract

We explored self-reported health and health related quality of life in a large group of divers (n = 1540; 56% response) compared to a non-diving group of offshore industrial workers (n = 1035; 51% response) with a questionnaire survey. We then validated the questionnaire responses by a detailed clinic assessment of a 10% random sample from each group. This included a range of objective tests and measurements. Finally, we studied reported 'forgetfulness or loss of concentration' in a case-control study, to determine the significance of this symptom and its relationship with diving practice. Health related quality of life (HRQOL) was similar in each group and within normative values. The major work related factor affecting HRQOL was industrial accident and this effect was most marked for offshore workers. A significant group of divers (18%) complained of 'forgetfulness or loss of concentration' and this was related to their diving experience. This complaint was associated with a significant moderate reduction in group mean HRQOL. A random sample of this group had a lower group mean performance on objective tests of cognitive function most particularly of memory and structural differences from control on cerebral MRI. The practice of welding had an unexpected amplifying effect in terms of the symptoms experienced by divers. There was a very high prevalence (50%) of objectively determined hearing disorder in both divers and offshore workers. This report and the work it describes were funded by the Health and Safety Executive (HSE). Its contents, including any opinions and/or conclusions expressed, are those of the authors alone and do not necessarily reflect HSE policy.

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Key words

Occupational diving, health surveillance, questionnaire, diving industry, morbidity, epidemiology

Commentary

David Elliott

Background

What follows is not a book review because the report is not a book. Nor is this an article reprinted from another journal. The publication to be reviewed is a research report that is available as a free download on the website of a government department. Though much of this rather lengthy report has been referred to as a whole or in part in various EUBS and UHMS abstracts, this encompassing project may be at risk of being overlooked because the original is invisible to Medline. Indeed the UK HSE website has a number of decompression-related and other research reports, each in full, just waiting to be opened by a visitor. Not all of them have been cited in the spotlight of traditional publications. No doubt web-crawlers are filling this gap but at present, though the 'advanced search' of one search engine reaches

some out-of-date pages of the HSE, they highlight much that is not peer-reviewed or that is just not relevant.

Our editor invited this particular review for the purpose of bringing this study, *The Examination of the Long Term Health Impact of Diving*, to wider attention and doing so concisely because the document's own abstract is too lengthy for direct reproduction in this journal (although the condensed version is presented above). The following synopsis is drawn from the writings of its ten authors and any misinterpretation is the error of this reviewer. Because the content surely has relevance to all in this field, the principal recommendation is that you download the article from the site detailed at the foot of this article, and read it in whole or in part. But first you need to know why.

Introduction

Recovery from decompression and other diving illnesses depends on the nature of the diving and severity of the condition. In many cases recovery is complete. Less certain are the possible long-term health effects of diving, particularly in the absence of a history of an injury or decompression illness (DCI).

Dysbaric osteonecrosis is an example that is associated with work in hyperbaric environments. The first cases, presenting as a painful shoulder or hip, were found to be due to subchondral collapse and not related to a recent decompression. The natural history of this condition is now thought to be quite different from that of other avascular necrosis such as idiopathic femoral head necrosis and, if not static, progress is measured in years rather than months. The assessment of this pre-clinical condition among an apparently healthy workforce led to the introduction of control measures, such as modification of the decompression procedures, and the subsequent monitoring of the population at risk by routine health surveillance. It is now a notifiable industrial disease.

In contrast, some papers on other aspects of diver health have not followed that basic sequence in occupational medicine of recognise, assess, control and monitor. This research has raised public concern even though few significant findings have been associated conclusively with a career in diving. The illnesses concerned include neurological abnormalities, inner-ear deficits and changes of lung function. Other findings have yet to be associated with any known disease state and some are incidental findings that seem to have little impact on the diver's quality of life.

The synopsis

In this project, professional divers and a comparison group of age-matched workers from the offshore oil and gas industry who had never dived were selected for review. They were required to have been working in their respective industry for at least 10 years prior to the study. This timing was set to allow medical conditions related to their career to become apparent. The study was in two parts, a postal survey and, for two sub-samples of responders, a clinic investigation.

QUESTIONNAIRE SURVEY

A postal questionnaire was sent to 2,958 divers and 2,708 offshore workers. It was designed to assess occupational history, general health complaints, diagnosed medical conditions and health-related quality of life (HRQOL). From a response rate of more than 50%, there were 1,540 divers and 1,035 offshore workers who met the inclusion criteria for the study.

From the broad range of other results presented in the report, the questionnaire survey identified three complaints that were more common in divers than offshore workers:

- 'forgetfulness or loss of concentration'
- 'joint pain or muscle stiffness'
- 'impaired hearing'.

Divers were three times more likely to report symptoms of 'forgetfulness or loss of concentration' than an age-matched group of offshore workers. The complaint of 'forgetfulness and loss of concentration' was found to be the most significant long-term health effect and was not explained by factors such as welding, three-day accidents, head injury, DCI and lifestyle (age, alcohol consumption, smoking). Divers reporting 'forgetfulness or loss of concentration' tended to have had longer diving careers. Also, specific diving techniques such as mixed-gas bounce, saturation and surface decompression diving appeared to have had dose-response effects for this subjective complaint, and those reporting it were more likely to have suffered DCI. Adjusting for this factor reduced the relationship between 'forgetfulness or loss of concentration' and surface decompression diving, but not its relationship with mixed-gas bounce and saturation diving. This suggested that this complaint was explained by DCI in surface decompression diving but not in mixed-gas diving.

Complaints of 'forgetfulness or loss of concentration' and 'joint pain or muscle stiffness' were associated with a significantly lower HRQOL (health-related quality of life) score. Despite divers reporting more factors that were associated with lower HRQOL (symptoms, accidents, head injuries), as a group their reported HRQOL did not significantly differ from that of offshore workers. Further investigation of this paradox demonstrated that these factors have less of an influence on the HRQOL of divers than that of offshore workers.

CLINIC STUDY OF RELIABILITY

In the first phase of the clinic study a 10% age-stratified random sample was made of those divers and offshore workers who had completed the questionnaire (n = 151 and n = 103 respectively). The purpose of this was to check the reliability of the postal questionnaire and to identify asymptomatic hearing or neuropsychological deficits that would not have been detected in the postal questionnaire.

Consistency of reported symptoms indicated a moderate strength of agreement between enquiries and that the diving history reported in the questionnaire survey was consistent with the data recalled in the occupational interview. Subjects complaining of 'forgetfulness or loss of concentration' in the questionnaire, as a group, performed less well on objective tests of memory and concentration. Subjects complaining of 'impaired hearing' were more likely to have a detectable abnormality in the audiograms than subjects not reporting this complaint.

Abnormality (below 1.65 standard deviations from the mean for the general population) for neuropsychological tests was selected for an incidence rate of 5% in the general population. The incidence of abnormality in non-complainers remained below this rate in all the tests, suggesting that there was no evidence of neuropsychological abnormality that the questionnaire failed to identify. In the hearing test, however, abnormal audiograms were identified for 42% of divers and 45% of offshore workers who reported that they did not suffer from hearing impairment. This finding was anticipated and is well recognised, because early signs of noise-induced hearing loss identified on audiograms are generally asymptomatic.

CASE-CONTROL STUDY

The second phase of the clinic study was a case-control study that comprised 'forgetful' divers (F divers: n = 102) while the controls were 'non-forgetful' divers (NF divers: n = 100) and 'non-forgetful' offshore workers (NF OSW: n = 100). Subjects completed the following tests: subjective and objective measures of neuropsychological function, lung function, hearing, balance, a medical examination and detailed occupational history (including diving experience and accidents). In addition to these tests, subjects in the case-control study had MRI of the brain, including voxel-based morphometry (VBM) to characterise regional cerebral volume.

F divers were found to perform more poorly on objective neuropsychological tests of memory and concentration. Diving experience continued to be significantly associated with what the authors refer to as 'caseness', with F divers having done significantly more dives than NF divers. However, among divers in the case-control study, there was no substantive relationship between objective cognitive performance and the amount of diving performed. Consistent with the questionnaire survey, a higher proportion of F divers had done mixed-gas bounce, surface-oxygen decompression and saturation diving than NF divers. Taking into account confounding factors, 'forgetfulness or loss of concentration' was found to be associated with an increased incidence of periventricular hyperintensities on MRI. Periventricular hyperintensities have been related in previous studies to lower cognitive performance. Divers were found to have an increased likelihood of subcortical or deep white matter hyperintensities compared with offshore workers, but this was not related to forgetfulness.

An interesting finding of the study was that divers, as a group, did not rate their HRQOL differently, despite a greater proportion of divers having symptoms that are usually associated with lower HRQOL. It appeared that symptoms such as joint pain, hearing loss and forgetfulness had less of an impact on divers than offshore workers. However, the case-control study found significant deficits in HRQOL of

F divers, compared with NF divers and NF offshore workers. This suggests that the HRQOL impact of forgetfulness could have been disguised in the screening survey when only the minority of the population reported the complaint of 'forgetfulness or loss of concentration'.

Conclusions

The ELTHI diving study did not identify any long-term health effects associated with professional diving amounting to a clinical abnormality. The extent of the neuropsychological effect observed in forgetful divers was at a level indicative of mild sub-clinical deficit. 'Forgetfulness or loss of concentration' was associated with significant impairment of HRQOL and MRI changes. The authors expressed the need for further study. Their concerns include whether their observation represents one point on a progressive decline of function or is merely the result of some form of diving-related insult with stable consequences. They also wish to establish whether any form of recovery occurs after cessation of diving.

In the opinion of this reviewer the main obstacle for the ELTHI team and other researchers will not be academic in nature but related to funding. I believe that this was a cross-sectional study because most government-funded departments do not have the authority to fund research in the longer term and I am aware that the HSE rejected several proposals for longitudinal studies (that included osteonecrosis with MRI). I hope they will relent.

Thus this report represents a substantial study that defies reduction to an adequate and accurate synopsis. One must also thank the UK HSE (diving and medical divisions) for their record of funding this and many other studies in the past thirty years. The recommendation of this reviewer is that each reader of this now downloads the original, reads it (in whole or in part), and then keeps it...filed under 'Milestones'.

Professor David H Elliott, DPhil(Oxon), FRCP, FFOM, OBE, is a Life Member of SPUMS. He claims to have retired.

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Readers can download the full report reviewed here from the HSE website at:

<<http://www.hse.gov.uk/research/rrhtm/rr230.htm>>

Letters to the Editor

Electrocardiographic abnormalities in young athletes and scuba divers

Dear Editor,

I found the case report by Dr Acott of the sad death of a 17-year-old diver with a long QT interval most interesting.¹ I would like to offer some thoughts on this case. The electrocardiogram (ECG) in this normothermic diver shows a clearly prolonged QTc with notching on the R wave. This notching may well represent an intraventricular conduction defect; however, it may also represent early after-depolarisation occurring in the repolarisation phase of the action potential. This may trigger a spontaneous discharge and has been shown in dog Purkinje fibre models to initiate *torsades de pointes* ventricular tachycardia.²

In the context of diving-induced hypothermia, however, a similar ECG pattern may be seen with pronounced notching of the R wave, and the second wave may be as tall as the preceding R wave, with prolongation of QT interval and sinus bradycardia. This was described in 1953 by Osborn as an injury current, called a J wave, and may initiate ventricular fibrillation during experimental hypothermia. As the body rewarms, the Osborn waves diminish in amplitude and finally fade away.³

In both the normothermic and hypothermic rescued diving victim, confusion may further occur in those suffering with the autosomal dominant disorder due to mutations in the sodium channel (often SCN5A), described in 1991, and known as the Brugada Syndrome. The typically young South-East Asian or Japanese, with a structurally normal heart, develops nocturnal syncope due to non-sustained polymorphic ventricular tachycardia. The resting ECG shows a combination of an RBBB pattern in VI-3 with variable ST-segment elevation and a coved or saddle-type appearance.⁴ Therefore, a similar ECG abnormality to your case may be seen in a young, rescued Oriental diver.

My second point is the rising incidence internationally of sudden death in young athletes (aged less than 35 years) with, as you are aware, hypertrophic cardiomyopathy (HCM) being the most common cause. This young age group encompasses a great number of scuba divers throughout the world. For more than 30 years the Italian government, as a result of the Medical Protection of Athletic Activities Act, has mandated national pre-participation screening and medical clearance of all young athletes who participate in organised sports. This screening involves a 12-lead ECG. Since the ECG is abnormal in up to 95% of patients with HCM, this programme permits identification of many athletes with previously undiagnosed disease.⁵ The ECG also identifies other rhythm disturbances and, in particular, the long QT syndrome, as in this case study.

I am both a designated aviation medical examiner and an approved examiner for scuba trainees. I am sure that the value of an ECG taken at the initial medical screen would have been discussed in open forum at SPUMS meetings. However, I am not aware of the specific reasons advanced to exclude the ECG from the examination. This 17-year-old would have been denied a medical clearance for scuba diving had his ECG revealed the prolonged QT interval.

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Key words

Deaths, cardiovascular, prolonged QT syndrome, letters (to the Editor)

Survey of skin and scuba divers in the December 2004 Indonesian tsunami

Dear Editor,

We are conducting a world-wide Internet survey of skin and scuba divers who were in or on the water in the Indonesian Tsunami of 26 December 2004. There has been little or no published information regarding the effects of natural disasters on divers. Drs Tom Skalko and Carmen Russoniello of East Carolina University in Greenville, NC, USA and I are studying what that experience was like for divers and how it has affected their lives since then. We need divers to help us by completing the survey.

Some of the survey questions may be upsetting, even months after the Tsunami. It will take about 10 to 15 minutes to complete. The survey will automatically skip past questions that do not apply based upon answers to earlier questions. Results will be compiled and completed as a group only.

No individual identifying information will be released to anyone. The risks of participating in this survey are considered minimal.

If you or any skin or scuba divers whom you know were there in the Tsunami, please ask them to go to: <<http://www.zoomerang.com/survey.zgi?p=WEB2247GPQ5XL9>> to complete the survey.

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Key words

Survey, trauma and stress, diving, underwater hazards

New requirement for the SPUMS Diploma of Diving and Hyperbaric Medicine

Dear Editor,

The SPUMS Committee have unanimously agreed that there should be a minor change to the SPUMS Diploma requirements. A 24-month SPUMS membership is now necessary before the Diploma will be awarded. This does not prevent any future candidate or new member of SPUMS from completing the Diploma within this time but means that the Diploma will not be awarded until 24 months' continuous membership has been completed. This change results from recent unreasonable demands on members of the SPUMS Academic Board, whose work is voluntary, in relation to the new ANZCA Certificate in Diving and Hyperbaric Medicine.

In addition, the SPUMS Committee is also considering that a condition of award is that SPUMS Diploma theses are to be published in the SPUMS Journal. As the Education Officer I proposed this change because the SPUMS Journal is indexed on EMBASE and in the near future we will be reapplying for Medline indexing. It is essential, in order for this to succeed, that the Journal publishes original work. Members' views on this move are sought.

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Key words

Medical society, qualifications, letters (to the Editor),

Maintenance of Professional Standards

Dear Editor,

The following Maintenance of Professional Standards (MOPS) points have been approved by the Australian and New Zealand College of Anaesthetists (ANZCA):

- 1 The 2005 SPUMS Annual Scientific Meeting (ASM) has been approved under Code 700 (other activities) for 38.5 CME and 7 QA points. The approval number for this activity is 0587.
- 2 The 2005 Undersea and Hyperbaric Medical Society (UHMS) Annual Scientific Meeting (ASM) in June has been approved as a Major CME Meeting under Code 111 for 3 CME points per hour. The approval number for this activity is 0580.

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Key words

MOPS, letters (to the Editor)

The ocean storage of nuclear waste

One of the most daunting issues facing the global community is the storage of nuclear waste. The need to address this issue is urgent as the problems are severe and will continue to worsen. One potential storage option is deep, ocean storage, which, according to advocates, may provide important benefits compared to land-based storage.

Over the last decade, international research of nuclear waste dumped by the former Soviet Union in the marine environment in the Arctic is leading scientists to suggest that a comprehensive project plan, coordinated by a neutral third-party project manager, is needed to fully evaluate ocean storage of nuclear waste.

The various aspects of an ocean storage system are:

- isolating nuclear waste in very-long-life, total containment systems at 1,000 to 2,000 metres' depth on the ocean bottom
- continuous monitoring
- controlled accessibility for recovery and repackaging, if necessary
- replaceable by unforeseeable technology alternatives and storage systems developed in the future.

It appears there is a long way to go yet before this becomes a feasible and safe option for nuclear waste storage.

(Summary of a discussion article by Weldon C, *Sea Technology*. 2003; September: 29-33.)

Book review

The ECHM Collection, Volume 1

592 Pages, hardback

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Price: \$US89.00 + P&P

The European Committee for Hyperbaric Medicine (ECHM) Collection 1994–1999 is an interesting book that is difficult to review as by definition it is out of date and research has advanced in the six years since its final consensus meeting. However, as stated in the introduction, it is an effort to bring the proceedings of four consensus conferences, three thematic workshops and two ECHM reports into a single volume of standardised format and type. In this it is a successful endeavour.

The ECHM Collection is broken up into four main sections each with several parts. The sections are:

- 1 A general introduction to the ECHM and its organisation and goals
- 2 Proceedings of the four main consensus conferences held by the ECHM
- 3 Proceedings of the ECHM workshops
- 4 ECHM reports

The first component is an interesting insight into the structure and aims of the ECHM, as well as a discussion of the principles for recommendations for hyperbaric oxygen therapy (HBOT) with the introduction of evidence-based medicine guidelines for recommendations. The book then lists most of the accepted indications and also some of the more controversial indications stating the evidence for and against the use of HBOT. This section, which is interesting, unfortunately suffers from the ravages of time in that several recent studies are obviously not included that may help strengthen or weaken the assumptions at the time of writing (for example, Weaver et al's recent study on carbon monoxide poisoning).

The second component (which is labelled section I) is the articles from the consensus conferences. There are four of these and they become more focused in intention as they progress. The first conference has a mixture of diving medicine, HBOT for wounds and also a significant area on hyperbaric unit safety. Most of the components have an 'introductory report', which obviously sets the meeting up, and then a 'final report', which seems to be as a result of the discussion of the meeting. It is a nice thing to see that even in a euro-centric organisation such as ECHM, highly

respected members from the USA (Moon and Thom amongst others) help bring more worldly experience into the fold and this has enhanced the status of the work considerably. (I unfortunately could not find any Australasian authors as contributors and I hope the next volume will correct this oversight.) A criticism of this section could be made that the reproduced photographs and graphics are often extremely hard to interpret, with the pictures of a skin bend on a pig (on page 1-22) impossible to view and therefore a waste of space.

The next section focuses on the second consensus meeting, which looks specifically at diving aspects. This section has some hidden gems, like Brubakk's monograph on the use of animals in decompression research with tables of saturation times of some of the common animals used in research. There is also a slightly more confusing monograph on the classification of diving injuries based on a descriptive methodology. After a few re-readings I was less confused, but still this is not an easy breakdown of diving illnesses.

The third consensus meeting looks at acute traumatic injury and takes a slightly different tack asking six questions of an expert committee after an introductory monograph is presented. It starts off with the questions and gives the consensus answers (which helps with the reading somewhat). Unfortunately the questions seem to be unanswerable with the current information given, but levels of evidence for their answers are presented and this seems to be a reasonable compromise.

The fourth consensus meeting focuses on the diabetic foot. It starts with a literature review then looks at different components of management. Quite correctly not only the hyperbaric treatment of the diabetic foot but also podiatric and surgical needs are addressed, as well as evaluating the role of hyperbaric oxygen in specific problems such as infection. There are also monographs on the 'cost-benefit' aspect of diabetic foot care and finally a look at a multidisciplinary unit in Lille, France, which shows obvious benefit in lower amputation rates and better outcomes for diabetic patients with foot disease.

The third component is dedicated to the ECHM workshops, which are a little surprising considering that 8 of the 10 monographs are dedicated to fire-suppression systems and management. These monographs look at each country's specific fire-suppression legislation and systems. Also in this section is a look at NFPA regulations in the USA, including a monograph by Tom Workman who is an acknowledged world expert in this field.

The final section is by far the shortest but looks at two components that have only been glossed over in the previous sections: the educational requirements of staff in a hyperbaric unit and the overall safety of multi-place hyperbaric chamber operations. Both of these, you get the feeling, are an attempt to create a 'European standard' that

can be used by the authorities and the publication seems to be on the right track with the two end subsections.

This is not a bedtime-read book, nor does it totally fulfil the requirements of a textbook. It seems to be a good historical record of the events and it attempts to standardise the way in which the evidence is presented. It is not a book that a person who was interested in hyperbaric medicine but not working in the field would use, and its main readership would be found in hyperbaric units already with good libraries and ongoing research commitments. It is not a fully integrated textbook that looks at the world picture and as such requires an equivalent volume from the major US interests (such as UHMS consensus workshops and meetings). All of this being equal it is a good summary collection of monographs that have been well researched and examined by the appropriate people and then judiciously edited to standardise the content.

I do have two complaints. I have already alluded to the poor reproductions of some of the graphics and pictures (which may be due to the original graphics being of poor standard). The second complaint is that it lacks an index. There is a lot of repetition of information throughout (albeit from slightly different angles) and a good index would make this book's reference potential much higher in my opinion: a task for future editors.

All in all this is an admirable attempt to get all the highly useful information that consensus meetings create into a usable form that is almost a pleasure to read (for a technical journal), and for that the editors and authors should be congratulated.

Glen Hawkins
Hyperbaric Fellow, Royal Adelaide Hospital

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

<www.hboevidence.com>

The

SPUMS

web site is at
<http://www.SPUMS.org.au>

The poetry doctor

Why Oh Why?

“Let’s dive, let’s dive
And stay alive”,
Everybody cries.
Yet every year
We always hear
A diver’s hurt or dies.

Deeper, deeper,
No time keeper,
Far too long descent.
Up to the top,
No safety stop
A paraplegic bent.

Drama, drama,
Barotrauma,
Equalisation poor.
Ears pain the worst
Till ear drums burst
Dive ending premature.

Wheezy, wheezy,
Breathe uneasy,
Chest cannot relax.
Full of fear,
No inhaler near
A new pneumothorax.

Bubble, bubble,
Dive in trouble,
Emergency ascent.
Air expanding,
Lungs not withstanding,
Air embolism sent.

Panic, panic.
Act mad and manic
Not thinking from the head.
Drop mouthpiece,
No weight release
So struggle till you’re dead.

Why oh why
We always cry,
Are they careless or just fools?
For we have explained
“Be fit and trained
And follow all the rules”.

John Parker

<www.thepoetrydoctor.com>

10 ways to avoid nitrogen narcosis

- Don't be pressured into doing any deep dive where you are likely to become overly anxious (how many divers go out of their depth on liveboards 'because I've paid for all the dives and I'm going to do them'?)
- 'Dive up' to any deep dives, i.e., do each one progressively deeper. This technique is used by the [Royal] Navy, though there are no guidelines for the time period over which this should take place.
- Use only equipment with which you are familiar on any deep dive – a 40 metre sea water (msw) dive is not the place to try out new kit.
- Consider a slower descent rate – there is some indication that a fast rate can bring on sudden narcosis.
- Attain a level of confidence in any new environment before going deep – a 40 msw dive in the Red Sea may be less narcosis-inducing than a 40 msw dive off the coast of Scotland.
- Practise your drills so that they are second nature. One of the biggest narcosis inducers is task-switching and coping with the unfamiliar.
- Do not dive deep on air! The BSAC limit for air diving is 50 msw [PADI 40 msw] and for good reason. So why are so many of you diving on air between 50 and 70 msw?
- Plan the dive and dive the plan. How many dives have you planned to go no deeper than 35 msw but ended up at 40 msw-plus?
- On any deep dive, have a plan for the onset of narcosis. As soon as you feel its onset, start to ascend. Do not wait for it to develop into 'anxious' or 'blackout' narcosis. If you are suffering some form of narcosis, it is highly likely that your buddy is too.
- Talk to regular buddies about their experiences so that you can watch out for symptoms in them and they can do the same for you. Discuss contingency plans in advance.

Reprinted with kind permission from O'Brien B. Mind Games. *Diver Magazine*. 2005; 50(4): 28-32.

SPUMS Journal CD

The SPUMS Journal, volumes 1-30, is available on CD.

To read and print these documents Adobe Acrobat Reader (version 3 or later) is required. This may be downloaded free of charge from the Adobe web site <www.adobe.com>

The CD is available to members for Aust \$25 (incl. GST or overseas mailing). The cost to non-members and institutions is Aust \$90 inclusive. Supplies are limited.

Cheques or money orders should be made payable to: 'South Pacific Underwater Medicine Society'.

Credit card facilities are not available for this.

Contact: Steve Goble, Administrative Officer

E-mail: <stevegoble@bigpond.com>



The International Journal of Diving History

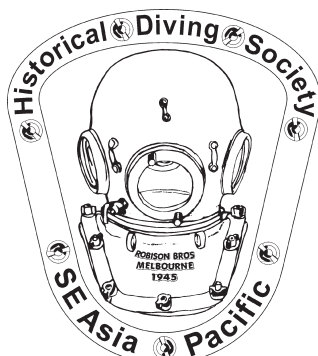
ISBN: 0-9543834-2-7

The first issue of The International Journal of Diving History appeared in April 2005, published by the Historical Diving Society. The journal is dedicated to "promoting and preserving our diving heritage". The Editor is Nigel Phillips and the journal is published once a year in April.

This first issue is mainly devoted to the development of the one-atmosphere suit; a very interesting read.

Single issues may be purchased from the Secretary of the Historical Diving Society, Michael Fardell, Little Gatton Lodge, 25 Gatton Road, Reigate, Surrey RH2 0HB, UK.

E-mail: <mjf@lglodge.freeserve.co.uk>



DIVING HISTORICAL SOCIETY

AUSTRALIA, SE ASIA

All enquiries to:

Diving Historical Society

Australia, SE Asia,

PO Box 2064,

Normansville, SA 5204,

Australia

Phone: +61-(0)8-8558-2970

Fax: +61-(0)8-8558-3490

E-mail: <bob@hyperbarichealth.com>

INTRODUCTORY DIVING MEDICINE COURSE FOR GENERAL PRACTITIONERS 2005

Dates: Friday 16 to Monday 19 September 2005
Venue: Navy Hospital, Devonport, Auckland, NZ

This course is specifically for registered medical practitioners who wish to learn practical and relevant skills to manage patients with diving-related questions or accidents. Participants who successfully complete the course will be registered with the New Zealand Department of Labour as a 'Designated Diving Doctor' and be able to perform Occupational Diving Medical Examinations.

For more details and application forms go to
<www.navyhyperbaric.co.nz>

UNDERSEA and HYPERBARIC MEDICAL SOCIETY

Annual Scientific Meeting 2006

Dates: 22 to 24 June 2006
Venue: Hilton in the Walt Disney World Resort
Orlando, Florida

For additional information:

Lisa Wasdin
c/o Undersea and Hyperbaric Medical Society
PO Box 1020, Dunkirk, Maryland 20754, U.S.A.
Phone: +1-410-257-6606 ext. 104
Fax: +1-410-257-6617
E-mail: <lisa@uhms.org>

INTERNATIONAL CONFERENCE ON DIVING AND HYPERBARIC MEDICINE Joint meeting of XVth ICHM and 31st ASM European Underwater and Baromedical Society

Dates: 7 to 10 September 2005
Venue: Barcelo Sants Hotel, Barcelona, Spain
Web site: www.barcelona-2005.com
E-mail: <congress@aopc.es>

INTERNATIONAL SYMPOSIUM ON NAVAL MEDICINE

Dates: 19 to 21 October 2005
Venue: Harbiye Military Museum, Istanbul
Hosts: Kasimpasa Naval Hospital
Chairperson: Surgeon Rear Admiral Vehbi Alpman
Topic of Interest
Motion Sickness in the Navy
Official languages: Turkish and English.
Phone: +90-532-414-8233
E-mail: <gunerigok@e-kolay.net>
Web site: www.gunerigok.net/dts/info.htm

SPUMS Annual Scientific Meeting 2006

Preliminary Notice

Confirmation of the dates and venue of the 2006 ASM are not available as we go to press.

It is likely that the meeting will be held in late April in Fiji

The theme is expected to be
"Back to basics"

Conference Convenor: Andrew Patterson
E-mail: <a.j.patterson@exemail.com.au>

Full details and registration documents will appear with the September issue of the Journal

HYPERBARIC TECHNICIANS and NURSES ASSOCIATION 13th ANNUAL SCIENTIFIC MEETING

Theme:

Hyperbaric care in the acutely ill patient

Guest speakers:

Tom Workman, UHMS
Armin Kemmer, Trauma Centre Murnau
Sue Churchill, Salt Lake City

Dates: 18 to 20 August 2005
Venue: Novotel St Kilda, Melbourne
Host institution: The Alfred Hyperbaric Service
Registration/Enquiries: <www.htna.com.au>
or <htna2005@alfred.org.au>
Phone: +61-(0)3-9276-2269
Fax: +61-(0)3-9276-3052



The logo for the Wanaka Conference 2005 features the word "Wanaka" in a stylized, blue, wavy font with a red squiggle above it. Below "Wanaka" is the word "Conference" in a bold, black, sans-serif font, followed by "2005" in a similar bold, black, sans-serif font.

**Edgewater Resort, Wanaka, New Zealand
12 to 16 September 2005**

The goals of the meeting are to combine continuing medical education, stimulating presentations, professional interaction and the exhilaration of skiing and snowboarding with family and friends.

For further information: contact Dr Greg Emerson
Phone: +61-(0)7-3636-7901
E-mail: <Greg_Emerson@health.qld.gov.au>
Web site: <www.wanakaconference.com>



THE UNIVERSITY OF AUCKLAND
FACULTY OF MEDICAL AND
HEALTH SCIENCES

Master of Medical Science
Postgraduate Diploma in Medical Science
Diving and Hyperbaric Medicine

Enquiries from registered medical practitioners are now being accepted for 2006 for the Masters and Postgraduate Diploma programmes in diving and hyperbaric medicine.

Faculty: Des Gorman, Michael Davis (Course Director), Simon Mitchell, Chris Acott, Kathleen Callaghan, William Baber and Drew Richardson.

Overview: These are distance-learning programmes, available internationally without a resident component in Auckland. However, attendance at a recognised short course in diving medicine is a prerequisite. Graduates will be able to practise effective clinical diving medicine in a primary care setting or to embark on clinical practice within a hyperbaric medicine environment.

The course titles are:

- Physiology and medicine of diving (obligatory)
- Health surveillance of divers and hyperbaric workers
- Hyperbaric medicine
- Clinical diving and hyperbaric practice (based at a recognised host hyperbaric unit)
- Research essay in diving or hyperbaric medicine
- Research project in diving or hyperbaric medicine

For further information, including fees, please contact the Course Coordinator: Jessica Rorich

Phone: +64-(0)9-373-7599, extn 88489

Fax: +64-(0)9-308-2379

E-mail: <j.rorich@auckland.ac.nz>

Full information on courses and admission regulations is available in the University of Auckland Calendar or online <<http://www.auckland.ac.nz>>

ROYAL AUSTRALIAN NAVY MEDICAL
OFFICERS' UNDERWATER MEDICINE COURSE

2006

Dates: 06 to 17 February, 2006

Venue: HMAS Penguin

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 contraindications to diving and the diving medical, together with the pathophysiology, diagnosis and management of the more common diving-related illnesses.

Cost: \$1833.00 (tbc)

For information and application forms contact:

The Officer in Charge, Submarine & Underwater Medicine Unit, HMAS PENGUIN,
 Middle Head Rd, Mosman, 2088 NSW, Australia

Phone: +61-(0)2-9960-0572

Fax: +61-(0)2-9960-4435

E-mail: <Sarah.Sharkey@defence.gov.au>

JAMES COOK UNIVERSITY AND ANTON BREINL
CENTRE FOR PUBLIC HEALTH AND TROPICAL
MEDICINE

Tropical marine and diving medicine (TM5508:03)

Location: Townsville

Availability: Semester 2, external subject with compulsory limited attendance residential

Dates: October 2005

Staff: Associate Professors G Gordon and P Leggat

Cost: TBC

Overview: This subject, conducted in association with the Townsville Hospital, presents the basic principles of underwater and hyperbaric medicine over a five-day residential programme. A series of lectures, demonstrations and practical sessions highlight the taking of a diving history, performing a diving medical examination, decompression sickness, management of near drowning, hypothermia, sinus and ear barotrauma, diving equipment, gases, physiology, diving hazards and diving techniques. A hyperbaric unit is located at the Townsville Hospital. Optional dives may be conducted.

For further information or to enrol contact:

Marcia Croucher, Senior Student Officer,
 Anton Breinl Centre for Public Health and Tropical
 Medicine,

James Cook University,
 Townsville, Queensland 4811, Australia

Phone: +61-(0)7-4781-6107

E-mail: <sphtm-studentofficer@jcu.edu.au>

ROYAL ADELAIDE HOSPITAL HYPERBARIC
MEDICINE COURSES 2005

Medical Officers Course

August 2005

Basic	1/8/05	to	5/8/05
Advanced	8/8/05	to	12/8/05

October 2005

Basic	17/10/05	to	21/10/05
Advanced	24/10/05	to	28/10/05

DMT Full Course

October 2005 3 weeks, 3/10/05 to 21/10/05

DMT Refresher Course

August 2005 2 weeks, 1/8/05 to 12/8/05

October 2005 (Wk 2), 10/10/05 to 14/10/05

For further information or to enrol contact:

The Director, Hyperbaric Medicine Unit
 Royal Adelaide Hospital, North Terrace
 South Australia 5000 or

E-mail: <Lmirabel@mail.rah.sa.gov.au>

Phone: +61-(0)8-8222-5116

Fax: +61-(0)8-8232-4207

Instructions to authors

(revised June 2005)

The *SPUMS Journal* welcomes contributions (including letters to the Editor) on all aspects of diving and hyperbaric medicine. Manuscripts must be offered exclusively to the *SPUMS Journal*, 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, SPUMS Journal,
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 Word 97 for Windows. 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 be subdivided into the following sections: an **Abstract** of no more than 250 words, **Introduction, Methods, Results, Discussion, Acknowledgements** and **References**. Acknowledgments should be brief. References should be in the format shown below. 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 the *SPUMS Journal*. 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.

The preferred length for original articles is 3,000 words or less. Inclusion of more than five authors requires justification as does more than 30 references per major article. Case reports should not exceed 1,500 words, with a maximum of 10 references. Abstracts are also required for all case reports and review papers. Letters to the Editor should not exceed 500 words (including references, which should be limited to five per letter). 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.

Tables are to be in Word for Windows, tab-separated text rather than using the columns/tables option or other software and each saved as a separate file. They should be

double-spaced each in a separate file. No vertical or horizontal borders are to be used.

Illustrations and figures should be in separate files in TIFF or BMP format. Our firewall has a maximum size of 5 Mb for incoming files or messages with attachments.

Photographs should be glossy, black-and-white or colour. Posting high-quality hard copies of all illustrations is a sensible back-up for electronic files. Colour is available only when it is essential and may be at the authors' expense. Indicate magnification for photomicrographs.

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.

References

The Journal reference style is the 'Vancouver' style (*Uniform requirements for manuscripts submitted to biomedical journals*, updated July 2003. Web site 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 and 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 are given below:

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

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

Any manuscript not complying with these requirements will be returned to the author before it will be considered for publication in the *SPUMS Journal*.

Consent

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

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DIVER EMERGENCY SERVICES PHONE NUMBERS

AUSTRALIA

1-800-088-200 (in Australia)

+61-8-8212-9242 (International)

The toll-free number 1-800-088-200 can only be used in Australia

NEW ZEALAND

0800-4-DES111 or 09-445-8454 (in New Zealand)

+64-9-445-8454 (International)

The toll-free number 0800-4-DES111 can only be used in New Zealand

The DES numbers are generously supported by DAN-SEAP

PROJECT STICKYBEAK

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 regards to identifying details, is utilised in reports and case reports on non-fatal cases. Such reports can be freely used by any interested person or organisation to increase diving safety through better awareness of critical factors.

Information may be sent (in confidence) to:

Dr D Walker

PO Box 120, Narrabeen, NSW 2101, Australia.

DIVING INCIDENT MONITORING STUDY (DIMS)

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

Diving Incident Report Forms (Recreational or Cave and Technical)

can be downloaded from the DAN-SEAP web site: <www.danseap.org>

They should be returned to:

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

PROJECT PROTEUS

The aim of this investigation is to establish a database of divers who dive or have dived with any medical contraindications to diving. At present it is known that some asthmatics dive and that some insulin-dependent diabetics dive. What is not known is how many. How many with these conditions die is known. But how many dive safely with these conditions is not. Nor is the incidence of diving accidents in these groups known.

This project is under the direction of Dr Douglas Walker and Dr Mike Bennett. The investigation has been approved by the Ethics Committee of the Prince of Wales Hospital, Randwick, approval number 01/047.

If you are in such a group please make contact. All information will be treated as **CONFIDENTIAL**.

No identifying details will appear in any report derived from the database.

Write to: Project Proteus

PO Box 120, Narrabeen, NSW 2101, Australia.

E-mail: <diverhealth@hotmail.com>

DISCLAIMER

All opinions expressed are given in good faith and in all cases represent the views of the writer and are not necessarily representative of the policy of SPUMS.

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