Vol 26 No.1 March 1996

SPUMS JOURNAL

ISSN 0813 - 1988

South Pacific Underwater Medicine Society Incorporated

CONTENTS

CONTENTS OF THE PEARL DIVING SUPPLEMENT ARE ON THE BACK PAGE

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.

Print Post Approved
PP 331758/0015

PRINTED BY
Snap Printing
344 Burwood Road
Hawthorn, Victoria 3122

OBJECTS OF THE SOCIETY

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

OFFICE HOLDERS

MEMBERSHIP

Membership is open to medical practitioners and those engaged in research in underwater medicine and related subjects. Associate membership is open to all those, who are not medical practitioners, who are interested in the aims of the society.

The subscription for Full Members is $A80.00 and for Associate Members is $A40.00.

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

Anyone interested in joining SPUMS should write to

SPUMS Membership, C/o
Australian and New Zealand College of Anaesthetists,
630 St Kilda Road,
Melbourne, Victoria 3004,
Australia.

AIR MAIL DELIVERY

The *SPUMS Journal* can be airmailed for the extra annual costs detailed below.

Zone 1	e.g. Papua New Guinea & the South Pacific	$ 11.00
Zone 2	e.g. Indonesia and Malaysia.	$ 13.00
Zone 3	e.g. India and Japan.	$ 16.00
Zone 4	e.g. U.S.A. and Israel	$ 19.00
Zone 5	e.g. Europe, Africa & Sth America	$ 21.00

Those interested in having their copies of the *SPUMS Journal* airmailed should write to

SPUMS Journal, C/o
Australian and New Zealand College of Anaesthetists,
630 St Kilda Road,
Melbourne, Victoria 3004, Australia.

INSTRUCTIONS TO AUTHORS

The SPUMS Journal welcomes contributions (including letters to the Editor) on all aspects of diving and of hyperbaric medicine. Manuscripts must be offered exclusively to the SPUMS Journal, unless clearly authenticated copyright exemption accompanies the manuscript.

Minimum Requirements for Manuscripts

All contributions should be typed, double-spaced, using both upper and lower case, on one side of the paper only, on A4 paper with 45 mm left hand margins. Headings should conform in format to those in the Journal. All pages should be numbered. No part of the text should be underlined. These requirements also apply to the abstract, references, and legends to figures. Measurements are to be in SI units (mm Hg are acceptable for blood pressure measurements) and normal ranges should be included. All tables should be typed, double spaced, and on separate sheets of paper. No vertical or horizontal rules are to be used. All figures must be professionally drawn. Freehand lettering is unacceptable. Photographs should be glossy black-and-white or colour slides suitable for converting into black and white illustrations. Colour reproduction is available only when it is essential for clinical purposes and may be at the authors' expense. Legends should be less than 40 words, and indicate magnification. **Two (2) copies of all text, tables and illustrations are required.**

Abbreviations do not mean the same to all readers. To avoid confusion they should only be used after they have appeared in brackets after the complete expression, e.g. decompression illness (DCI) can thereafter be referred to as DCI.

The preferred length for original articles is 2,500 words or less. Inclusion of more than 5 authors requires justification. Original articles should include a title page, giving the title of the paper and the first names and surnames of the authors, an abstract of no more than 200 words and be subdivided into Introduction, Methods, Results, Discussion and References. After the references the authors should provide their initials and surnames, their qualifications, and the positions held when doing the work being reported. One author should be identified as correspondent for the Editor and for readers of the Journal. The full current postal address of each author, with the telephone and facsimile numbers of the corresponding author, should be supplied with the contribution. No more than 20 references per major article will be accepted. Accuracy of the references is the responsibility of authors. Acknowledgments should be brief.

Abstracts are also required for all case reports and reviews. Letters to the Editor should not exceed 400 words (including references which should be limited to 5 per letter).

References

The Journal reference style is the "Vancouver" style, printed in the Medical Journal of Australia, February 15, 1988; 148: 189-194. In this references appear in the text as superscript numbers.[1-2] The references are numbered in order of quoting. Index Medicus abbreviations for journal names are to be used. Examples of the format for quoting journals and books are given below.

1 Anderson T. RAN medical officers' training in underwater medicine. *SPUMS J* 1985; 15 (2): 19-22
2 Lippmann J and Bugg S. *The diving emergency handbook*. Melbourne: J.L.Publications, 1985

Computer compatibility

The SPUMS Journal is composed on a Macintosh using Microsoft Word 5.1 and PageMaker 4.2. Contributions on 3.5" high density discs, preferably in Microsoft Word, or in any program which can be read as "text" by Microsoft Word (Microsoft Word for DOS, Microsoft Word for Windows, Word Perfect for DOS, Interchange Format (RTF) and some text files) are welcome. Discs must be accompanied by hard copy set out as in **Minimum Requirements for Manuscript**.

Consent

Any report of experimental investigation on human subjects must contain evidence of informed consent by the subjects and of approval by the relevant institutional ethical committee.

Editing

All manuscripts will be subject to peer review, with feedback to the authors. Accepted contributions will be subject to editing.

Reprints are not available

Address for all contributions

Dr John Knight, Editor, SPUMS Journal, C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004, Australia. Fax 61-(0)3-9819 5298.

Telephone enquiries should be made to Dr John Knight (03) 9819 4898, or Dr John Williamson (08) 222 5116.

SPUMS JOURNAL Vol 26 No.1 March 1996

PEARL DIVING SUPPLEMENT

CONTENTS

DIVER EMERGENCY SERVICE

PHONE NUMBERS 1-800-088-200 (Australia) 61-8-373-5312 (International)

The DES number 1-800-088-200 can only be used in Australia.

For access to the same service from outside Australia ring ISD 61-8-373- 5312.

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

P.O. Box 120, Narrabeen, N.S.W. 2101.

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 any incident occurring in your dive party, but do not identify anyone. Most incidents cause no harm but reporting them will give valuable information about which incidents are common and which tend to lead to diver damage. Using this information to alter diver behaviour will make diving safer.

To obtain Diving Incident Report forms write to DIMS, GPO Box 400, Adelaide, South Australia 5000.

THE EDITOR'S OFFERING

This year, on May 3rd SPUMS, will have its 25th birthday. Since that first meeting in the Wardroom of HMAS PENGUIN the Society has grown from 5 members to over 1,100. The Newsletter that Carl Edmonds produced on the Royal Australian Navy's Roneo machine has blossomed into a Journal, which appears regularly, if not always on time, four times a year. Last December's issue was the largest yet, and before it was in the envelopes the Editor was faced with five papers about pearl diving in Australia which should be published in the near future. As readers can see they would have filled the Journal on their own. So the decision was taken to produce them as a supplement, as was done with the Proceedings of the joint SPUMS and Republic of Singapore Navy meeting in 1980.

The SPUMS Journal depends on members to submit original work and on members and guest speakers to provide the texts of the presentations at the Annual Scientific Meeting (ASM) to provide the main filling for its pages. However these two sources are intermittent and guest speakers have been known to speak and fade away. While one can reconstruct a paper from the recording made at the time this is labour intensive and, if the Editor may speak freely, irritating. As the main aim of the Society it to educate and spread the word about diving medicine and diving safety we have no qualms about asking permission from Editors of other Journals for permission to reprint articles of interest which may not otherwise reach our, mainly Australasian, membership. Over the years about one third of the Journal has been these reprinted papers.

Perhaps it would be desirable to devote each issue to a specific topic, which has been suggested in the past, but it is most unusual for about 40 or 50 pages about one topic to arrive in a year. With this in mind there is little alternative to the current policy of publishing something for everyone (we hope) in each issue. If we have failed in this endeavour please write in and let us know.

This issue has a decompression theme running through it. Michal Kluger's paper, reviewing the attitudes of the various Australasian hyperbaric units to five clinical scenarios reveals a very individual approach to treatment. As recreational divers our readers may be less than impressed by the advice from the majority of the units that a person with advancing neurological decompression illness, 1,000 km from the nearest hyperbaric facility, should accept the inevitable progress of his or her disease while, breathing 100% oxygen and being given intravenous fluids, a retrieval is organised. Nearly 30 years ago Carl Edmonds introduced his underwater oxygen treatment for decompression sickness, as it was in those days, for just such a scenario. His recent paper in the September 1995 issue showed that the cost benefits are enormous to the patient.

Then there is Fred Bove's discussion of decompression from last year's ASM. Unfortunately his wide ranging discussion had to be edited down to what your read. Some people are unlucky and get into trouble, although most of us can avoid the problems with attention to some basic rules. To enlighten us about neurological decompression illness we have a paper from Chris Acott discussing the conditions for a return to recreational diving after a neurological insult. Surgeon Commander Broome RN, was working in Bethesda, Maryland with the US Navy until this year. His research related to neurological decompression sickness in pigs (years ago the Editor remember a friend mentioning that pigs made excellent experimental animals as they weighed much the same as humans and the post-experiment corpse was very tasty). His paper shows that some of the bleeding into the spinal cord is post decompression and that the appearances described by Graham Blick, reprinted in the supplement, occurred in his pigs.

Bob Halstead pops the cat among the Queensland pigeons with his paper explaining that diving is adventure, and the diver must take responsibility for his or her safety. This is, of course, at odds with the steady stream of court cases where people try to obtain compensation for "negligent" actions on the part of those who take them out to dive from their boats. While it is comforting to blame others for ones misfortunes, it is always possible that the fault is the victim's for doing something that they should not have done with their current level of skills.

We provide information for those interested in "technical diving" or even in using oxygen enriched air. These topics are the meat in the meal provided at the Annual Scientific Meeting. One reason for reprinting articles from *Diver* is that the British diving scene is much more club diving than in Australasia. This means that there is a view available which is not necessarily that of the commercial diver training organisations.

Finally the Pearl Diving Supplement is an introduction to a different world. Repetitive dives with a 20 minute surface interval for 400 minutes underwater a day, appear certain to cause damage. But the Doppler bubble count does not peak until the diver has been out of the water for 60 minutes on these dives. So perhaps the second, third., fourth or tenth, dive prevents the bubbles causing trouble. Oxygen decompression underwater, may be the key to preventing too much nitrogen build up over the diving trips which last for up to 8 days, depending on tidal conditions. 8 days of 10 dives to 18 m, for 40 minutes of bottom time each dive, would see most recreational divers checking their DAN insurance and their travel insurance for retrieval cover. But pearl divers are no longer developing neurological decompression illness.

ORIGINAL ARTICLES

INITIAL TREATMENT OF DECOMPRESSION ILLNESS: A SURVEY OF AUSTRALIAN AND NEW ZEALAND HYPERBARIC UNITS

Michal T Kluger

Summary

Current treatment for decompression illness (DCI) is usually based on experience, current research data and ongoing clinical assessment. In order to ascertain the current Australasian practice in the treatment of DCI, a four page survey was forwarded to doctors associated with hyperbaric units in Australia and New Zealand. This comprised five clinical scenarios with questions relating to initial therapies, need for hyperbaric oxygen, treatment tables and follow up practice. Thirteen completed forms and protocols from two hyperbaric units were returned (63% response). Results indicated a wide variation in initial assessment policies, treatment protocols and follow up for patients presenting with various categories of DCI. Current opinion is that some recompression profiles may not prevent long term sequelae of DCI, however interpretation of outcome data is difficult due to the variability in treatment policies. It would be of value to have standardised protocols for assessment, treatment and follow up of DCI in order to formulate a rational and effective treatment plan for this group of patients.

Introduction

Anecdotal experience and application of current research data, combined with widely differing backgrounds in training, ensure that there is no uniform approach to the treatment of patients requiring hyperbaric treatment for DCI. This variable approach means that it is difficult to compare the results from various units and to indicate which are the most appropriate treatment protocols. As a result of discussions about the treatment tables used in the management of a group of divers with delayed presentation of DCI, it became obvious that there was a wide variation in treatment options and therefore an attempt was made to ascertain the treatment protocols of the different units around Australia and New Zealand.

Methods

A four page questionnaire was sent to medical staff involved in the clinical decision-making in all the hyperbaric units in Australia and New Zealand. This involved the completion of a structured series of questions relating to specific clinical scenarios that could be encountered in the daily running of a hyperbaric unit. The questionnaire was confidential and anonymous.

The recipients were asked to answer the following five questions for three scenarios.

1 What investigations would you perform, if any, before recompression?
2 What recompression tables would you use initially? What recompression schedule would you use if the symptoms failed to respond to the initial therapy?
3 What other measures would you use in conjunction with recompression therapy?
4 What follow up treatments would you use?
5 How long would you continue to treat the patient?

CASE 1

A 27 year old Japanese diver made a rapid uncontrolled ascent from 15m. Immediately on surfacing, he lost consciousness and started to convulse. He was immediately dragged into the boat where he started to breathe normally and stopped fitting. He remained unconscious. 100% oxygen was administered. Within 90 minutes of surfacing he was transported 100 km by helicopter, flying at 25 m altitude, to the local recompression chamber. (Acute neurological DCI, probable CAGE)

CASE 2

A 39 year old tuna farm diver, who had been treated for DCI 3 weeks previously, presented with increasing malaise, excessive lethargy, inability to sleep, recurrence of his shoulder pain and slight paraesthesia in his left hand. These symptoms were similar to his initial presentation, but less intense. (Relapsing neurological and musculoskeletal DCI)

CASE 3

An experienced cave diver presented to your unit following a series of dives to 55 m on air. His last dive was 6 hours previously (with in water decompression using DCIEM tables). He phoned you complaining of difficulty with walking, weakness in his legs with paraesthesia and patchy loss of sensation in his legs. He had one episode of urinary incontinence. He was transported to your unit by ambulance on 100% O_2, with no significant relief of symptoms. On examination he had flaccid paralysis of both lower limbs and a palpable bladder. This was associated with loss of sensation to light touch and pin prick to a sensory level of T 8. There was nothing else significant in his dive profile or past history. (Progressive neurological DCI)

For the other two scenarios the questions were different.

CASE 4

You were contacted from a Pacific island 1,000 km from the nearest recompression facility. A 29 year old accountant, on holiday, presented to the resort doctor with severe pain in both shoulders and slight weakness in both legs following a wall dive to 30 m for 40 minutes. The diver felt the weakness was progressing. He had no travel insurance. (Acute progressive DCI without a chamber)

Q1 What would be your advice?
Q2 Would you consider in-water oxygen therapy?

CASE 5

A 45 year old phoned you for advice 2 days after a weekend of cray fishing. He undertook a series of 5 dives in 2 days at depths of no greater than 7 m; in-water time was approximately 70 minutes each dive. He complained of slight paraesthesiae in his hand, but felt otherwise well. His GP found no objective signs of DCI on examination, however a trial of 100% O_2 produced a slight decrease in his feeling of pins and needles in his hand. He was 6 hours from the nearest recompression facility. (Possible mild DCI)

Q1 Would you recommend HBO treatment?
Q2 What would be your initial treatment?

Results

A total of 24 questionnaires were sent out. Thirteen (54%) were returned completed. A further two responses were given as flow charts of departmental protocols, giving a total response rate of 63%. In some cases some respondents failed to answer all questions, while in other questions more than one response was indicated on the survey form.

Two units did not respond directly to the questions set, but forwarded their set protocols. One unit commences all patients on RN 62 table and either completed the RN 62 or went to a Comex 30 ($He:O_2$) + 50 m extension. Treatments were followed up by RN 61 or RN 62 tables.

The other unit categorised their patients into *Critically ill* (requiring CPR) treated with RN 63, *Severely ill* (unconscious or with severe CNS problems) given Comex 30 ($He:O_2$) and *Mild to moderately ill* treated with RN 62 or Comex 30. Follow up treatments would be either Comex 30, RN 62 or RN 61

Initial investigations

Investigations performed before recompression for cases 1-3 are shown in table 1. The initial investigations for DCI ranged from none to CT and MRI scanning. It is interesting to note that whereas in other branches of acute medicine, blood screening tests, e.g. complete blood count, electrolytes, liver function tests, cardiac enzymes and a coagulation screen, are often routinely performed, this is not the norm in this group of patients. While the cost-benefit analysis of such "routine" tests must be taken into account, objective evidence of disease severity and treatment progression needs to be identified. Recently it has been suggested that serum creatine kinase may define severity and possible prognosis in divers presenting with gas embolism.[1] Other markers of DCI, such as complement activation, platelet count, white cell count and activity, need further study in man for diagnostic, therapeutic and prognostic purposes. Divers need to be assessed in a similar fashion to other acutely ill patients. Full radiological and blood test investigations should be considered before they are subjected to potentially long periods of recompression. However as in other aspects of acute care, delays incurred by these investigations must be balanced against the urgency of recompression and the patient's condition.

Adjuvant therapy

Most respondents used intravenous fluid therapy, with other modalities such as lignocaine, non-steroidal analgesics, steroids, dextran and inotropes used less commonly (Table 2). Lignocaine as adjuvant therapy was considered by a large number of respondents both for use within the hyperbaric chamber and also as first line therapy if HBO was not immediately available. The benefits of lignocaine are well documented in animal studies and include reduction in neutrophil-endothelial adhesion, reduction in free oxygen radical release along with reducing intracranial hypertension.[2,3] Human data is small and confined to anecdotal case histories where it has been used in cases refractory to recompression therapy.[4] Prospective controlled studies are needed to assess the potential role of lignocaine in the treatment of DCI. Important questions to answer include dose response data, length of therapy and efficacy. Consideration also needs to be given to the possible role of lignocaine in association with hyperbaric oxygen and any effect it may have on CNS convulsive thresholds. The same comments can be aimed at the other forms of adjunctive therapy. Well constructed, randomised clinical studies have not been done to validate most of the adjunctive treatments in man.

Treatments

The initial recompression schedules used to treat cases 1-3 are shown in table 3. Table 4 defines the treatment tables used by the respondents. The reason for using the stated table was not specifically asked for, however it is likely that the choice is determined by several factors which include past experience along with current

TABLE 1

PRE-TREATMENT INVESTIGATIONS*

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Chest X-ray	7	Chest X-ray	1	Chest X-ray	5
CT scan	2	Cervical spine X-ray	2	CT scan	3
Full blood count	6	MRI scan	1	MRI scan	3
Arterial blood gas	2	Shoulder X-ray	2	Electrolytes	5
Electrocardiogram	2	Full blood count	5	Full blood count	5
		Electrolytes	3	Electrocardiogram	3
		Somatosensory evoked potentials	1	Spirometry	1

* Each reply may have had more than one response

TABLE 2

ADJUNCTIVE TREATMENT FOR DCI

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Intravenous fluids	13	Intravenous fluids	5	Intravenous fluids	11
Lignocaine	9	Lignocaine	5	Lignocaine	7
Steroids	1	NSAIDS	1	NSAIDS	2
Inotropes	1				
Dextran	1				

Each reply may have had more than one response
NSAIDS; non-steroidal anti-inflammatory agents

TABLE 3

TREATMENT SCHEDULES FOR DCI*

	Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
Initial	RN 62	9	RN 62	11	RN 62	12
	RN 63	2	18: 60: 30	1	30 m[a]	3
	Comex 30	2	14m[a]	1	50 m[a]	1
Fail	RN 62 (extended)	4	RN 62 (extended)	2	RN 62	4
	RN 63	4	Comex 30	1	30 m[a]	6
	30 m[a]	8	Continue with current table[b]	9	50 m[a]	1

* details of recompression schedules are described in Table 4
a Depth but not table stated
b In 9 cases, if the symptoms had not settled, the original table would be continued without alteration.

TABLE 4

DEFINITION OF TREATMENT TABLES.

Some respondents only gave depths rather than specific tables, therefore details of exact treatment schedules cannot be given.

RN 61

Royal Navy table 61 or United States Navy (USN) table 5. Maximum depth 18 m. 18 m for 45 minutes then decompress at 0.3 msw/minute to 9 m; 9 m for 25 minutes then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 2 hours 15 minutes.

RN 62

Royal Navy table 62 or USN table 6. Maximum depth 18 m. 18 m for 75 minutes, then ascend to 9 m at 0.3 msw/minute; 9 m for 135 minutes then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 4 hours 45 minutes.

RN 63

Royal Navy table 63 or USN table 6A. Maximum depth 50 m. 50 m for 30 minutes then decompress to 18 m at 8 msw/minute; 18 m for 75 mins then decompress at 0.3 msw/minute to 9 m; 9 m for 150 minutes then ascent to surface over 30 minutes. Breathing medium oxygen and air, however modifications using helium oxygen mixtures are used. Duration 5 hours 19 minutes.

18: 60: 30.

Maximum depth 18 m. 18 m for 60 minutes, then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 1 hour 30 minutes.

10: 90: 30.

Maximum depth 10 m. 10 m for 90 minutes, then 30 minutes ascent to surface. Breathing medium oxygen with air breaks. Duration 2 hours.

Comex 30

Maximum treatment depth 30 m. There are several variations on this table. An example of one is as follows: 30 m for 120 minutes, then decompress to 24 m over 35 minutes; 24 m for 35 minutes then decompress to 18 m over 35 minutes; 18 m for 95 minutes then decompress to 12 m over 35 minutes; 12 m for 185 minutes then ascent to surface over 25 minutes. Breathing medium 50-50 helium-oxygen or nitrogen-oxygen and oxygen with air breaks. Duration 7 hours 15 minutes.

There are many ways of measuring (expressing) chamber pressure. All involve a gauge which measures the pressure above atmospheric. These may be expressed as depth, feet of sea water (fsw), metres of sea water (msw)(which have been used in this paper) or as pressures, pounds per square inch (psi) (seldom used in Australasia), bar (equal to 10 msw), Pascals (Pa = Newton x m^2) or multiples of the Pascal such as kilopascal (kPa) and megapascal (MPa). KPa are convenient as they are used in measuring medical gas supplies. Msw and fsw should not be used for expressing partial pressures of gases.

Users of any measuring system must remember to add atmospheric pressure to the chamber pressure before trying to calculate actual gas pressures. This is difficult using msw so the conversion table below is provided for readers to work things out for themselves in more familiar units.

Depth to pressure conversions.

Depth	Pressures	
msw	bar	kPa
At surface	1.0	100
9	1.9	190
10	2.0	200
12	2.2	220
18	2.8	280
24	3.4	340
30	4.0	400
50	6.0	600

research data. There is at present debate as to the effectiveness of the US Navy schedule, with long term problems being reported despite initial treatment success.[5] Similarly the use of helium as part of the recompression schedule may have a place in clinical practice based upon data from Hyldegaard[6] along with clinical data in man with spinal cord DCI.[7] Interim data from the Royal New Zealand Navy heliox study, whilst showing a reduction of initial recompression treatments and cost saving, failed to show significant clinical benefit of heliox over oxygen.[8] Finally, saturation treatments have been used for the initial treatment and for those refractory cases of DCI, however again there are no controlled trials of efficacy.

Recompression schedules used for follow up treatments were even more variable than those used for initial therapy (Table 5). Nine replies indicated that recompression therapy would continue until the symptoms has failed to improve or resolved plus one additional treatment i.e. plateau + 1. Two units continued for a plateau + 2 while one reply indicated a set protocol was used, but did not state the details.

TABLE 5

FOLLOW-UP TREATMENTS FOR DCI

Case 1 Acute neurological DCI probable CAGE		**Case 2** Relapsing neurological and musculoskeletal DCI		**Case 3** Progressive neurological DCI	
RN 62	3	RN 61	1	RN 62	2
RN 61	3	18: 60: 30	2	RN 61	3
18: 60: 30	2	14 m[a]	1	18: 60: 30	3
14 m[a]	1	10 m[a]	6	14 m[a]	1
10 m[a]	4	RN 61 + 10 m x 3	1	10 m[a]	4

[a] Depth but not table stated

There appears to be no consensus on the appropriate oxygen dose (partial pressure, duration or frequency) of follow up treatment tables, although most units would continue treatment in the acute phase up to either resolution or plateauing of symptoms. In one of the few studies looking at follow up treatments in cases of DCI, Wilson suggested in a retrospective study that there were fewer recurrences of DCI in divers treated at 2.8 ATA compared to 2.4 ATA.[9] This needs to be repeated in a controlled prospective study.

Patients are increasingly demanding to know the rationale for treatment in all branches of medicine. Hyperbaric medicine needs to be able to provide hard data on which to base treatment plans. This uniformity of treatment would also help reduce the possibility of insurance and other funding debating management. Moreover in these days of Casemix, DRGs and fiscal constraints, there is a real need to justify and support rationales for different therapeutic modalities.

In-water oxygen therapy

The advice given to case 4 included 100% surface oxygen (10), intravenous fluids (10) and the need for retrieval (9). Lignocaine was advocated for use by 2 respondents. Specific problems of in-water recompression were mentioned in some replies. These included, "symptoms of DCI may worsen in water", "in-water oxygen convulsions" "divers have died using in-water oxygen" and "unable to evaluate a diver in the water". The major problem seen by the respondents to this survey was the danger of in-water convulsions leading to barotrauma, gas embolism and death. Other problems included the inability to adequately clinically evaluate divers underwater. Perhaps the view that, for the trained specialist with technical and resuscitative skills who has performed the exercise before, it may be a possible temporising measure until definitive treatment is available, is too restrictive.

While in-water decompression on oxygen has been shown to be safe in over 18,000 dives when breathed at depths between 3 and 6 m, its use at deeper depths for in-water recompression is not supported.[10-12] Indeed at the 1994 UHMS meeting in Denver, a panel of four diving medical specialist all agreed that they would seriously consider the use of in-water oxygen if the clinical condition warranted it. Experience in navies and off shore commercial diving has shown that immediate treatment gets better results than treatment delayed by an hour or two. In water oxygen therapy has a simple protocol, which can be carried out wherever there is 6 m of protected water. It is an emergency treatment approved for use when appropriate by the Royal Australian Navy (RAN) and the United States Navy (USN).[12] The patient is conscious and accompanied by an attendant diver at all times. He or she can assess changes in their own condition. Case 4 had progressive neurological DCI and many hours to wait for evacuation, just the case that naval diving manuals suggest should be treated by underwater oxygen at 6 m. However respondents in the survey preferred 100% surface oxygen and intravenous fluid hydration.

Overseas retrieval

Diving incidents overseas can be financially disastrous for those uninsured. As an example, from a Diving Emergency Service (DES) call in 1994, the cost of a retrieval from Fiji to Melbourne was costed in the vicinity of A$40,000. It is also necessary that divers are adequately insured so that retrieval can be an easily carried out possibility. Severe DCI requires rapid assessment by a doctor knowledgeable in diving related problems and

subsequent retrieval, if necessary to a recompression facility. This type of retrieval however is not without risk as was shown in 1995 over the South Pacific when a plane retrieving two divers crashed, killing all aboard.

Late vague symptoms

The problem of a diver who has vague neurological symptoms a few days after a dive is not uncommon. 10 respondents elected to treat the diver with a RN 62, with one considering taking him to 50 m if the symptoms failed to resolve. The question to treat is often tempered by the distance factor; i.e should one suggest that a diver with a mild ache and paraesthesiae in his wrist which is not worrying, and possibly getting better, travel hundreds of kilometres to the nearest recompression facility. Does the present data support such advice? That question is still unanswered.

Discussion

The survey was intended to obtain a snapshot of how the various hyperbaric units in Australasia treat various types of decompression illness. Although the response rate was only 63%, there was at least one reply from each unit in Australia and New Zealand, therefore the survey does represent current practice in this area. There is wide variation in the assessment, preliminary investigations, adjunctive therapy and treatment tables used for DCI.

While the results from conventional recompression therapies may be variable, there is great difficulty in comparing the results from different units due to the difference in treatment regimens. Australia and New Zealand are in a unique position, both geographically and because of the small number of doctors involved in hyperbaric medicine, to foster the collaboration of the various hyperbaric units in the quest for more rational and uniform treatment profiles. Groups such as the South Pacific Underwater Medicine Society (SPUMS) or the Australian and New Zealand Hyperbaric Medicine Group (ANZHMG) could provide a platform on which to base consensus statements on policies regarding assessment, investigation, treatment profiles and follow up management.

There is already established a national database for decompression illness in the United Kingdom. A larger prospective study looking at the incidence and treatment of DCI is being planned in Europe under the guidance of Dr Henrik Stanstrup. The aim is to collect multi-centre data from all hyperbaric units in Europe, with the goal of providing a valid database for future research studies. This European study hopes to submit data on 1,000 incidents of DCI annually. The numbers from Australia are approximately 300-400 per year. If such data could be collated and interpreted in a uniform and consistent way, perhaps some of the uncertainties may become clearer.

Acknowledgments

I would like to thank all of those members of the Hyperbaric Units in Australia and New Zealand who completed and returned the survey forms.

References

1 Smith RM and Neuman TS. Elevation of creatine kinase in divers with arterial gas embolism. *N Engl J Med* 1994; 330: 19-24

2 Peck S, Johnston RB, Johnston JR and Horowitz LD. Reduced neutrophil superoxide anion release after prolonged infusions of lidocaine. *J Pharm Exp Ther* 1985; 235: 418-422

3 Donegan MF and Bedford RF. Intravenously administered lidocaine prevents intracranial hypertension during endotracheal suctioning. *Anesthesiology* 1980; 52: 516-518

4 Drewry A and Gorman DF. Lidocaine as an adjunct to hyperbaric therapy in decompression illness: a case report. *Undersea Biomed Res* 1992; 19 (3): 187-190

5 Sutherland A, Veale A and Gorman DF. Neuropsychological problems in 25 recreational divers one year after treatment for decompression illness. *SPUMS J* 1993; 23 (1): 7-11

6 Hyldegaard O, Moller M and Madsen J. Protective effect of oxygen and heliox breathing during development of spinal decompression sickness. *Undersea Hyperbaric Med* 1994; 21 (2): 115-128

7 Kol S, Adir Y, Gordon CR and Melamed Y. Oxy-helium treatment of severe spinal decompression sickness after air diving. *Undersea Hyperbaric Med* 1993; 20 (2): 147-154

8 Drewry A and Gorman, DF. A progress report on the prospective, randomised double-blind controlled study of oxygen and oxygen-helium in the treatment of air-diving decompression illness. *Undersea Hyperbaric Med* 1994; 21 (Suppl): 98

9 Wilson M, Scheinkestel CD and Tuxen DV. Comparison of 14 and 18 metre tables on the resolution rate of decompression sickness (DCS) in divers. *Undersea Biomed Res* 1989; 16 (Suppl): 87-88

10 Fife CE, Pollard GW, Mebane GY, Boso AE and Vann RD. A database of open water, compressed air, multi-day, repetitive dives to depths between 100 and 190 fsw. In *Proc. Repetitive Diving. AAUS Diving Safety Publication.* Lang MA and Vann RD. Eds. Costa Mesa, California: American Academy of Underwater Sciences, 1992; 45-54

11 Imbert J and Bontoux M. Production of procedures: COMEX. In *Decompression in surface based d iving. 36th UHMS Workshop. UHMS publication 73*. Nashimoto I and Lanphier EH. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987; 90-100
12 Edmonds C. Underwater treatment of decompression sickness. *SPUMS J* 1995; 25 (3): 122-128

Key words
DCI, hyperbaric facilities, investigations, tables, treatment.

Data from this survey was presented at the XXIst Annual Meeting of EUBS. The abstract is published in Proceedings of the XXIst Annual Meeting of European Underwater and Biomedical Society held in Helsinki, Finland, June 28th-July 1st 1995. Editors Seppo A Sipinen and Matti Leinio. Helsinki, Finland: Finnish Society of Diving and Hyperbaric Medicine, 1995; 197.

Dr. M.T. Kluger's address is Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000

THE VALSALVA MANOEUVRE A CRITICAL REVIEW

David Taylor

Abstract

The Valsalva manoeuvre is commonly used in diving to equalise middle ear pressures during descent. A forceful expiration with the nostrils and mouth held shut results in an increased nasopharyngeal pressure and opening of the Eustachian tubes. The correctly performed manoeuvre is easily taught, effective and usually without complications.

When performed incorrectly, prolonged periods of raised intrathoracic pressure may lead to decreased venous return, decreased arterial pressure and increased pressures within the superior and inferior vena cavae. An intact autonomic nervous system will initiate compensatory cardiovascular reflexes. The manoeuvre has clinical and research uses which rely on induced physiological changes and the initiation of reflex responses. The physiology and clinical uses of the manoeuvre are discussed.

The inappropriate use of the manoeuvre has been associated with significant morbidity. This includes pulmonary and aural barotrauma, hyper- and hypotension, cardiac arrhythmias, arterial and venous haemorrhage, gastric reflux and stress incontinence. The complications of the manoeuvre are discussed.

Introduction

The Valsalva manoeuvre was first described in 1704 by the Italian physician Anton Maria Valsalva (1666-1723) as forced expiration against a closed glottis. For divers it is the process of making a forceful attempt at expiration while holding the nostrils closed and keeping the mouth shut for the purpose of adjusting middle ear pressure.

For many divers the manoeuvre is their only means of equalising middle ear pressures upon descent. However, it is not without its dangers and the inappropriate use of the manoeuvre has been associated with significant morbidity and even mortality. The purpose of this review is to summarise the physiological changes which occur during a forceful Valsalva manoeuvre and to describe some of the documented dangers and complications.

To put this it into its proper perspective, the vast majority of manoeuvres are performed correctly and without the generation of intrathoracic or intravascular pressures likely to lead to complications.

Physiology

A standardised Valsalva manoeuvre has been described and is divided into four phases.[1] The patient is requested to exhale against a resistance of 40 mmHg for 20 seconds while heart rate and blood pressure are monitored. During Phase 1 of the manoeuvre, the increase in intrathoracic and intra-abdominal pressure will cause aortic compression and an increase in peripheral resistance resulting in a transient increase in blood pressure.

The increase in intrathoracic pressure during Phase 2 (maintenance or strain phase) hinders venous return to the heart and pressures in the superior and inferior vena cavae are increased.[1-3] The decrease in venous return leads to a decrease in ventricular end-diastolic volume, cardiac output and consequently systolic arterial pressure.[1,3-7] This fall in arterial pressure is detected by baroreceptors in the carotid artery sinuses and results in a decrease in afferent nervous discharge from the sinuses to the brain stem via Herring's and then the glossopharyngeal nerves. The glossopharyngeal nerves relay in the nucleus tractus solitarius and the decrease in their rate of discharge has an inhibitory effect upon the vagus nerve centre (parasympathetic) and an excitatory effect upon the vasomotor centre (sympathetic). This results in a reflex tachycardia and peripheral vasoconstriction after about seven seconds of strain.[1,3] The increased pressures within the vena cavae are transmitted in a retrograde fashion along

the venous blood column. This results in raised venous pressures especially within the head and neck, genitalia and the limbs.

Phase 3 (release phase) begins with the cessation of forced expiration and leads to a further decrease in blood pressure as the pressure surrounding the aorta decreases. During Phase 4 (overshoot phase), the accumulated venous blood is pumped by the heart into the constricted vascular bed causing an "overshoot" of arterial pressure above the normal level.[1,3,5,6] This is detected by the carotid sinuses and results in a reflex bradycardia, mediated by the pathway described above. Ultimately, with the return of normal respiration and intrathoracic and intra-abdominal pressures, arterial and venous pressures and pulse rate return to baseline levels.

Clinical uses

The Valsalva manoeuvre is familiar to all those who have undertaken scuba diving where it is commonly used in the equalisation of middle ear pressures during descent. The manoeuvre increases air pressure in the nasopharynx which separates the Eustachian tube cushions and forces air into the middle ear. The pressures required to do this vary from 20 to 100 cmH_2O.[8] As the glottis must be open to allow transmission of the increased intrathoracic pressure to the nasopharynx, the mouth and nares need to be held shut in order to prevent the escape of air pressure. The manoeuvre is frequently used in other situations where the ambient atmospheric pressure changes rapidly e.g. descent when flying.

It is generally accepted that the Valsalva manoeuvre should be used in the examination of Eustachian tube function as part of the pre-diving medical examination. Indeed, Moser and Wolf described the Valsalva manoeuvre, with otoscopic visualisation of the excursion of the eardrum, as the most reliable test of tubal function.[9] However, while this manoeuvre is simple and accessible to the examining physician, it can prove frustrating if eardrum excursion is not demonstrated, particularly if the history from the examinee suggests no difficulty in the equalisation of middle ear pressures during ambient pressure change. In this situation the physician has not been able to demonstrate Eustachian tube function objectively and may elect to try other manoeuvres, re-examine at a latter date or refer the examinee for specialist ear, nose and throat examination. Some physicians will rely solely upon the history in the assessment of Eustachian tube function. While this approach may be considered unacceptable by some, others consider it as a practical necessity and draw attention to the work of McNicholl who concluded that immobility of the tympanic membrane on performance of the Valsalva manoeuvre does not necessarily denote the presence of Eustachian tube dysfunction.[10]

Among the most common uses of the Valsalva manoeuvre in clinical medicine is in the management of paroxysmal supraventriclar taccycardia. Upon termination of a prolonged and forceful manoeuvre, the vagally-induced reflex bradycardia seen during phase 4 is accompanied by decreased conductance through the atrio-ventricular node which often is sufficient to interrupt and terminate the circus movement responsible for the arrhythmia.[11,12]

The Valsalva manoeuvre can be used to test both the sympathetic and parasympathetic divisions of the autonomic nervous system. With sympathetic dysfunction, the fall in blood pressure occurring during phase 2 may not be followed by reflex tachycardia or vasoconstriction.[1] Consequently, the blood pressure increase at the start of phase 4 will be attenuated. With parasympathetic dysfunction, the baroreceptor-mediated reflex bradycardia response to the elevated blood pressure in phase 4 will not occur.[1] Accordingly, the manoeuvre is useful in the investigation and quantification of generalised autonomic failure, autonomic neuropathies, distal small-fibre neuropathy and adrenergic function.[13-16] It is of particular importance in the assessment of cardiovascular and anaesthetic risk in diabetic patients.[17,18]

Other uses for the manoeuvre in clinical practice rely on venous and intracardiac pressure changes. They include the evaluation of cardiac murmurs and left ventricular function,[16] diagnosis of congestive cardiac failure,[19] facilitation of insertion of central venous catheters,[20] enhancement of lower limb colour Doppler flow imaging[21] and the ultrasonographic detection of biliary obstruction.[22] Furthermore, the manoeuvre is being used clinically in the investigation of atrial septal defects (ASD) and patent foramen ovale (PFO) where the intrathoracic pressure changes lead to a transient inversion (right atrial pressure higher than left atrial pressure) of the inter atrial pressure gradient. This leads to flow of blood from the right atrium (venous blood) directly into the left atrium and into the arterial circulation thereby bypassing the lungs. This arterialised venous blood can be detected using contrast transthoracic and transoesophageal echocardiography,[23-25] oxygen saturation step-up analysis[25] and transcranial Doppler sonography after galactose or saline microbubble injection.[26,27]

A modified Valsalva manoeuvre is often used to induce changes in intraocular pressure and amplitude of tonography pulse pressure which aid in the diagnosis of assorted ocular disorders.[28]

Complications

Complications associated with the Valsalva manoeuvre are usually only seen when it is performed either too forcefully or for too long a period. These

complications are a consequence of the exaggerated physiological changes which occur, in particular the rises in intravascular, intrathoracic (pulmonary) and intra-abdominal pressures and the vascular reflexes.

Not surprisingly, the exaggerated cardiovascular reflexes induced by a forceful and prolonged manoeuvre have been associated with significant morbidity and even mortality. Most of the observed complications are consequent upon the induced hypertensive reflex. The hypertension induced by resistance exercise has been shown to be exaggerated by the accompanying Valsalva manoeuvre[29] and even symptomatic severe paroxysmal hypertension has been attributed to subconscious Valsalva-like manoeuvres.[30] Not all symptoms arise from induced hypertension and significant hypotension can be induced during the strain phase of the manoeuvre (phase 2) as a consequence of greatly diminished venous return.[31]

In subjects with fixed stenoses of the coronary vasculature, where myocardial blood flow is compromised, it is conceivable that myocardial ischaemia may be precipitated by either hypotension during the Valsalva strain phase or an increase in the myocardial oxygen demand/supply ratio caused by the hypertension induced upon release. However, it is unlikely that these symptoms would by seen without either repeated and prolonged manoeuvres or associated physical activity. Kern et al., in a study of subjects with normal coronary arteries, showed that despite a marked reduction in the mean arterial pressure during the Valsalva strain phase, the reduction of coronary blood flow velocity was not significant.[4]

Interest has recently been paid to the dangers of an ASD or PFO in scuba diving. These lesions may result in paradoxical gas embolization and serious morbidity.[32] This situation may occur in a diver suffering decompression sickness where venous bubbles pass into the arterial circulation during the transient reversal of blood flow through an ASD or PFO following a Valsalva manoeuvre. Wilmshurst et al., in a study of divers with a large PFO, have suggested that the foramen may be associated with clinically significant arterial desaturation and unusual responses of heart rate and blood pressure after the performance of a Valsalva manoeuvre.[33]

The Valsalva manoeuvre has been associated with cardiac arrhythmias and rarely even sudden death.[34-36] Piha et al. assessed the possible dysrhythmogenic effect of cardiovascular autonomic function tests, including the Valsalva manoeuvre, and documented the precipitation of ventricular extrasystoles and non-sustained tachycardia, conduction block and atrial fibrillation.[35] While the precipitation of some arrhythmias is likely to be associated with significant alterations in vagal tone, Taggart et al. have suggested that Valsalva-induced changes in ventricular loading influence cardiac repolarisation.[36] They found that when ventricular wall motion was abnormal the effects on regional endocardial repolarisation were often opposite in direction to those when motion was normal. Thus, regional differences in wall motion could generate local electrophysiological inhomogeneity which may be relevant to the association of arrhythmia with impaired left ventricular function.

Most neurological symptoms associated with the Valsalva manoeuvre are the result of the drop in mean arterial blood pressure during the strain phase. Cough and defaecation syncope are well-described syndromes secondary to Valsalva-like manoeuvres.[37,38] Significant decreases in cerebral perfusion pressures have been demonstrated clinically during the strain phase[6,7] and, while a modest but significant decrease in vascular resistance has been demonstrated during this phase, it is not rapid enough or of sufficient magnitude to maintain constant cerebral perfusion.[6] Significant blood pressure rises following the release phase of the Valsalva manoeuvre can produce rapid shifts in cerebrovascular blood flow which have been implicated in cerebral aneurysm rupture and rebleeding.[39,40] Valsalva-induced fluctuations in cerebrospinal fluid pressure around an aneurysm may also play a part in its rupture.[40] The Valsalva-like manoeuvre of nose blowing is thought to be associated with some cases of spontaneous rhinorrhoea. The presence of congenital dehiscences and the formation of small meningoceles which rupture during nose blowing is thought to be the aetiological basis.[41]

While the increase in intrathoracic pressure during the strain phase of the Valsalva manoeuvre has been shown to decrease pulmonary venous flow,[42] airways mucosal blood flow[43] and the size of the upper airways,[44] perhaps the most important respiratory complications result from barotrauma. There have been many reports describing Valsalva-induced alveolar rupture with consequent pneumothorax, pneumomediastinum, pneumopericardium and subcutaneous emphysema.[45-50] Curiously, pulmonary barotrauma has been reported following the smoking of marijuana and cocaine and it is considered that the Valsalva manoeuvre, often performed in an attempt to increase the effects of these drugs, has led to the rupture of marginal alveoli. The concurrent use of tobacco with these drugs is thought to accelerate lung disease and the likelihood of pulmonary barotrauma.[49,50]

Damage to the middle and inner ear following a forceful Valsalva manoeuvre during a diving descent has been well documented.[8,51] There are three postulated mechanisms for labyrinthine round window fistulae during diving. Firstly, sudden descent without volume equalisation forces the eardrum in and the force is tranmitted through the ossicles to the stapes which raises the perilymph pressure sufficiently to rupture the round window. Secondly, the sudden clearance of a blocked Eustachian tube allows the transmission of the high nasopharyngeal pressures into the middle ear chamber, driving the

tympanic membrane outwards and jerking the stapes outwards. As the inner ear is effectively a closed system, if the movement is large enough the round window is subjected to an inwards pull beyond the elastic limits of its membrane leading to a tear. This is described as implosive rupture of the round window in the ENT literature. The third possibility is a rupture (described as explosive in the ENT literature) caused by the transmission of a pressure wave in the cerebrospinal fluid through a widely patent cochlea aqueduct during a forceful manoeuvre.[8,52] Round window fistulae, oval window fistulae, probably due to damage from the stapes footplate, cochlear and vestibular haemorrhage and internal inner ear membrane ruptures have all been reported after forceful Valsalva manoeuvres.[8]

The term "Valsalva haemorrhagic retinopathy" refers to haemorrhage in and around the macula in response to a sudden rise in intrathoracic or intra-abdominal pressure during the Valsalva strain phase. The increased intravenous pressure is felt to be transmitted to the retinal circulation with resulting retinal capillary rupture.[53] Numerous reports of ocular haemorrhage have been made including retinal artery macro-aneurysms, suprachoroidal, preretinal, retinal and vitreous haemorrhages, cilio-choroidal detachments and haematomata of the lids.[53-57]

Complications of the Valsalva manoeuvre involving the gastrointestinal and genito-urinary tracts are uncommon and are the result of the increased intra-abdominal pressures which are generated. The manoeuvre has long been associated with gastroesophageal reflux and stress incontinence of urine and more recently it has been reported that reversal of scrotal vein blood flow may occur leading to the development of scrotal varicoceles or even their rupture.[58-60]

Summary

It is appropriate that diving physicians understand the physiology and pathophysiology of the Valsalva manoeuvre as this knowledge is relevant to their clinical practice. Diving instructors must also be cognizant of the dangers of the manoeuvre and must instruct their students in its correct and appropriate use.

Whilst the Valsalva manoeuvre will remain as the most commonly used means of equalising middle ear pressures, consideration should be given to safe alternatives. The "Frenzel manoeuvre" is a technique which involves closing the mouth and nose, both externally and internally (this is achieved by closing of the glottis) and then contracting the muscles of the floor of the mouth and the pharyngeal constrictors. Thus, the nose, mouth and glottis are closed and the elevated tongue can be used as a piston to compress the air trapped in the nasopharynx and force it into the Eustachian tube. Pressure of less than 10 cm H_2O are required to achieve this manoeuvre.[8] This technique has the advantage of avoiding large changes in intrathoracic and intra-abdominal pressures and the consequent physiological changes and reflexes. It also avoids rapid increases in middle ear pressures with the sudden opening of one or both Eustachian tubes. While this technique is more difficult to teach, it is often performed subconsciously by the more experienced diver or learned by novice divers as a modification of a "deliberate swallow", itself a safe and often effective technique used to equalise middle ear pressures. Yet another technique is to hold one's nose, shut one's mouth and then blow gently and swallow while blowing.

It has been shown that a prolonged or forceful Valsalva manoeuvre is accompanied by complicated and significant physiological change and that many complications of the manoeuvre have been documented. However, it needs to be emphasised that complications of the manoeuvre are rare and that the correct use of the manoeuvre by the sports diver is both safe and effective. It is unlikely that any diver would suffer from a complication of the manoeuvre if it were performed for very short durations and if forceful attempts were avoided.

References

1 Stein JH. *Internal Medicine. 3rd Edition*. Boston, Toronto, London: Little, Brown and Company, 1990; 1901-1902

2 Gindea AJ, Slater J and Kronzon I. Doppler echocardiographic flow velocity measurements in the superior vena cava during the Valsalva maneuver in normal subjects. *Am J Cardiol* 1990; 65: 1387-1391

3 Porth CJ, Bamrah VS, Tristani FE and Smith JJ. The Valsalva maneuver: mechanisms and clinical implications. *Heart Lung* 1984; 13: 507-518

4 Kern MJ, Gudipati C, Tatineni S, Aguirre F, Serota H and Deligonal U. Effect of abruptly increased intrathoracic pressure on coronary blood flow velocity in patients. *Am Heart J* 1990; 119: 863-870

5 Tsai LM and Chen JH. Abnormal haemodynamic response to Valsalva maneuver in patients with atrial septal defects evaluated by Doppler echocardiography. *Chest* 1990; 98: 1175-1178

6 Greenfield JC, Rembert JC and Tindall GT. Transient changes in cerebral vascular resistance during the Valsalva maneuvre in man. *Stroke* 1984; 15: 76-79

7 Welding W, Sadel S, Jimendez D, Rosenwasser R and Buchheit W. Cardiovascular and cerebrovascular effects of the applied Valsalva manoeuvre in anaesthetized neurosurgical patients. *Eur J Anaesthesiol* 1994; 11: 81-87

8 Edmonds C, Lowry C and Pennefather J. *Diving and subaquatic medicine.2nd edition*. Sydney: Diving Medical Centre, 1984

9 Moser M and Wolf G. Recurrent sinusitis and impairment of Eustatian tube function in air passengers and crew. *Aviat Space Environ Med* 1990; 61: 662-665

10 McNicoll WD. Tympanic membrane mobility on otoscopy as an indicator of Eustachian function. *Laryngoscope* 1983; 93: 630-634

11 Ornato JP. Management of paroxysmal supraventriclar tachycardia. *Circulation* 1986; 74 (6 pt 2): IV108-110

12 Chance JF, Warner JG and Elsawy T. Augmented Valsalva manoeuvre for supraventricular tachycardia in the young. *Ann Int Med* 1992; 116 (6): 523

13 Low PA. Autonomic nervous system function. *J Clin Neurophysiol* 1993; 10: 14-27

14 Low PA. Composite autonomic scoring scale for laboratory quantification of generalized autonomic failure. *Mayo Clin Proc* 1993; 68: 748-752

15 Low PA. Evaluation of autonomic function. *Curr Opin Neurol Neurosurg* 1992; 5: 461-463

16 Nishimura RA and Tajik AJ. The Valsalva manoeuvre and response revisited. *Mayo Clin Proc* 1986; 61: 211-217

17 Maser RE, Pfeifer MA, Dorman JS, Kuller LH, Becker DJ and Orchard TJ. Diabetic autonomic neuropathy and cardiovascular risk. Pittsburgh epidemiology of diabetes complications study III. *Arch Intern Med* 1990; 150: 1218-1222

18 Linstedt U, Jaeger H and Petry A. The neuropathy of the autonomic nervous system. An additional anaesthetic risk in diabetes mellitus. *Anaesthetist* 1993; 42: 521-527

19 Marantz PR, Kaplan MC and Alderman MH. Clinical diagnosis of congestive heart failure in patients with acute dyspnoea. *Chest* 1990; 97: 776-781

20 Armstrong PJ, Sutherland R and Scott DH. The effect of position and different manoeuvres on internal jugular vein diameter size. *Acta Anaesthesiol Scand* 1994; 38: 229-231

21 Gao J, Kazam E, Rubenstein W, Whalen JP and Hom T. The use of deep inspiration preceeding the Valsalva manoeuvre and rapid expiration in colour Doppler imaging of the lower extremity veins. *Clin Imaging* 1993; 17: 266-268

22 Quinn RJ, Meredith C and Slade L. The effect of the Valsalva manoeuvre on the diameter of the common bile duct in extrahepatic biliary obstruction. *J Ultrasound Med* 1992; 11: 143-145

23 Chen WJ, Kuan P, Lien WP and Lin FY. Detection of patent foramen ovale by contrast transoesophageal echocardiography. *Chest* 1992; 101: 1515-1520

24 Di Tullio M, Sacco RL, Venketasubramanian N, Sherman D, Mohr JP and Homma S. Comparison of diagnostic techniques for the detection of a patent foramen ovale in stroke patients. *Stroke* 1993; 24: 1020-1024

25 Ferguson JJ 3d, Miller MJ, Aroesty JM, Sahagian P, Grossman W and McKay RG. Assessment of right atrial pressure-volume relations in patients with and without an atrial septal defect. *J Am Coll Cardiol* 1989; 13: 630-636.

26 Jauss M, Kaps M, Keberle M, Haberbosch W and Dorndorf W. A comparison of transesophageal echocardiography and transcranial Doppler sonography with contrast medium for detection of patent foramen ovale. *Stroke* 1994; 25: 1265-1267

27 Sullivan M. Patent foramen ovale (letter to the ¡Editor). *SPUMS J* 1994; 24: 206

28 Booth FM, Saad R and Tait SA. Tonography, Valsalva manoeuvre and central visual fields. A study of 429 patients. *Aust N Z J Ophthalmol* 1991; 19: 53-59

29 Linsenbardt ST, Thomas TR and Madsen RW. Effect of breathing techniques on blood pressure response to resistance exercise. *Br J Sports Med* 1992; 26: 97-100

30 Nielsen PE, Jorgensen F, Conway J and Sleight P. Severe paroxysmal hypertension induced by subconscious Valsalva-like manoeuvres. *Clin Auton Res* 1992; 2: 281-284

31 Kluger M. Acute severe hypotension during the Valsalva manoeuvre. *SPUMS J* 1993; 23: 156

32 Stoddard MF, Keedy DL and Dawkins PR. The cough test is superior to the Valsalva maneuver in the delineation of right-to-left shunting through a patent foramen ovale during contrast transesophageal echocardiography. *Am Heart J* 1993; 125: 185-189

33 Wilmshurst PT, Treacher DF, Crowther A and Smith SE. Effects of a patent foramen ovale on arterial saturation during exercise and on cardiovascular responses to deep breathing, Valsalva manoeuvre and passive tilt: relation to history of decompression illness in divers. *Br Heart J* 1994; 71: 229-231

34 Sperry K. Achalasia, the Valsalva maneuver and sudden death: a case report. *J Forensic Science* 1994; 39: 547-551

35 Piha SJ and Voipio-Pulkki LM. Cardiac dysrhythmias during cardiovascular autonomic reflex tests. *Clin Auton Res* 1993; 3: 183-187

36 Taggart P, Sutton P, John R, Lab M and Swanton H. Monophasic action potential recordings during acute changes in ventricular loading induced by the Valsalva manoeuvre. *Br Heart J* 1992; 67: 221-229

37 Desser KB, Harris CL and Benchimol A. Carotid blood velocity during cough studies in man. *Stroke* 1976; 7: 416-418

38 Broughy CM, Evans L and Sumpio BE. Defecation syncope secondary to functional inferior vena caval obstruction during a Valsalva maneuver. *Ann Vasc Surg* 1993; 7: 374-377

39 Therrien B. Position modifies carotid artery blood flow velocity during straining. *Res Nurs Health* 1990; 13 : 69-76.
40 Schievink WI, Karemaker JM, Hageman LM and van der Werf DJ. Circumstances surrounding aneurysmal subarachnoid haemorrhage. *Surg Neurol* 1989; 32: 266-272
41 Tolley NS. A clinical study of spontaneous CSF rhinorrhoea. *Rhinology* 1991; 29: 223-230
42 Meijburg HW, Visser CA, Westerhof PW, Kasteleyn I, van der Tweel I and Robles de Medina EO. Normal pulmonary venous flow characteristics as assessed by transoesophageal pulsed Doppler echocardiography. *J Am Soc Echocardiogr* 1992; 5: 588-597
43 Breitenbucher A, Chediak AD and Wanner A. Effect of lung volume and intrathoracic pressure on airway mucosal blood flow in man. *Respir Physiol* 1994; 96: 249-258
44 Stanford W, Galvin J and Rooholamini M. Effects of awake tidal breathing, swallowing, nasal breathing, oral breathing and the Muller and Valsalva maneuvers on the dimensions of the upper airway. Evaluation of ultrafast computerized tomography. *Chest* 1988; 94: 149-154
45 Panacek EA, Singer AJ, Sherman BW, Prescott A and Rutherford WF. Spontaneous pneumothorax: clinical and natural history. *Ann Emerg Med* 1992; 21: 1222-1227
46 Sands DE, Ledgerwood AM and Lucas CE. Pneumomediastinum on a clinical service. *Am Surg* 1988; 54: 434-437
47 Manco JC, Terra-Filho J and Silva GA. Pneumomediastinum, pneumothorax and subcutaneous emphysema following the measurement of maximal expiratory pressure in a normal subject. *Chest* 1990; 98: 1530-1532
48 Takahashi K et al. Two cases of idiopathic pneumopericardium. *Kokyu To Junkan* 1991; 39: 95-97
49 Feldman AL, Sullivan JT, Passero MA and Lewis DC. Pneumothorax in substance-abusing marijuana and tobacco smokers: three cases. *J Subst Abuse* 1993; 5: 183-186
50 Salzman GA, Khan F and Emory C. Pneumomediastinum after cocaine smoking. *South Med J* 1987; 80: 1427-1429
51 Sidi J and Tovi F. Traumatic labyrinthine fistulas. *Isr J Med Sci* 1979; 15: 156-158
52 Lamm K. Experimental defects of the round window membrane. *HNO* 1992: 40: 374-380
53 Avins LR and Krummenacher TK. Valsalva maculopathy due to a retinal arterial macroaneurism. *Ann Opthalmol* 1983; 15: 421-423
54 van Meurs JC and van den Bosch WA. Suprachoroidal haemorrhage following a Valsalva manoeuvre. *Arch Ophthalmol* 1993; 111: 1025-1026
55 Williams DF, Mieler WF and Williams GA. Posterior segment manifestations of ocular trauma. *Retina* 1990; 10 suppl 1: S35-44
56 Suan EP, Rubsamen PE and Byrne SF. Bilateral ciliochoroidal detatchments after Valsalva manoeuvre. *Arch Ophthalmol* 1993; 111: 304
57 Schipper I. Valsalva's manoeuvre: not always benign. *Klin Monatslb Augenheilkd* 1991; 198: 457-459
58 Thompson JK, Koehler RE and Richter JE. Detection of gastroesophageal reflux: value of barium studies compared with 24-hour pH monitoring. *AJR Am J Roentgenol* 1994; 162: 621-626
59 Eskew LA, Watson NE, Wolfman N, Becktold R, Scharling E and Jarow JP. Ultrasonographic diagnosis of varicoceles. *Fertil Steril* 1993; 60: 693-697
60 Aliabadi H and Cass AS. Nontraumatic rupture of varicocele. *Urology* 1987; 29: 421-422

Key Words
ENT, physiology, barotrauma.

Dr David McD Taylor, MD, FACEM, is Acting Director of Emergency Medicine, Ballarat Base Hospital, Victoria 3350, Australia

Dr Taylor's address for correspondence is 242 Myers Street, Geelong, Victoria, 3220, Australia. Telephone (Home) (052) 29 1552, (Work) (053) 20 4459. Fax (Work) (053) 31 8205.

THE WORLD AS IT IS

ACTION DOWN UNDER

Richard Moon

While most scholarly medical societies are organised by physicians. I recently attended a meeting of a society in which nurses and hyperbaric technicians have taken the lead. The September 1995 meeting of the Hyperbaric Technicians and Nurses Association (HTNA), with Dave King as President, superbly hosted by the Alfred Healthcare Group Hyperbaric Service in Melbourne, Australia, was a winner.

While the setting for the gathering was unsurpassed, it was more than matched by the content. Among many

others were presentations of several cases of decompression illness with clearly documented exacerbation with altitude after apparently successful treatment, use of hyperbaric oxygen (HBO) for necrotising wounds due to white-tail spider bites, a review of the Hyperbaric Incident Monitoring Study, report of decompression illness after technical diving and interim results of a randomised study of hyperbaric vs normobaric oxygen in the treatment of carbon monoxide poisoning.

The Australian and New Zealand Hyperbaric Medicine Group (ANZHMG), consisting of civilian and military hyperbaric physicians form the region, held an executive meeting immediately afterwards. The 1994-1995 Committee, chaired by Dr Harry Oxer of Fremantle, discussed several issues of mutual interest, including safety guidelines for administration of HBO for sport injuries and clinical trials. Organisation and promotion of clinical trials is a role which this body has decided to take on, with the worthy goal of one trial in each area of HBO and diving medicine to be initiated each year. It may be an advantage of a relatively small group of hyperbaricists, most of whom practice in or near a major teaching hospital, that nation-wide consensus and co-operation in multi-centre trials and safety issues may be readily achieved.

In Australia there are 8 civilian facilities and 2 fixed military chambers serving a population of 18.3 million people. This ratio is similar to the US and Canada, in which there are 215 chambers serving 292 million people. Statistics of cases treated in Australia were published in the Proceedings. In 1994-1995 there were 8,736 treatments of 1,044 patients. The conditions treated were familiar, but unlike American practice, Australian treatments are dominated by emergencies, which account for 77% of patients. In comparison, in 1993 only 40% of North American patients treated were for emergencies. The huge amount of recreational diving per capita, particularly in Queensland, is reflected by the fact that more than half of all emergency patients have decompression illness. The majority of Australians live in or near a major city and thus hyperbaric therapy is accessible to most of the population. The only major population centre without a local hyperbaric facility is Brisbane, in which there are plans to install one. The smaller number of non-emergency treatments suggests considerable scope for expansion of clinical services.

There was a general consensus that the HTNA is becoming the society representing the interests of hyperbaric oxygen treatment in Australia and New Zealand. Judging by this meeting the next HTNA meeting in Hobart, Tasmania, on August 29-31 1996, should be an excellent one.

Key Words

Hyperbaric facilities, meeting, treatment.

Reprinted, at the request of the HTNA, by kind permission of the Editor and UHMS, from Pressure, the newsletter of the Undersea and Hyperbaric Medical Society, 1995; 24 (6): 3.

Dr Richard Moon is President of the Undersea and Hyperbaric Medical Society. The Society's address is 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA

HYPERBARIC MEDICINE UNIT OPENS

The Hyperbaric Medicine Unit in Christchurch, New Zealand, became fully operational on February 27th 1996.

This restores the emergency hyperbaric service in the South Island that previously existed from 1979 to mid 1994 but with an enhanced capability now that the Unit is in the main Base Hospital for the Region.

The Unit is staffed medically from the Department of Anaesthesia, with Dr Michael Davis as Medical Director.

Canterbury Health CHE is only contracted to provide an emergency service for the Southern Regional Health Authority, as well as the treatment of a limited number of patients with osteoradionecrosis of the mandible.

We, therefore, still have some way to go to achieve a full hyperbaric medicine service in the geographically spread region of about one million people.

Daytime non-emergency contact is achieved as follows:

Hyperbaric Medicine Unit
Christchurch Hospital
Private Bag 4710
Christchurch, New Zealand

Phone
03-364-0045 (HMU) or 03 364 0288 (Anaesthesia)

Fax
03-364-0187 (HMU) or 03 364 0289 (Anaesthesia)

Emergency calls should be directed to the Christchurch Hospital telephone office 03-364-0640 or via the Diver Emergency Service, Auckland 09-445-8454.

SPUMS NOTICES

S0UTH 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 a financial member of the Society.

2 The candidate must supply documentary evidence of satisfactory completion of examined courses in both Basic and Advanced Hyperbaric and Diving Medicine at an institution approved by the Board of Censors of the Society.

3 The candidate must have completed at least six months full time, or equivalent part time, training in an approved Hyperbaric Medicine Unit.

4 All candidates will be required to advise the Board of Censors of their intended candidacy and to discuss the proposed subject matter of their thesis.

5 Having received prior approval of the subject matter by the Board of Censors, the candidate must submit a thesis, treatise or paper, in a form suitable for publication, for consideration by the Board of Censors.

Candidates are advised that preference will be given to papers reporting original basic or clinical research work. All clinical research material must be accompanied by documentary evidence of approval by an appropriate Ethics Committee.

Case reports may be acceptable provided they are thoroughly documented, the subject is extensively researched and is then discussed in depth. Reports of a single case will be deemed insufficient.

Review articles may be acceptable only if the review is of the world literature, it is thoroughly analysed and discussed and the subject matter has not received a similar review in recent times.

6 All successful thesis material becomes the property of the Society to be published as it deems fit.

7 The Board of Censors reserves the right to modify any of these requirements from time to time.

MINUTES OF THE EXECUTIVE COMMITTEE MEETING

held on the 25 November 1995 at the
Royal Adelaide Hospital Hyperbaric MedicineUnit
Adelaide

Opened 0945

Present

Drs D Gorman (President), A Slark (Past President), S Paton (Treasurer), C Meehan (Secretary), J Knight (Editor), G Williams (Public Officer), M Davis (NZ Chairperson), C Acott, R Walker and J Williamson (committee members).

Apologies

Dr Davies (Education Officer)

1 Minutes of the previous meetings

Committee minutes of 24 May 1995 be accepted as a true record. Proposed Dr Knight, seconded Dr Acott.

Committee minutes of 16 July 1995 be accepted as a true record. Proposed Dr Knight, seconded Dr Davis.

AGM 1995 motions and minutes read.

2 Business arising from the minutes:

2.1 Further research into new computers for the Treasurer and the Secretary suggested that a laptop (notebook) computer would prove more convenient and versatile. Purchase of such was agreed. Dr Gorman was to enquire as to the cost effectiveness of buying these in New Zealand.

2.2 North American chapter update on mailing and bank account. It was decided to close the bank account as it was now not needed as all the journals were sent Economy Air. The journals also go Economy Air New Zealand.

It was proposed that all overseas journals would in future be sent by Economy Air. Moved Dr Davis, seconded Dr Acott.

As some of the overseas journals had already been sent by surface mail, it was decided to send these members a conference booklet for the 1996 ASM in the Maldives by airmail in order for the information to arrive in time for them to book.

2.3 Fiji ASM 1995 finance finalising. This was again discussed and reinforced that the amount previously offered was the final settlement. Dr Paton to follow this up.

2.4 Maldives ASM 1996 update. The costing change between the tender document and the conference booklet was discussed. Dr Williams was to investigate the audit trail. Dr William's letter of the 16 October to Allways Dive Expedition was to be ignored and normal trade standards were to be used.

2.5 New Zealand ASM 1997 update. Dr Davis gave a comprehensive update on the proposed arrangements for the 1997 ASM in New Zealand. The theme of the conference is to be the Pathophysiology and Treatment of Decompression Illness, and the workshop is to formulate first aide guidelines for the management of a diving accident.

Dr Davis will write a short abstract on the proposed meeting for publication in the SPUMS Journal.

3 Treasurer's Report

Accepted. Moved Dr Knight, seconded Dr Acott.

A bank account is to be opened in New Zealand to help facilitate the expenses incurred in connection with the 1997 ASM.

4 Correspondence:

4.1 Uncover Company, 15 July 1995, was looked at. SPUMS does not require copyright royalties for republished material. No action needed.

4.2 American Medical Association, July 1995. This has been addressed by Dr Knight

4.3 National Water Safety Strategy. No action needed.

4.4 Letter from Dr Jim Marwood, June 1995, with motion for the 1996 AGM. This was discussed and the motion will be presented at the 1996 AGM. It was decided that the Gala Dinner should have more structure. It was proposed there would be a Master of Ceremonies who would be the convenor at the time, that the President would give a short after dinner speech including special thanks and acknowledgments, and that there may then be a hand full of special awards, including recognition of the best free paper.

4.5 J. L Publications, 11 November 1995. A flyer will go in the next journal advertising the book Scuba Safety in Australia at a reduced price.

4.6 PADI Australia, 8 August 1995.

4.7 Dr John Couper-Smart, 17 July 1995, offering to assist with the Journal. Passed to Dr Knight.

4.8 Dr Douglas Walker, 17 August 1995. Dr Walker to be notified that the matter is at present under active consideration.

5 Other Business:

5.1 Matters regarding indemnity policy recommended by Michael Gatehouse (Knight).

5.2 Operation Wallacea (Meehan).

5.3 Reprinting schedules for SPUMS Diving Medical, Statement of Purposes and Rules and forms (Meehan). Dr Knight and Dr Meehan to coordinate on this.

5.4 Ratification of minutes before printing in the Journal (Gorman).

5.5 Role of convenor to be defined and guidelines written. Dr Acott will address this.

5.6 It was proposed that an Ex-Presidents of SPUMS committee to be formed. Dr Gorman to put a motion to the 1996 AGM *about* this.

5.7 A representative from SPUMS is sought to be on a panel for the UHMS discussion on diving and diabetes. Dr Williamson proposed and accepted.

5.8 Dr Williamson gave an update on HTNA and ANZHMG activities.

5.9 Agenda for the 1996 AGM to be printed in the March journal. A call for motions to go into the December Journal.

6 SPUMS Business

6.1 All articles printed in other journals and attributed to SPUMS must have the author clearly identified.

6.2 Only the President and the Secretary have the authority to make a statement on behalf of SPUMS.

6.3 The North American Chapter to elect a new committee.

6.4 The honorarium for the Editor to be increased to $15,000 per annum. The duties of the editor are expanded to include mandatory attendance at the Annual Scientific Meeting and AGM.

Closed at 1430

AGENDA FOR THE ANNUAL GENERAL MEETING OF SPUMS

to be held on Paradise Island, Maldives,
Friday 26th April 1996

1 Minutes of the previous Meeting:

Minutes of the previous meeting have been published (SPUMS J 1995; 25 (3): 130-133).

Motion that the minutes be taken as read and are an accurate record:

2 Matters arising from the minutes

3 Annual Reports

3.1 President's Report

3.2 Secretary's Report

4 Financial Statement and Treasurer's Report

5 Subscription fees for the coming year

6 Election of office bearers

Nominations are as follows:

President: Dr Guy Williams

Secretary:	Dr Cathy Meehan
Treasurer:	Dr Robyn Walker
Editor:	Dr John Knight
Public Officer:	Dr Guy Williams
Education Officer:	Dr David Davies
Committee Members:	Dr Chris Acott
	Dr Roger Capps
	Dr Vanessa Haller
	Dr Michal Kluger
	Dr Douglas Walker

A postal ballot of financial members has been held to elect the three committee members. Votes will be counted at the AGM.

7 Appointment of the Auditor

8 Business of which notice has been given

8.1 That Mr John Lippmann be elected to Full Membership of SPUMS.

8.2 That at this and subsequent Annual General Meetings opportunity be given for members to raise matters of concern for which prior notice has not been given.
(Dr Jim Marwood)

8.3 The Committee proposes to bring the following 12 motions before the 1996 AGM of SPUMS.

** Proposed changes and corrections to the Rules of the Society. The stock of the Statement of Purposes and Rules of the Society is nearly exhausted and a reprinting is necessary. This is a good opportunity to correct typographical errors, the address of the Society and clarify some obscure wording in the current Rules as an amendment to the Rules is necessary to establish the Presidents' Committee (Motion 12).

** The proposal for a Presidents' Committee follows a submission from Dr Carl Edmonds. The proposal is analogous to a similar committee that serves the Undersea and Hyperbaric Medical Society. It is proposed by the current President of the Society, Dr Des Gorman, and has both the unanimous support of and is seconded by the extant Society Executive Committee. The past presidents of the Society are Carl Edmonds (Life Member), Ian Unsworth (no longer a member), John Knight (Life Member), Chris Lourey (Life Member), Chris Acott and Tony Slark

1 That typographical errors in Rules 7.2.1 (b), 7.2.3, 7.2.3.(b), 10 (f), 12 (a), 33 (a), 39 and in the heading of Meetings of Committee and resolutions of committee on page 15, be corrected.

2 An amendment to Rule 3 (e)

That the words "appointed on" be replaced by "appoint a". The amended Rule 3 (e) would then read (changed words in italics)

(e) Any corporate organisation in sympathy with the aims of the Association may be elected by the Committee as a corporate member of the Association and it may *appoint a* delegate to attend meetings of the Association.

3 An amendment to Rule 4 (a)

That the words "Australian College of Occupational Medicine, P.O.Box 2090, St Kilda West, Victoria 3182" be replaced by "Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004." The amended Rule 4 (a) would then read (changed words in italics).

(a) Any person seeking full membership or associate membership or corporate membership may apply by writing to SPUMS Membership *C/o Australian and New Zealand College of Anaesthetists, 630 St Kilda Road, Melbourne, Victoria 3004.*

4 An amendment to Rule 4 (c)

That the words "Editor (or the Secretary)" shall be replaced by the word "Secretary" and that the words "kept by him" be deleted. The amended Rule 4 (c) would then read (changed words in italics).

(c) Upon notification by the Treasurer that membership has commenced the *Secretary* shall enter the applicant's name in the register of members.

5 An amendment to Rule 4 (d) (ii)

That the word "his" shall be deleted. The amended Rule 4 (d) (ii) would then read.

(d) (ii) terminates upon the cessation of membership whether by death or resignation or non-payment of subscription or otherwise.

6 An amendment to Rule 4 (e)

That the words "Editor (or the Secretary)" shall be replaced by the word "Secretary". The amended Rule 4 (e) would then read (changed words in italics)

(e) The *Secretary* shall also inscribe the name of any life member or honorary member in the register of members and shall delete the name of any person ceasing to be a member from the register immediately after such person ceases to be a member.

7 An amendment to Rule 11 (a) (vii)

That "Rule 8 (d) (iii)" be added after the word "under". The amended Rule 11 (a) (vii) would then read (changed words in italics)

(a) (vii) Announcement of the newly elected Committee and the holding of any ballots necessary under *Rule 8 (d) (iii)*;

8 An amendment to Rule 16

That Rule 16 be deleted and replaced by the following words

16. A question arising at a general meeting of the Association shall be determined on a show of hands unless a poll is demanded. A declaration by the Chairman that a resolution has been carried or lost, and an entry to that effect in the Minute Book of the Association is evidence of that fact. Motions, other than those conferring membership, passed at general meetings shall have no effect until approved by the full membership. A notice of all motions passed shall appear in the next issue of the Journal. Approval by three fourths majority of the members, as specified by Rule 37, shall be assumed if no member informs the Secretary, in writing, of an objection to the motion or motions within one calendar month of the publication of that issue of the Journal. If an objection is received a postal ballot shall be held (Rule 20).

** This amendment is needed for Rule 16 to describe correctly the Society's long standing (20 years) practice dictated by the need to provide a democratic method of governing the Society. As the Annual General meeting has never been attended by more than 10% of the membership, the Committee has followed this procedure since 1976. So far no member has ever objected to the Secretary and no postal ballot has had to be held.

9 An amendment to Rule 17 (b)

That the words "specifying the member's intention in writing". The amended Rule 17 (b) will then read (changed words in italics)

(b) All votes shall be given personally or by proxy *specifying the member's intention in writing*.

10 An amendment to Rule 22 (c)

That the word "its" be deleted and replaced by "the Association's". The amended Rule 22 (c) would then read (changed words in italics)

(c) In the event of a casual vacancy in any office referred to in sub-clause (a), the Committee may appoint one of *the Association's* members entitled to vote to the vacant office and the member so appointed may continue in office up to and including the conclusion of the annual general meeting next following the date of that person's appointment.

11 An amendment to Rule 37

That Rule 37 be deleted and replaced by the following words

37. The Statement of Purposes and these Rules may only be altered, rescinded and/or added to in the following manner: by a three fourths majority of the full members and life members who, being entitled under the Rules so to do, vote in a postal ballot, if required, as specified in Rule 16.

12 Amendments to Rules 29, 41 and their headings.

That the words "branch" and "regional branch" be replaced by "Chapter". The amended Rules 29 and 41 would then read (changed words in italics)

***Chapters* to provide information**

29. Any *chapter* of the Association shall furnish to the Treasurer or the Committee, within a reasonable time, account of any financial transactions if requested by the Treasurer or the Committee to do so.

Chapters

41. (a) There shall be *chapters* of the Association for the purpose of organising meetings, field excursions and activities consistent with the objects of the Association. C*hapters* may charge members to cover costs. Each *chapter* shall maintain proper accounts.

(b) A *chapter* of the Association may be established at any place to further the objects of the Association in that place.

(c) Any person wishing to establish a *chapter* shall apply in writing to the Secretary who shall submit the application for approval by the Committee.

(d) Each *chapter* shall be directed by a chapter sub-committee of at least two members.

(e) Each *chapter* shall be governed by these Rules. The action of *chapters* shall be subject to the overriding authority of the Committee of the Association, which shall do everything to assist *chapters* in their operation.

(f) Should the Committee resolve that the activities or conduct of any *chapter* are not in accordance with the best interest of the Association, the Committee may withdraw its approval and the *chapter* shall cease to be a *chapter* of the Association forthwith. Such action shall be submitted for approval at the next annual general meeting of the Association.

(g) The records, accounts (and funds) of all *chapters* are the property of the Association and in the event that a *chapter* ceases to exist, the funds held by that *chapter* shall be forwarded to the Treasurer of the Association forthwith.

12 Proposed that the following new rule be added and that Rules 29-42 be renumbered.

Presidents' Committee

29 This standing committee will be composed of life- or ordinary members who have served at least one year as the President of the Society. The Committee will meet at the Annual Scientific Meeting of the Society, at the member's expense, and at the same time as the Executive Committee at one other time during the year, at the Society's expense. The Presidents' Committee will also be able to conduct telephone conferences. Chairmanship of the Committee will be the responsibility of the immediate past-President and minutes will be kept by members in rotation. The Presidents' Committee will answer directly to the current Society President and be responsible for the development of actual and draft Society policy on issues identified by the Society. The Presidents' Committee will report its activities in the Society Journal and provide an annual report to the Society at the Annual Scientific Meeting.

ANNUAL SCIENTIFIC MEETING
SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
Paradise Island, The Maldives
20th-28thApril 1996
"TECHNICAL DIVING"

Guest Speakers Professor David Elliott and Dr R.W. "Bill" Hamilton

Sunday 21st April 1996 1900 Official Opening

Monday 22nd April 1996

Opening Address

Scientific Meeting Day 1

Chairperson **Guy Williams**

Marine Envenomation C Acott

Changes in lung function in asthmatics during scuba diving. H Staunstrup

Technical Diving Workshop Part 1
Ethics

Chairperson **C Acott**

The ethics of risk taking and risk assessment. Des Gorman

Mental fitness for technical diving in sport scuba divers. S Schioberg-Schiegnitz

Historical aspects of Technical Diving: a personal view. Mike Davis

The scope of non-conventional recreational diving. Bill Hamilton

(includes the history of technical diving and what is happening now)

Tuesday 23rd April 1996

Scientific Meeting Day 2

Chairperson **Chris Acott**

Normobaric oxygen therapy in diving accidents. Jurg Wendling

Pain perception during scuba diving. K Kroner

Epidemic of decompression sickness in the Miskito Indians Tom Millington

Technical Diving Workshop Part 2
Gas toxicities

Chairperson **Des Gorman**

Oxygen toxicity. Dr Bill Hamilton

Deep water blackout. Prof. David Elliott

(includes carbon dioxide toxicity, deep air diving and hypoxia)

Wednesday 24th April 1996

Scientific Meeting Day 3

Chairperson **Cathy Meehan**

Antarctic diving/hypothermia. Dr J Taylor

Submarine escape and rescue. Dr R Walker

Round window membrane rupture in scuba divers. Dr Noel Roydhouse

Technical Diving Workshop Part 3
Emergent technologies. Rebreathers

Chairperson **Guy Williams**

Introduction. David Elliott

Testing the performance of rebreathers. Hans Ornhagen

Physiology. Bill Hamilton

North Sea Diving technology. David Elliott

Thursday 25th April 1996

Technical Diving Workshop Part 4
Exotic Gas Mixtures

Chairperson **Dr Chris Acott**

Nitrox Mixtures. David Elliott

Development of the PADI Nitrox Program. Drew Richardson

O_2/He and Trimix. Bill Hamilton

(includes thermal stress and how the tekkies do it)

Use of in-water oxygen decompression. Des Gorman

Technical Diving Workshop Part 5
Treatment implications

Chairperson **Mike Davis**

Treatment. David Elliott

In-water oxygen therapy; treatment or first aid? C Acott

Friday 26 April 1996

Technical Diving Workshop Part 6
Formulation of SPUMS Policy

Chairperson **Des Gorman**

Introduction

Personal view. David Elliott

Personal view Bill Hamilton

Discussion

1900 Annual General Meeting
2000 Conference Dinner

SPUMS ANNUAL SCIENTIFIC MEETING 1997

The 1997 Annual Scientific Meeting is to be held at the Waitangi Resort, Paihia in Northland, New Zealand from April 13th to 20th.

The theme of the meeting will be "The Pathophysiology and Treatment of Decompression Illness" and the Workshop will be devoted to the "First Aid Management of Diving Accidents".

Confirmed speakers are Dr James Francis, until recently at the Naval Medical Institute, Alverstoke, England and Dr Richard Moon of Duke University Medical Centre, Durham, North Carolina, U.S.A. Both are excellent speakers and acknowledged experts in the field of decompression illness.

The venue is a first class resort hotel immediately adjacent to the historic Waitangi Treaty House and situated right on the foreshore of the beautiful Bay of Islands. There is outstanding temperate water diving in the region, especially at the Poor Knights Islands with water temperatures at that time of the year around 20-22°C. The region is renowned for its sailing and game fishing and there is a huge range of land based activities for registrants and their families.

Conference conveners are Dr Michael Davis, Medical Director, Hyperbaric Medical Unit, Christchurch Hospital, Private Bag 4710, Christchurch, New Zealand and Associate Professor Des Gorman, Department of Occupational Health, University of Auckland School of Medicine. Enquiries should be addressed to Mike Davis in Christchurch.

LETTERS TO THE EDITOR

WHAT IS TECHNICAL DIVING ?

Hamilton Research, Ltd.
80 Grove Street
Tarrytown
New York 10591-4138 USA

5/1/96

Dear Editor

With no intention to either condemn or praise the practice, I would like to take issue with the definition of "technical recreational diving" in Des Gorman's review of the Safe Limits Symposium which was in the June issue (1995; 25 (2): 110-113).

Des' list of technical diving practices included diving deeper than 50 msw and diving with oxygen-nitrogen mixtures. Now, some hot-dog divers have been diving air deeper than 40 or 50 msw since not long after air was invented, with nothing more technical than any other dive. Likewise, diving with oxygen-enriched air involves nothing any different from an ordinary dive except a little more knowledge, certainly nothing "technical" about the dive part (making and analysing the mixes yourself, okay, that is technical).

Technical diving is diving beyond the normal range using special equipment, techniques and competence. One good minimal definition of a technical dive is a dive involving a change of gases. (That has to be extended to include diving with a rebreather.)

Come to the meeting in the Maldives to hash this one over.

Bill Hamilton

Key Words
Letter, technical diving, nitrox.

Editor's note

Dr R W Hamilton, PhD, is one of the guest speakers at the 1996 Annual Scientific Meeting in the Maldives from April 20th to 28th 1996.

UNDERWATER OXYGEN TREATMENT FOR DECOMPRESSION SICKNESS

PO Box 623
Woollhara
New South Wales 2025

31/1/1996

Dear Editor

I have read with interest the editorial in the September 1995 edition of the SPUMS Journal and the review of underwater oxygen treatment of decompression sickness by Dr Carl Edmonds.[1,2]

In this I see considerable discussion on the underwater use of oxygen for the prevention of decompression sickness (DCS). Dr Carl Edmonds' review deals with the Australian oxygen underwater tables and the use of oxygen for DCS treatment in the areas of tropical island divers and North West Australian pearl divers. Both of these cases are remote from the majority of treatment areas.

I feel that a more in depth discussion should be undertaken on the use of underwater oxygen treatment for recreational divers undertaking a higher level of risk in their diving. These deep and/or technical divers (be it either deep wreck, reef or cave divers) who, through more advanced diver training, are pushing the traditional limits of recreational diving face an increased DCS risk and the understanding of and ability to respond to any hyperbaric trauma should be a primary point of discussion.

As a diver personally involved in undertaking this type of "technical" diving as well as a part-time instructor in this level of advanced training I would ask both Dr Carl Edmonds and the SPUMS medical membership to examine closely the practice of immediate underwater oxygen recompression in cases of the risk of hyperbaric exposure. From a personal perspective I believe that those individuals undertaking this type of diving, as well as those supplying the boat charter services, should be given guidelines on immediate in-water oxygen treatment. Oxygen underwater recompression has been used successfully both in Australia (Case 1) and overseas whereas the denial of this treatment (Case 2) may have resulted in a fatality.

Case 1

The successful use of underwater oxygen recompression occurred with a female Australian sports diver undertaking a deep wreck dive in mid 1992.

The diver having descended to a depth of 66 m noticed her buoyancy increasing and returned to the anchor line to deal with the situation. She became more buoyant, needing to hold on to the anchor line, and signalled her buddy. It was discovered that the low pressure inflator had over-inflated her buoyancy control device (BCD) and when disconnected it was free flowing. The regulator in question was shut down (she was using twin tanks with separate first stage regulators, each with a second stage and a low pressure feed, one set connected to the BCD and the other to "wings", a redundant BCD), she swapped to the second tank regulator and both divers began their ascent. They met a second pair of divers during the ascent, signalled they required assistance and all the divers began to ascend.

During this ascent, she signalled one of the fresh divers accompanying her that she was low on air and needed to share air. Then, with one hand holding her loan regulator and one the accompanying diver, she was unable to vent her BCD properly. The pair lost contact with the anchor line and began an immediate uncontrolled ascent to the surface. She reached the surface having had an 18 minute dive time to a depth of 66 m without any decompression but with no obvious signs of DCS. The boat operator immediately placed her on the decompression lines and she was taken to six m using pure oxygen. She was given, using open circuit scuba, oxygen for 30 minutes at six m, then at three m for 30 minutes.

At all stages she was accompanied by one or more divers monitoring her continuously to ensure that if there was any sign of oxygen toxicity she would immediately be returned to the surface. Upon surfacing, she was placed flat in the boat and given oxygen for another 30 minutes. On landing she was immediately transported to the recompression chamber.

Within one hour of being admitted to the hospital, the diver showed signs of lethargy and loss of co-ordination. She was recompressed over three treatments at the chamber and subsequent examination showed no deficit remaining. She resumed diving six weeks later.

Case 2

The second case involves and father and son diving off the east coast of the United States on a submarine known as the "U-Who".

They descended to a depth of 70 m, removed their decompression tanks and entered the submarine for their planned dive, in one end and out the other. During the penetration, the divers became disorientated due to a collapse of part of the internal structure and following a careful dive through low visibility, exited the submarine. They were now beyond their planned 20 minute bottom time and began their return journey along the submarine to collect their decompression tanks. Having extended their bottom time 11 minutes longer than planned without reaching their decompression tanks and being very low on air, they began a direct ascent to the surface.

Upon surfacing the father had limited use of his arms and legs and while being assisted onto the boat, went into respiratory failure and 20 minutes later cardiac arrest. The son was conscious on the surface but without feelings in his arms or legs. He was transported to a recompression chamber and responded to initial treatment (USN Table 6a) but during his first air break his heart stopped and resuscitation was unsuccessful.

It has been discussed in technical diving journals that immediate in-water oxygen recompression may have averted the second fatality.

Discussion

I am a deep wreck and cave diver and part-time "technical" instructor. **These are my private comments. They do not represent, in any way, the views of any technical diving training agency nor those of the technical diving community in Australia**.

It maybe unwise to directly compare the Australian case with the American incident, however, it is obvious the use of immediate underwater oxygen recompression for treatment delayed, if not offset, any hyperbaric trauma to the diver in the Australian case.

I am interested in both Dr Carl Edmonds' response to the use of such treatment as well as the position and views of the SPUMS Committee and medical membership as to how the ever increasing band of technical divers will be advised on how such treatment can be best undertaken with minimal risk but maximum benefit to the DCS patient.

Richard Taylor

References

1 Editorial. *SPUMS J* 1995; 25 (3):121
2 Edmonds C. Underwater oxygen treatment of decompression sickness. *SPUMS J* 1995; 25 (3):122-128

Key Words
Letter, decompression illness, treatment, oxygen.

Diving Medical Centre
66 Pacific Highway
St Leonards NSW 2065

5th March, 1996

Dear Editor

In reply to Mr Taylor's query I would like to thank him for his letter. My reply is below.

I do not believe there is any real doubt, nowadays, about the immense value of a very early response to developing decompression sickness.

There seems also little doubt regarding the value of the underwater oxygen option. Nevertheless, the pearl divers do it one way, the abalone divers do it another, the US Navy do it quite differently and they are all somewhat at variance with my original recommendations. That is fair, because we are all probably treating somewhat different divers i.e. divers who have behaved quite differently in order to get their DCS.

The Australian pearl divers have a very stereotyped and set regime, which probably works very well as their dives are also very stereotyped. Thus they can probably get away with just one protocol.

The abalone divers, doing all sorts of different things, tend to use all sorts of underwater oxygen treatments.

The US Navy, dealing with their own divers who have always either done the "correct decompression" dive or one that would only impinge very slightly into decompression profiles, get away with their regime.

I originally introduced the underwater oxygen for divers in remote areas, where nothing else is available, and there was often a considerable delay in even getting the oxygen. Thus I had to be a little bit more flexible in my regime than, say, the pearl divers.

You have now introduced another type of diver, the technical diver. I have great philosophical difficulties with this. I have no problem with people doing whatever they want to themselves, as long as they do not entice or involve others. My reservations are that compressed air diving is hazardous enough, especially to those who are not very experienced, and I worry when inadequately trained kids are encouraged to do deep and extended diving, often with apparatus that has limits beyond their understanding. Thus I am not in favour of "technical diving", except for the extremely capable, experienced and very well trained diver.

When the latter is affected with a diving illness, it is my belief that it is likely to be far more significant and potentially more hazardous than most compressed air diving situations. Many would argue with this, and claim that because the physiological principles are the same, so should the illnesses be.

It is my view that if technical diving was used to genuinely reduce hazards, by reducing the duration of the dives, then I would be far more in favour of it. The opposite is usually the real intention for its use.

Having said all this, it does not mean that I would not use the oxygen underwater treatment. I probably would in the individual case. I would just be worried that its application to the technical diving group could lead to multiple problems including oxygen toxicity or serious decompression illness, which are less in the conventional and recreational compressed air diver. I see this as a potential for attributing disrepute to a valuable first aid option.

I would also be a little concerned that many of the technical divers might believe that their knowledge and experience is such that they do not need to follow up the underwater oxygen treatment with either further treatment or diving medical assessment.

As an analogy, splints may be very valuable for treating people involved in motor vehicle accidents due to speeding. The answer is not to make splints more available, it is to stop speeding.

In a similar way, I would not argue against people using the underwater oxygen technique, when they develop decompression sickness. I would just prefer them not to require the first aid treatment.

C Edmonds

Key Words
Letter, decompression illness, treatment, oxygen

DIVING COMPUTER PROBLEMS

Hyperbaric Medicine Unit
Townsville General Hospital
North Ward, Townsville
Queensland 4810

2/1/96

Dear Editor

It is established practice to collect information about the diving history in cases of decompression illness. Depths, times, surface intervals, numbers of dives per day and numbers of days diving are all recorded. The presumed reason for the collection of this information is to make deductions about nitrogen uptake and elimination, together with adherence or not to the safer diving practices of making subsequent dives shallower and not diving too frequently.

These deductions have been based on the assumption of square profile dives and the relationship between actual dives and the precepts laid down in the various diving tables. Now, with the massive increase in the use of dive computers, the value of the information received has become questionable. Computers are now available for hire on most dive trips, while it is perhaps true that most experienced divers now own computers. The information recorded in the log book is still depth and time, but the depth is a maximum depth and the duration of the dive is well over that possible in a square profile dive. Every dive is a multilevel dive, so that, without interme diate depth and time data, nothing useful can be deduced about nitrogen and the probability of decompression illness. Without knowledge of residual nitrogen, surface intervals become meaningless.

Is it still safer to make dives progressively shallower? For example, the first multilevel dive could consist of a short excursion to 30 m, with the rest of the dive spent mainly at 10 m.

The second could have a maximum of 25 m, then "push the envelope" allowed by that particular computer all the way to the surface. Is this safe diving practice?

It would appear that the logged dive history of a computer diver is of little use when that diver develops decompression illness. We will have to stay in the dark until every bent diver arrives with a computer which can be interrogated by the desktop computer of the doctor. As those dive computers which can be downloaded have different interfaces and incompatible programmes, the waiting may be prolonged.

Tom Fallowfield

Key Words
Letter, computers, decompression illness, treatment.

SPUMS ANNUAL SCIENTIFIC MEETING 1995

DECOMPRESSION

Fred Bove

Introduction

This is a discussion of decompression theory, gas kinetics and tissue uptake to give an idea of the issues related to decompression, diving tables and the basis for the different tables, based on some general concepts one of which is that with increasing ambient pressure there is increased dissolved nitrogen in the tissues.

Physics

There are several physical principles which govern the movement of inert gas into and out of tissues, these govern the amount of nitrogen that exists in tissues in the body. Boyle's law, volume is equal to one over pressure multiplied by the constant, deals with the pressure and volume relationship.[1] This is most important at the lower pressure end of the diving spectrum because the rates of changes in volume are the greatest then. Henry's law tells us that the concentration, that is the number of molecules per volume, in a tissue is proportional to the partial

pressure of the gas that is in equilibrium with the tissues. And Dalton's explains the partial pressure relationships.

There are two basic problems. One is that gases go into tissue any time there is an increase in ambient pressure. That never causes problems unless the ambient pressure is quite extreme when one gets cell membrane effects of the inert gases such as narcosis. That is not a solubility issue, but changes in the structure of cell membranes caused by nitrogen. The other problem is getting the gas out of the tissue without damaging the diver as the ambient pressure is reduced.

Gas uptake and excretion

One needs to know how much extra nitrogen is present because by figuring out how much is present we can plan how to get rid of the nitrogen safely. The issues are the flow of the gases via the blood into the tissue capillaries and diffusion of gas from the microvasculature into the tissue. These two factors tell us how fast gases will move in and out of different tissues. The other factor is the solubility, that is the amount of gas that the tissue will hold. One can think about the process as filling a bucket. The solubility determines the size of the bucket and the inflow is determined by the size of the hose filling the bucket. The governing mathematics say that the rate of flow into the tissue is proportional to the gradient between the arterial gas concentration and the tissue gas concentration. If there is no differences in concentration, there is no gas flow. And the greater the difference in concentration, the more rapid the gas flow. An increase in pressure gives an increase in concentration of the gas. Henry's law says that if one doubles the pressure there is twice as much dissolved gas in the tissue. Henry's law does indicate that the gas flow takes time. As the pressure difference between the surroundings and the tissues decreases so does the rate at which gas enters the tissues.

Gas uptake follows an exponential curve that is varied by the solubility of the gas in the various tissues. Water and fat have quite different solubilities for nitrogen. Blood is similar to water, but fat has a five or six times greater capacity for nitrogen. Helium has quite different solubility co-efficients so one cannot use an air table to decompress from a dive on helium, one must use a helium table. They are different tables because solubility and diffusibility are different and these factors control the uptake and the elution of gas from tissues.

When one looks at the body as a collection of different tissues, one finds that different tissues allow gas in at different rates.[2,3] The reason being that the flows into tissues are different and the solubility co-efficients, which govern the uptake, are different and some tissue have a larger capacity. For example, the vitreous humour of the eye is a tissue that has essentially no blood supply. So it has almost no uptake of nitrogen when diving, whereas the blood itself takes up nitrogen very quickly because it is in direct contact with the lung alveolar surface and nitrogen is quickly transmitted to the blood. In some experimental air saturation diving by the US Navy, to 18-21 m (60-70 ft), the only problems were bubbles in the vitreous, because decompression tables had not accounted for these very, very slow uptake tissues in the saturation decompression. Very slow tissues need be accounted for but certainly not in sport diving.

The problem for divers is not the gas uptake. The problem is in returning to the one atmosphere environment. The rates of egress of gas from tissues varies, based on the amount of gas that is in the tissues and the different factors which control gas uptake. Some tissues get rid of gas faster than others. It is often assumed that gas washout is a mirror image of the uptake. It has been demonstrated in many studies over the last twenty years that, in anaesthesia and diving, equivalent volumes of inert gases may take longer to come out than to go in. Washout of nitrogen, or any other inert gas, from tissues is different because pressure differentials driving gas out may be lower than those driving gas in. Cold may reduce blood flow through the skin and muscles by vasoconstriction. Exercise and work warm up and vasodilate the muscles, so the warm tissues takes up more nitrogen. The diver may start the ascent with more of a nitrogen load than the tables assume and may develop decompression sickness (DCS). So uptake of nitrogen and the wash out of nitrogen can occur at different rates in the same tissue. Most diving tables are based on the assumption that uptake and washout are mirror images and work well in practice.

Decompression tables

JBS Haldane first mentioned the idea that tissue supersaturation could occur and that the degree of supersaturation determined the incidence of decompression sickness.[4] A diver can rapidly change the environmental pressure by reducing the depth. A diver can ascend from 18 m (60 ft) to the surface in a minute or two. The diver cannot get rid of the all the gas that has entered the tissues, as a result of being under pressure, in those one or two minutes. This circumstance produces supersaturation of nitrogen dissolved in the tissues. Supersaturated solutions are unstable. They will begin to precipitate whatever is supersaturated in the solution. With a supersaturated solution of sugar in water, eventually sugar will crystallise out of solution and precipitate leaving a saturated solution. When supersaturated gas precipitates bubbles form in blood and tissues. Bubble production has pathophysiological consequences which are prevented by decompression tables and computers.

Haldane did several experiments to show the effects of exposure to increasing pressure. He used goats as his

experimental animals as they raised the affected limb when they developed DCS. He calculated gas uptakes for different non-anatomical "tissues" assuming that different tissues were gaining nitrogen at different rates. When the dive ended and the goats began to surface, all the tissues had raised nitrogen levels. He found that if he reduced the chamber pressure by halves the goats did not bend. After a period at that pressure the chamber pressure could be halved again without injuring the goats. Haldane proposed that goats and men could tolerate some supersaturation. He suggested that a supersaturation of twice the ambient pressure could be tolerated by the tissues without decompression sickness. He assumed that there would not be bubbles but we know now that is probably not the case.

Haldane proposed that a diver should ascend to a depth that was half the pressure of the tissue nitrogen on the bottom, then wait until the tissues nitrogen pressures came to a point where the ambient pressure could be halved and then move up again. One could come step wise toward the surface never allowing any tissue to exceed that level of supersaturation. This is the idea behind all the diving tables we use. No matter whether one has them on a card or whether one has them in a computer, whether they belong to PADI, the US Navy (USN), DCIEM or the Royal Navy, they are all based on the Haldane concept, that tissues supersaturate, that if we come toward the surface and do not allow supersaturation to exceed a certain ratio, then we will not get bubbles, or we will not get DCS. Those two are not necessarily the same condition.

In the application of this idea, there were a variety of different hypotheses. The group at the Experimental Diving Unit (EDU) in the USN in the sixties came up with M values.[5] M values are numbers that represent the partial pressure of nitrogen in a tissue which could exist upon surfacing and still minimise the risk for bubbles. The Goodman and Workman group at EDU tested human subjects and discovered that there were certain partial pressures of nitrogen that could be tolerated in tissues with an excursion back to 0.8 bar of nitrogen; that is the surface. In fact the ratio was not two to one. Goodman and Workman mathematically modelled six tissues, which do not represent any anatomical parts of the body but only rates at which gas enters and leaves the tissue, and later found, by experiment, that the allowable supersaturations decreased with tissue half time.

Each of the model tissues had a specific partial pressure of nitrogen that would allow one to reach the surface without problems. One could then calculate how much nitrogen was present in each tissue and as long as it did not exceed the M value for each of the tissues one could come to the surface. And if gas partial pressure did exceed any of these values one would have to wait while the nitrogen left the tissues. Once the M values were reached one could come to the surface. This provides a method to create a decompression table.

The computers that we use in diving use the principle of preventing a certain level of supersaturation from being exceeded. Each tissue has a surfacing M value and one can use a computer program to find the tissue that will stop the diver from ascending to the surface. A computer can calculate the time needed to degas sufficiently to ascend. Repetition of these calculations produces a diving table. The PADI tables differ from the USN tables, they were designed independently, and were tested with a small number of dives that were not adequate for true validation, but they are being used. Let us compare the two for a 30 m (100 ft) dive for twenty five minutes, that is the USN no-stop limit, followed by a three hour surface interval and then an 18 m (60 ft) dive for thirty six minutes. This is what the US navy table would allow you. The calculated partial pressures of nitrogen in the ten minute tissue would be just at the M value, but everything else would be below the M value. The 30 m dive for twenty five minutes would allow one to come to the surface because all the calculated nitrogen partial pressures are below the allowable maximum partial pressure before ascending. The PADI tables have a no-stop time of twenty minutes at 30 m (100 ft). They required a five minute safety stop at 15 ft (4.5 m). After a three hour surface interval PADI would allow fifty minutes for an 18 m (60 ft) dive, whereas the USN would only allow thirty six minutes.

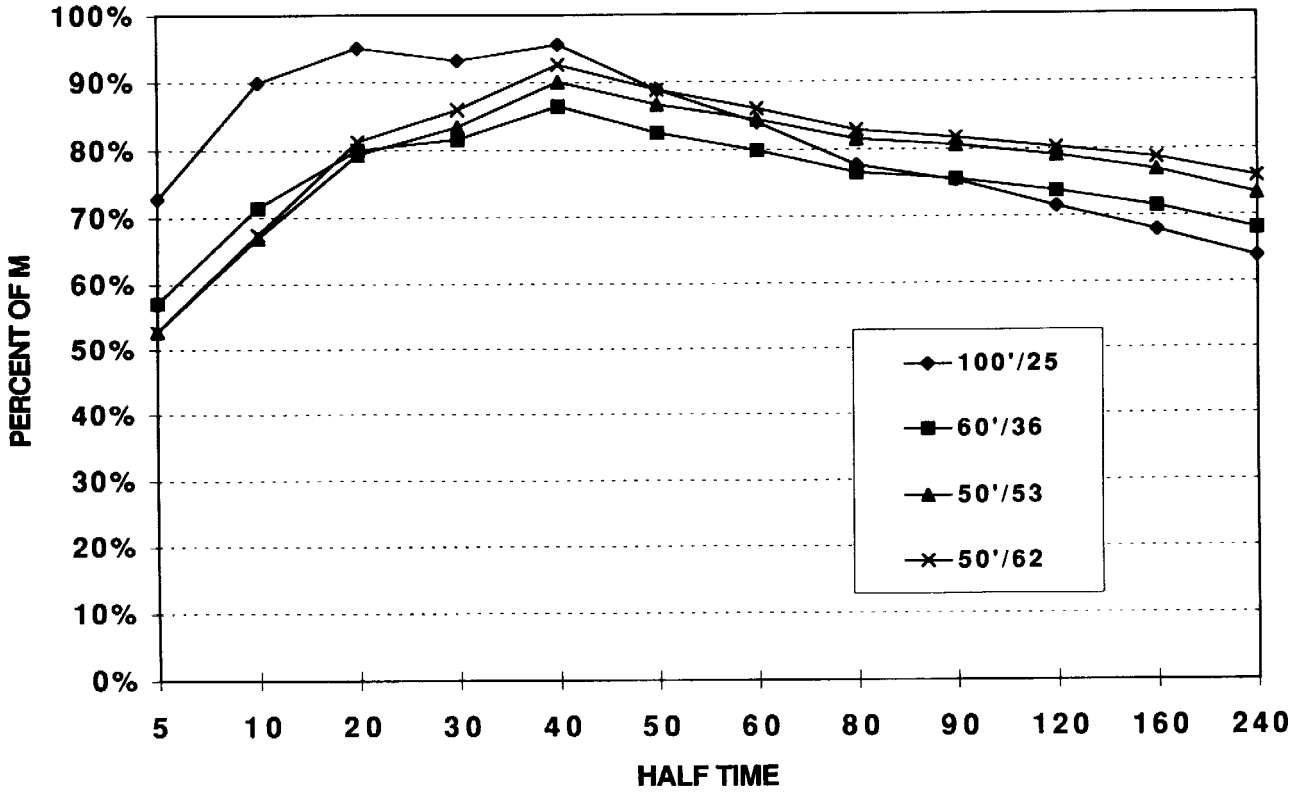
All the numbers are well below the allowable surfacing values on both dives. This is one way to look at a dive table and find out if it is providing the appropriate amount of safety in ascending after a certain kind of dive.

With the first dive and the second dive with the USN table the ten minute tissue, gas partial pressure almost reaches one hundred percent of the M value. The 30 minute tissue and all others are well below the acceptable level in both dives. Using the PADI table the percentages are below a hundred percent on the first dive but the second dive approaches 100% in the ten, twenty, thirty and sixty minute tissues. One need not run every table through a computer program to find this out. In fact that is what the computer is doing during a dive. It checks the pressure and time that one is below an acceptable surfacing value and shows that it is safe to surface. If one has exceeded the M value in one of the tissues the computer will give the decompression stops required.

Figure 1 is a graph of the percentage of the M value for multiple dives with the USN tables, to 30 m (100 ft) for 25 minutes, 18 m (60 ft) for 36 minutes, 15 m (50 ft) for 53 minutes and 15 m (50 ft) for 62 minutes with appropriate surface intervals would not require decompression stops. This would be an acceptable series of four dives based on the navy table. With each dive the long half time tissues move towards one hundred percent of M value. If one does a fifth dive and sixth dive some of these slower tissues become critical, whereas with most sport diving the critical

FIGURE 1

USN TABLES USED TO CALCULATE FOUR NO-STOP REPETITIVE DIVES



The sequence starts with a 30 m (100 ft) dive to the no-stop limit and continues with the no-stop limit for each dive. A 4.5 m (15 ft) safety stop follows each dive. The curves show percentage of the tissue M value for 12 tissues half times. Long half time tissues increase in saturation in the later dives.

tissues are in the 20, 30 and 40 minute range. With diving all day and accumulating gas, the longer half time tissues begin to become important. This is one of the reasons why the multi-day, multi-dive type of exposures might cause DCS. Some of the early computers did not account for these longer tissues. Now most computers do.

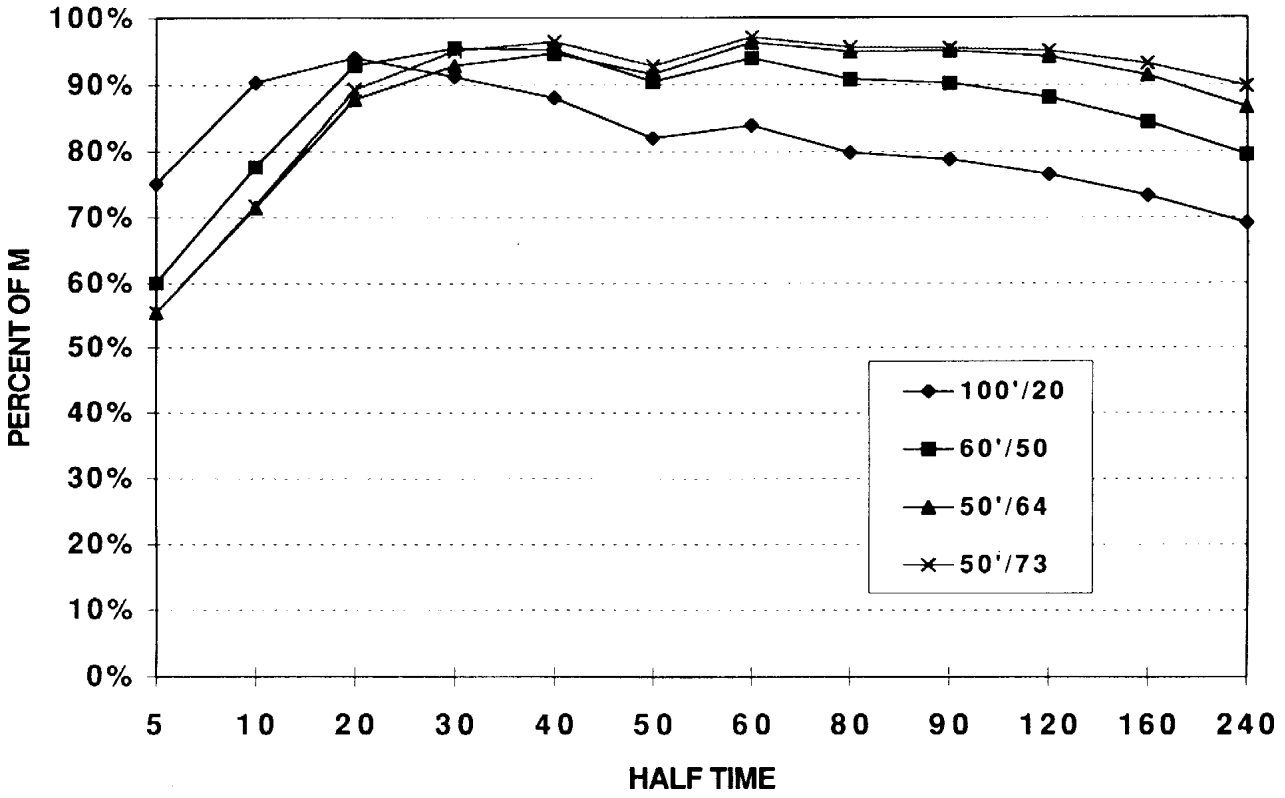
Figure 2 shows the percentage of the M values using the PADI table times for the four dives. The dives become 30 m (100 ft) for 20 minutes, 18 m (60 ft) for 50 minutes, 15 m (50 ft) for 64 minutes and 15 m (50 ft) for 73 minutes. It is clear that 100% of M values is approached earlier in all the slower tissues. It is this multi-dive profile that increases risk for DCS. Doing six dives a day for five or six consecutive days is risky because the longer half life tissues do not completely clear in twenty four hours and adding more nitrogen begins from a higher baseline.

Problems with tables

Haldane's group in 1908 worked out the ideas of gas wash in and gas wash out. And they asked whether there were effects that they could not account for with their theory. They asked if there was an independent pressure effect. They exposed goats to a series of different depths, with the time of exposure adjusted so that tissue inert gas loads were about the same, then decompressed the goats on what they thought were appropriate decompression profiles and looked at the number of symptoms in the goats. These were approximately equivalent gas loads, but as the pressure increased there was a higher incidence of DCS. This is one of the concerns with all tables, because even though we do the theoretical calculation, when the pressure is higher, there are factors we cannot account for and there is more risk of DCS. The tables we use and the computers that we use still do not properly account for some of the unknown factors that contribute when one is at the extremes of depth. They also studied the time effects, at a series of depths. All these times and depths were designed to set approximately the same tissue gas concentrations. Yet they found an increasing incidence of DCS in the goats as they expanded the time trying to keep the gas tensions the same. Although excess pressure and excess time are nicely accounted for in the mathematics, whenever we push to extremes, the tables are not going to

FIGURE 2

PADI TABLES USED TO CALCULATE FOUR NO-STOP REPETITIVE DIVES



The sequence starts with a 30 m (100 ft) dive to the no-stop limit and continues with the no-stop limit for each dive. A 4.5 m (15 ft) safety stop follows each dive. The curves show percentage of the tissue M value for 12 tissues half times. Long half time tissues increase in saturation in the later dives.

be perfect and we must account for those differences empirically.

Nitrox

How can we get more bottom time and not accumulate gas in the tissues? Physical laws dictate how much nitrogen one takes up. To reduce nitrogen uptake one can reduce the amount of nitrogen in the breathing gas. To do that one has to the proportion of nitrogen in the compressed air by adding oxygen. Nitrox is now being offered to recreational divers. Nitrox reduces the amount of nitrogen taken up by the tissue because a diver breathes a lower partial pressure of nitrogen. Knowing the oxygen percentage the diver can calculate the equivalent air depth, which is the depth equivalent if a diver was breathing air at the same nitrogen partial pressure. For a thirty percent nitrox mixture (30% oxygen and 70% nitrogen) 30 m (100 ft) is equivalent to about 24 m (80 ft) using air, so a diver can do a 30 m (100 ft) dive and use the 24 m (80 ft) air table to get a bottom time. The USN no-stop limit for 30 m (100 ft) is 25 minutes whereas at 24 m (80 ft) it is 40 minutes. So using 30% nitrox at 30 m (100 ft) allows a 40 minute bottom time. Nitrox was used extensively for mine clearance in shallow waters at the end of the Second World War, and was used in civilian life for commercial divers doing shallow work. Working at 15 to 18 m (50 to 60 ft)if a diver breathes 30% or 40% oxygen there is significant extension of bottom time with minimal decompression risk.

The problem with nitrox is that there is a limit to the partial pressure of oxygen because of acute neurological oxygen toxicity. If a diver breathes oxygen below 1.6 bar the risks of seizures is quite low. If one breathes oxygen above 1.6 bar the risk of seizures is high. Having a fit underwater has a very high risk of causing death by drowning. Using air the partial pressure of oxygen reaches 1.6 bar around 60 m (200 ft). So one could go to 60 m (200 ft) without getting to a dangerous level of oxygen. At that depth one has narcosis which is, in a sense, an intrinsic safety limit. A scuba diver will have very little time at 60 m (200 ft) because gas utilisation will be high, and will never reach the threshold for oxygen toxicity. Using 30% nitrox, when a diver reaches 39 m (130 ft) actual depth the

oxygen partial pressure exceeds 1.6 bar. Breathing 30% nitrox a diver cannot go deeper than 39 m (130 ft) because of the risk of oxygen toxicity. This fact seems to be lost on some nitrox divers who think that with nitrox they can extend their depth without getting extra nitrogen gas load. For example, the equivalent air depth for 30% nitrox at 45 m (150 ft) is 36 m (120 ft) so one would expect more bottom time. The problem is if a diver goes to 45 m (150 ft) with 30% nitrox he is at high risk for an oxygen seizure. With 40% nitrox a diver reaches the toxic level of oxygen at about 21 m (70 ft). With 40% nitrox the equivalent air depth for 30 m (100 ft) is 18 m (60 ft) and one could theoretically do a 30 m (100 ft) dive for 60 minutes with no decompression. Unfortunately a diver is likely to have a seizure long before the 60 minutes are up because he is well over 1.6 bar toxic range. The problem with nitrox is that although it can extend bottom time it adds risk. It can only extend bottom time safely in depths of 18 m (60 ft) or less, where it is quite safe and the 0_2 partial pressure will not reach seizure levels.

Saturation

The other way a diver could extend bottom time is to stay at some depth and saturate the tissues with inert gas. One would then have an excursion range up and down that would not exceed the levels of super saturation needed for bubble formation. The risk of DCS would come when the diver wanted to return to the surface. In commercial deep diving, where there are long jobs to be done, divers reside in a deck chamber at the working depth and travel to the work site in a pressurised bell. At the end of the work they decompress for days in the chamber. Usually the gas is helium and not nitrogen.

Isobaric counter diffusion occurs when one switches breathing gases between mixtures with different inert gases. The phenomenon was first described changing from nitrogen to helium.[6] If a diver is breathing a nitrogen oxygen mixture, with the chamber pressurised with the same mixture and switches to helium by mask without changing the chamber pressure nitrogen comes out of the tissues and helium enters. As helium is more diffusible it enters faster. In a chamber divers may develop skin injury because the high nitrogen level in the chamber prevents nitrogen diffusing from the skin. With helium entering the skin net concentration of inert gas increases and bubbles form in the skin. This can be a problem in commercial diving when divers switch breathing gases. Some switches can produce excess supersaturation without a change in pressure.

Whales

Somebody asked me why whales do not get bent. If one takes a standard whale that weighs 100 tons or 90,000 kg, it is about 30 m (100 ft) in length. Lung volume is 5,000 litres. Blood volume is 6,300 litres. The total number of molecules of nitrogen in these different places is calculable based on percentage of body fat and so on. If one does some mathematics, one finds that when a whale dives, and they dive deep for surprisingly long times (up to two hours), the tissues take gas up from the lungs. The whale's lungs can collapse to zero volume because the ribs are disarticulated from the spine. Their trachea and main airways are rigid and do not collapse. As the whale descends, not only are the lungs compressed, but the alveoli are emptied of air so that there is reduced uptake. Residual air is pushed into the respiratory dead space so it does not exchange. There is only a certain amount of nitrogen in the lung, not enough to supersaturate the tissues. So, in a breath hold dive a diving mammal stays out of trouble because there is not enough nitrogen available to saturate the tissues and diffusion from the lungs is reduced.

Decompression sickness

When bubbles are present, decompression sickness is present. With small numbers of bubbles the disease is subclinical and is usually unrecognised and not recorded. I will present the classification decompression sickness as Type 1 and Type 2. In the original papers[7] Type 1 was designated as a non-systemic decompression sickness, in the skin or the joints. Type 2 covered all systemic related symptoms. If one breaks down the pathophysiological concepts of DCS into those that affect only the joints or skin and everything else is systemic, clear concept of the pathophysiology can be developed.

There is also an interesting combination of disease which we published a number of years ago. The diver, typically a sport diver, does a dive, has an inert gas load, starts for the surface and sustains an air embolism.[8] The diver has a gas load, with supersaturated tissue, add free bubbles to the body from gas embolism which become foci for growth of excess gas. There results a more severe syndrome with combined pulmonary barotrauma and arterial gas embolism with an inert gas load in the tissues.

Ian Unsworth treated a hundred cases in Sydney.[9] Here are some of his statistics. Forty nine of them had greater than three repetitive dives. Forty four dived to greater than 30 m. This is the depth and time relationship. Thirty eight had exercised immediately after diving. Twenty three missed decompression time and thirty three had other problems like fatigue and equipment failure. Fifteen were in very cold water, fourteen did not record any times, fourteen had out of air emergency ascents, there was a number of inexperienced divers, ethanol was involved in a few, some were flying and one was relying on a computer alone.

Radiculopathy after diving

An unusual diving problem is that some lumbar discs have an air pocket in their centres. Radiologists call these vacuum discs. Some cases seem to indicate that a barotrauma like syndrome related to this vacuum space in the disc may aggravate an already present radiculopathy. These spaces obviously take up gas when diving. On ascent when the gas expands, the volume change can cause symptoms of a radiculopathy when a bit of the disc herniates out into the spinal canal. There have been a number of people who have had cervical symptoms or low back symptoms, clearly related to radiculopathy and not spinal cord injury, which is a distinction one can make by careful examination, after diving, usually related to degenerative disc disease. Symptoms of radiculopathy after diving may not be spinal cord DCS, but the equivalent of a Boyle's law effect on some of these vacuum spaces in the discs.

How to provoke DCS

Some basic ideas on provoking DCS can be derived from these concepts. Miss decompression stops, do a rapid ascent or go to altitude, dive deeper than 50 m (165 ft), do more than three dives a day, or repetitive deep diving. If your buddy gets bent, and you have been diving together all day, you might get decompression sickness.

Audience participation

Knight

Anaesthetists, when I started my anaesthetic training in 1957, had accepted that anaesthetic gases went in quicker than they came out. It is still not fully accepted in the diving community it seems. And that will explain quite a lot of the failures of calculation. Tables are extreme simplifications of an extremely complicated diffusion-perfusion relationship. If one relies on a decompression computer's beautiful algorithm, remember that a lot of the computers have shown that they do not actually dive safely, if one puts them in a chamber they will let one do all sorts of dives that we know bend people.

Bove

No computer that has had clinical testing to prove the algorithm. The PADI tables have had some clinical testing but people argue that not enough dives were done to prove the algorithm. The US Navy did a thousand dives during the last revision of the air tables, which is soon to be published. The work cost ten million dollars and ten years of time. They are obviously going to be used by navy divers but the majority of their value will be for the sport diver. We do not have a lot of tested tables. I am hoping that the navy algorithms will be built into computers so that one set of algorithms for sports divers will be well tested.

Unidentified speaker

It is very interesting that the new US navy tables are going to be very similar to the Canadian DCIEM ones, which I would say is the other major database that was reasonably well tested.

Bove

I agree that the DCIEM tables were well tested.

Molvær

A question about repetitive breathhold diving. We had a colleague visiting our submarine escape tower, or tank, which is 18 m deep. He played around doing repetitive dives to that depth for a day and got neurological decompression sickness.

Bove

It was the Norwegians doing multiple free dives in a submarine escape tank who first discovered that the instructors could get bent. Obviously on a single dive there is not enough inert gas to load the tissues, but it is quite possible with rapid repetitive breath hold diving carried out for a long time. Taravana was described in Tuamotu Islanders, pearl divers who did free dives all day to 24-27 m (80-90 ft), and was in fact decompression sickness.

Molvær

One comment on oxygen. You mentioned acute oxygen toxicity. You did not mention the effect on the lung. It is an aggressive element and we have seen that the diffusion capacity will go down if you bring oxygen partial pressures up.

Bove

Pulmonary oxygen toxicity is a well described and very important issue. It usually is of minor importance in a one to two hour time frame. One sees some decreases in vital capacity at about 60-90 minutes, but that is not clinically important unless one keeps breathing the high partial pressures of oxygen. So in the diving community the most important problem is the acute neurotoxicity of oxygen, not the more chronic pulmonary toxicity. Rebreathers are coming for sport diving and the companies building the rebreathers want to set the partial pressure of oxygen at 1.4 bar. 1.4 bar gives fairly good decompression profiles because of the lower nitrogen, but I think if people swim around for two or three hours with 1.4 bar of oxygen we are going to see pulmonary oxygen toxicity. Every time we try to defy the laws of physics we get into another problem.

References

1 Boyle R. New pneumatic experiments about respiration. *Phil Trans R Soc London* 1670; 5: 2011-2031

2 Kety S. The theory and applications of the exchange

of inert gas at the lungs and tissues. *Pharm Review* 1951; 3: 1-41
3 Perl W, Rackow H, Salantire E, Wolfe GL and Epstein RM. Intertissue diffusion effect of inert fat-soluble gases. *J Appl Physiol* 1965; 20: 621-627
4 Boycott AE, Damant GCC and Haldane J. The prevention of compressed air illness. *J Hyg Cambridge* 1908: 8: 342-443
5 Workman RD. *Calculation of decompression schedules for nitrogen-oxygen and helium-oxygen dives. US Navy Experimental Diving Unit research report 6-65*. Washington, DC: 1968
6 Lambertsen CJ and Idicula J. A new gas lesion syndrome in man induced by isobaric counterdiffusion. *J Appl Physiol* 1975; 39 (4): 434-443
7 Golding RC, Griffith P, Hempleman HV, Paton WDM and Walder DN. Decompression sickness during construction of the Dartford tunnel. *Br J Indust Med* 1960; 17: 167-180
8 Neuman TS and Bove AA. Combined arterial gas embolism and decompression sickness following no-stop dives. *Undersea Biomed Res* 1990; 17: 429-436
9 Unsworth IP. Analysis of 100 cases of decompression sickness among sports divers. *Undersea Biomed Res* 1990; 17 (Suppl): 89-90

Key Words
Decompression illness, tables, nitrox, physiology.

Dr A A (Fred) Bove was the Guest Speakler at the 1995 Annual Scientific Meeting. His address is Chief of Cardiology, Temple University Medical Center, 3401 North Broad Street, Philadelphia, Pennsylvania 19140, U.S.A.

NEUROLOGICAL INJURY AND A RETURN TO RECREATIONAL DIVING.

Chris Acott

Introduction

The development of guidelines for a return to recreational diving following any neurological injury is hindered by the lack of objective data. Furthermore, the available data are limited to commercial or military divers.

A return to diving should depend upon a negative response to all of the following questions:

a Will the continued diving make the condition of the diver worse?
b Will the condition of the diver compromise the diver's or buddy's safety in the water?
c Will the condition of the diver predispose to or aggravate a diving illness?

In addition, if the neurological injury was caused by diving, negative answers must be obtained to the following questions before any consideration of a return to diving can be given:

a Did the diver suffer pulmonary barotrauma?
b Was the diver's illness commensurate with the diving exposure?
c Did the diver respond well to treatment?
d Has the diver any residual problems from the diving injury?

Decompression induced neurological injury

Permanent neurological damage due to decompression illness has been known for more than a 100 years and while physicians agree that a diver with any objective neurological deficit after a decompression accident is unfit to dive,[1] the suitability of such a diver to return to diving if the deficit "resolves" is debateable. For example animal model data have demonstrated that diving can induce "silent" central nervous system damage.[2] In addition, there is post mortem evidence that lesions may persist in the spinal cord after decompression injury and without clinically evident neurological residua in humans.[3,4] Palmer[5] and Mork[6] in separate studies have also shown a positive correlation between cerebral vasculopathy and diving in post mortem studies of divers with or without a history of decompression injury. These divers studied, as far as it is known, were not incapacitated in any form. Overall, there is a paucity of objective data showing that nervous system damage, that occurs silently (in the absence of both clinical symptoms and signs) after diving and persists, causes any loss of function or impairs activities of daily living. That is, the presence of an abnormality at post mortem does not indicate an inevitable impairment of function. Nevertheless, on the basis of these animal studies, some physicians maintain that any episode of neurological decompression illness permanently disqualifies a diver. This stance may actually delay or suppress the reporting of symptoms, and hence delay treatment, for fear of subsequent disqualification. Although this argument is especially relevant to professional divers, it is still applicable to recreational divers.

The original observation that decompression injury may cause an encephalopathy must be assessed cautiously as the study lacked either suitable controls or established neuropsychological tests and the subjects continued to work in compressed air,[7] it is noteworthy that similar study outcomes are reported. For example, a very recent Norwegian study showed a positive correlation between central nervous system symptoms (problems with concentration, memory, irritability and depression) and un-

treated symptoms following decompression in both commercial and recreational divers.[8]

Neuro-investigative techniques (CT, MRI, SPECT, PET) have been either too sensitive or too insensitive in the identification of decompression injury residua.[9-12] Evoked response studies are equally unrewarding, only being abnormal in those divers with an obvious deficit.[13]

The use of fluorescein angiography to examine the retina of divers has shown a positive correlation between perfusion deficits and retinal pigment changes in divers who have had decompression illness[14] and may be an indication of subtle cerebral lesions. However, these changes are often seen in older non-diving individuals and so the clinical relevance, particularly with regard to fitness to return to diving is not established.

The electroencephalogram (EEG) has been used to study divers with decompression illness.[15-17] These studies have shown an association between dysbaric illness and EEG abnormalities but are associated with both significant false positive and false negative results.

Neuropsychological screening tests have been used to investigate brain injuries after decompression illness, but the frequency of persistent neurological deficits following treatment varies with the type of neuropsychological assay.[13,18-21] Data can not be compared between studies unless the same neuropsychological tests are used, even if the same population is studied.[22-25] For example, conclusions from a study on Australian abalone fisherman-divers depended entirely upon the test used. Furthermore, neuropsychologists are yet to agree on test standards and diagnostic criteria for disorders in higher mental functions.[17] Hence these tests need further testing and standardisation. However, provided the same tests are administered by the same person they can be, and have been, used to establish the return of a diver's cognitive function during recovery from a decompression injury.[13,26]

Consequently, the primary follow up for a diver who has had a decompression injury remains a meticulous clinical neurological history and examination with emphasis on the diver's mental function. The ideal frequency of these reviews is not established, but there should be one done at least one week after treatment to ensure that a treatable relapse has not occurred. It is now apparent that the pathological processes of decompression illness do not return to normal for at least one month, even following successful treatment.[27] It is reasonable to perform another review 4 or 6 weeks after treatment and it is at this review that a decision can be made about a return to diving. A longitudinal study of 25 recreational divers who had decompression illness, showed that 6 of the 12 divers who were neurologically normal at discharge, had overt neuropsychological problems at one year.[26] This study demonstrated that divers need continued assessment for 12 months after a decompression injury. The overall data suggest that at a minimum: divers should be reviewed one week, 4 or 6 weeks and one year after treatment for decompression illness; and that 4 weeks after treatment is the earliest time to consider a return to diving. I suggest further reviews be conducted 3 and 6 months after treatment and that fitness to continue diving be assessed at each of these reviews. A return to diving should be contingent upon a decompression injury that is commensurate with exposure, a good response to treatment, the absence of sequelae and the absence of any identifiable risk factors for decompression illness. Residual deficits should exclude the diver from future diving and indications that there is persistent tissue damage despite the recovery of function (incomplete initial response to treatment and a gradual resolution over months) are relative contraindications to further diving as they represent an increased risk of further damage, due to a decreased neuronal pool, if a decompression injury recurs.[15,28]

Divers with a history of pulmonary barotrauma, with or without neurological sequelae and whether "deserved" or "undeserved", should be excluded from further diving. The precipitating cause is thought to be an abnormality of the diver's lungs and because of the demonstrated inability of available investigative techniques, such as radioisotope scanning,[29] pulmonary computerised tomography[30] and methacholine challenge testing,[31] to sensitively or specifically identify someone at risk from pulmonary barotrauma, these divers have to be assumed to be at considerable risk of recurrence. A recommended distinction between "deserved" (eg a rapid breath hold ascent) and "undeserved" (eg a controlled slow ascent) barotrauma with respect to diving fitness[32] is unacceptable because of the subtle undetectable pulmonary scarring that may occur after injury.[1]

Divers whose injury is thought to have been associated with a cardiac shunt should also be advised not to return to recreational diving. Although the significance of a patent foramen ovale (PFO) in decompression illness is established, the utility of bubble contrast echocardiography in assessing future fitness to dive is controversial.[27,33,34] For example, could it be justifiable to use a contrast medium that contains a suspension of bubbles to demonstrate a shunt when there is the possibility that these same bubbles will flow to a cerebral circulation that has already been damaged by the passage of earlier bubbles? In addition, a negative test for a PFO does not exclude a pulmonary or any other anatomical shunt. However, once established, a PFO may not exclude professional divers from continuing to dive because they may be able to avoid diving patterns likely to result in venous gas emboli, which is unlikely in recreational diving.

Divers who have suffered neurological symptoms despite a seemingly trivial exposure and any recreational diver who has returned to diving and suffers another decompression injury without any apparent cause should be advised to cease diving because they may be inherently at risk of decompression illness due to risk factors that have yet to be defined.

Non-decompression induced neurological problems

Traumatic brain injury will affect fitness for diving and consequently considerable information about the nature of the injury is necessary. Details of localising signs, the period of unconsciousness, the duration of post traumatic amnesia, the presence of intracranial bleeding or of a penetrating injury or skull fracture and the likelihood of future seizures must be known. A study of closed head trauma in 1,000 patients reported the lowest incidence of traumatic epilepsy were in an uncomplicated head injury with post traumatic amnesia of less than 1 hour.[35] A history of a loss of consciousness of greater than 10 minutes, the presence of localising signs, an amnesic period of greater than one hour, the presence of intracranial bleeding, any skull fracture other than an uncomplicated linear one and of a penetrating injury would disqualify from future diving because of the risk of post traumatic epilepsy. However, a five year seizure free period without medication and after a meticulous history and examination may allow a diver to return to recreational diving if one uses idiopathic epilepsy as a guide,[36] although some may consider this too conservative because 80% of post traumatic epilepsy will present within 2 years of injury.[37] My view is that any risk of an underwater convulsion is a contraindication to further recreational diving.

There are different opinions about the fitness of divers who suffer migraine headaches. Hickey describes these as an absolute contraindication,[38] while Greer has an opposing view.[36] Diving may precipitate a migraine headache by hypercarbia, hyperoxia, hypothermia, cold water exposure, stress and arterial bubbles. The development of a migraine headache may impair a diver's safety underwater with the development of vertigo, visual disturbances, nausea and vomiting or produce a post dive diagnostic dilemma. Obviously each diver needs to be assessed individually with attention focused on prodromal symptoms and syncope or any loss of consciousness.

A peripheral neuropathy may interfere with the diver's dexterity and can be confused with symptoms of decompression illness. These divers should not continue to dive.

The development of a degenerative or demyelinating disease of the nervous system, spinal cord trauma or intracranial bleeding, with the risk of epilepsy, should disqualify the diver from future diving because any subsequent development of decompression illness may worsen the diver's neurological status.

Remedial spinal surgery (e.g. for an intervertebral disc prolapse with neurological symptoms and signs) should not exclude a diver from continuing to dive; although there is at least the theoretical risk of interference with the para-vertebral venous plexus drainage causing venous stasis predisposing to spinal cord decompression illness. More data are needed.

Any medication that can have central nervous system side effects should disqualify a diver during the course of the prescription. Anti-motion sickness (in particular the belladonna alkaloids) and one anti-malarial medication (mefloquine) are in this category. The belladonna alkaloids are commonly used to prevent motion sickness, particularly preparations containing hyoscine, and when taken as prescribed should not cause any incapacitation.[39] However, side effects such as drowsiness and blurred vision may interfere with the safe conduct of a dive. In addition, when taken to excess, which has occurred on live-aboard dive boats, these drugs can result in an acute brain syndrome.[40] This may be difficult to differentiate from acute decompression illness and/or have fatal consequences underwater. Any physician prescribing an anti-motion sickness medication to a diver should stress the importance of taking it only as directed and of beginning therapy 12 hours before going to sea (both to establish motion sickness prophylaxis and to allow time for any adverse effect to manifest before diving).[41]

The anti-malarial mefloquine (Lariam) has adverse effects even when taken as prescribed (spatial disorientation, vertigo, myalgia, joint pain, syncope, confusion, psychotic manifestations, headache and convulsions) and must never be prescribed for malarial prophylaxis to anyone who wishes to dive.[42] Anyone who is taking mefloquine and wanting to dive should stop taking it four weeks before diving. They should not start on chloroquine as the combination can cause asystole. The risk of convulsions from Lariam is 3%. I think a 3% risk of convulsing underwater is not acceptable. A lot people get symptoms of nausea and vomiting and vertigo, neither of which increase underwater safety.

Acknowledgment

I would like to thank Dr D F Gorman for his review and criticism of this manuscript.

References

1 Elliott DH. Residual effects and the return to diving. In *Diving Accident Management. 41st. Undersea*

and Hyperbaric Medical Workshop. Bennett PB and Moon RE. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1990; 235-243

2 Dick EJ and Broome JR. Occult spinal damage in clinically normal pigs following diving. *Undersea Biomed Res* 1994; 21 (Suppl): 68

3 Palmer AC, Calder IM, McCallum RI. and Mastaglia FL. Spinal cord degeneration in the case of "recovered" spinal decompression sickness. *Brit Med J* 1981; 283: 888

4 Palmer AC, Calder IM and Hughes JT. Spinal cord degeneration in divers. *Lancet* 1987; ii: 1365-1366

5 Palmer AC, Calder IM and Yates PO. Cerebral vasculopathy in divers. *Neur Pathol Appl Neuro Biol* 1992; 18: 113-124

6 Mork S. CNS findings from 40 diver autopsies. In *Report of an open meeting of the MRC Decompression Sickness Panel's Long Term Health Effects Working Group at the University of Hawaii, 2nd. International Symposium on Man in the Sea*. Halsey MJ. Ed. London: Medical Research Council. 1988.

7 Rozsahegyi I. The late consequences of the neurological forms of decompression sickness. *Brit J Ind Med* 1959; 16: 311-317

8 Brubakk AO, Bolstad G and Jacobsen G. Central nervous symptoms are related to untreated symptoms of decompression sickness. *Undersea Biomed Res* 1994; 21 (Suppl): 90

9 Hodgson M, Beran RG and Shirtley G. The role of computed tomography in the assessment of neurological sequelae of decompression sickness. *Arch Neurol* 1988; 45: 1033-1035

10 Rinck PA, Svihus R and De Franciso P. MR imaging of the central nervous system in diving. *J Magnetic Resonance Imaging* 1991; 1: 293-299

11 Moon RE, Hoffman JM, Hanson MW, Reiman RE, Coleman RE, Thiel DR, Fawcett TR, Francis JJ, Gorback MS and Massey EW. ^{18}F-Deoxyglucose positron emission tomography (PET) in the evaluation of neurological decompression illness. *Undersea Biomed Res* 1991; 18(Suppl): 18

12 Hodgson M, Smith DJ, Macleod MA, Houston AS and Francis TJR. Case control study of decompression illness using ^{99}Tc^{m}-HMPAO SPECT. *Undersea Biomed Res* 1991; 18 (Suppl): 17

13 Gorman DF, Edmonds CW and Parsons DW. Neurological sequelae of decompression sickness: A clinical report. In *Proceedings of the IXth Symposium on Underwater and Hyperbaric Physiology*. Bove AA, Bachrach AJ and Greenbaum LJ Jr. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987; 993-998

14 Polkinghorne PJ, Sehmi K Cross MR, Minassian D and Bird AC. Ocular fundus lesions in divers. *Lancet* 1988; ii: 1381-1383

15 Gorman DF, Pearce A and Webb RK. Dysbaric illness treated at the Royal Adelaide Hospital 1987. A factorial analysis. *SPUMS J* 1988; 18 (3): 95-101

16 Todnem K, Skeidsvoli H, Svihus R, Rinck PA, Ruse T and Kambestad BK. Electroencephalography, evoked potential and MRI brain scans in saturation divers. An epidemiological study. *Electroencephalogr Clin Neurophysiol* 1991; 79: 322-329 1991.

17 Elliott DH and Moon RE. Long term health effects of diving. In *The Physiology and Medicine of Diving. 4th.Edn*. Bennett PB and Elliott DH. Eds. London: W.B. Saunders, 1993; 585-604

18 Kelly PJ and Peters BH. The neurological manifestations of decompression accidents. In *International Symposium Man in the Sea*. Hong SK. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1975; 227-232

19 Levis HS. Neuropsychological sequelae of diving accidents. In *International Symposium Man in the Sea*. Hong SK. Ed. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1975; 233-241

20 Peters BH, Levis HS and Kelly PJ. Neurological and psychological manifestations of decompression sickness in divers. *Neurology* 1977; 27: 125-127

21 Brew SK, Kenny CT, Webb RK and Gorman DF. The outcome of 125 divers with dysbaric illness treated at HMNZS PHILOMEL. *SPUMS J* 1990; 20: 226-230

22 Edmonds C and Boughton J. Intellectual deterioration with excessive diving. *Undersea Biomed Res* 1985; 12: 321-326

23 Edmonds C and Hayward L. Intellectual impairment with diving. A review. In *Proceedings of the IXth. International Symposium on Underwater and Hyperbaric Physiology*. Bove AA, Bachrach AJ and Greenbaum LJ Jr. Eds. Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1987; 877-886

24 Anderson G, Holt P, Edmonds C, Lowry C, Cistull P, McKay B, Mistra S and Sutton G. Does non-clinical decompression stress lead to brain damage in abalone divers? *Med J Aust* 1986; 144: 399-401

25 Williamson AM, Clark B and Edmonds C. Neurobehavioural effects of professional abalone diving. *Brit J Ind Med* 1987; 44: 459-466

26 Sutherland AFN, Veale AG and Gorman DF. The neuropsychological problems prevalent in a population of recreational divers one year after treatment for decompression illness. *SPUMS J* 1993; 23: 7-11

27 Moon RE, Kisslo JA, Massey EW, Fawcett TA and Thiel DR. Patent foramen ovale and decompression illness. Undersea Biomed Res 1991; 18(Suppl):15

28 Moon RE and Gorman DF. Treatment of the Decompression Disorders. In *The Physiology and Medicine of Diving. 4th.Edn*. Bennett PB and

Elliott DH. Eds. London: W.B. Saunders, 1993;506-541
29 Fuentes RT and Holmes RA. Reverse radioaerosol/ radioperfusion distribution in pulmonary endobronchial obstruction. *Clin Nucl Med* 1990; 15: 217-221
30 Nakata H, Kimoto T, Nakayama T, Kido M, Miyazaki N and Harda S. Diffuse peripheral lung disease; Evaluation by high-resolution computed tomography. *Radiology* 1985; 157: 181-185
31 Kang BC, Grizz V and Phillips B. Non-wheezing bronchial asthma diagnosed by methacholine challenge. *Am J Med* 1990; 88: 675-677
32 Davis JC Ed. *The return to active diving after decompression sickness or arterial gas embolism*. Bethesda, Maryland: Undersea Medical Society, 1980
33 Moon RE, Camporesi EM and Kisslo JA. Patent foramen ovale and decompression sickness in divers. Lancet 1989;1:513-514
34 Wilmshurst PT, Byrne JC and Webb-Peploe MM. Relation between interatrial shunts and decompression sickness in divers. *Lancet* 1989; 2: 1302-1306
35 Jennett B and Lewin W. Traumatic epilepsy after closed head injury. *J Neurosurg Psych* 1960; 23: 295-310
36 Greer H. Neurological consequences of diving. In *Diving Medicine* Bove AA and Davis J. Eds. Philadelphia: W.B. Saunders, 1990; 223-234
37 Mebane GY and McIver NKI. Fitness to dive. In *The Physiology and Medicine of Diving. 4th.Edn*. Bennett PB and Elliott DH. Eds. London: W.B. Saunders, 1993; 53-76
38 Hickey DD. Outline of medical standards for divers. *Undersea Biomed Res* 1984; 11:407-430
39 Williams TH, Wilkinson AR, Davis FM and Frampton CMA. Effects of transcutaneous scopolamine and depth on diver performance. *Undersea Biomed Res* 1988; 15: 89-98
40 Wood-Smith FG, Vickers MD and Stewart HC. *Drugs in Anaesthetic Practice*. London: Butterworth, 1973.
41 Gorman DF and Acott CJ. Motion sickness in adults and children. *New Ethicals* 1992; Nov: 57-64
42 Acott CJ. Malaria, the "bad air" for divers. *Undersea J* 1993; 3: 50-52 .

Key Words

Decompression illness, treatment sequelae, fitness to dive.

The original version of this paper was presented at the XXth Annual Scientific Meeting of the European Undersea Biomedical Society at Istanbul in 1994.

Dr C J Acott's address is Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000.

WOMEN IN DIVING

Robyn Walker

Abstract

The issue of female fitness to dive is really no different from male fitness and both genders should be assessed on an individual basis. The physiological features exclusive to women which have an influence on diving fitness are few. A literature review covering these topics is presented.

Introduction

Female divers are no longer considered a rarity and the image of scuba diving being a male dominated sport is long past. Previously much has been written on the topic of female divers, as if the very presence of oestrogen imparts an ominous prognosis. However, the issue of female fitness to dive is really no different from male fitness and both genders should be assessed on an individual basis. The physiological features exclusive to women which have an influence on diving fitness are few and a literature review covering these topics is presented.

Performance

Females typically have a lower threshold for peak sports performance than males and generally can produce less power, speed and have lower work capacity and stamina. As a consequence a female can not generally achieve a maximal oxygen consumption per kilogram equivalent to a male.[1]

Females possess a higher percentage of body fat which persists despite training. A 20 year old sedentary female has approximately 25% fat, a trained female 10-15% fat and a trained male 7-10% fat. Trained males have relatively more muscle(40% of total body mass) while comparatively fit women have only 23% muscle.[1]

Thermal Stress

Females are able to conserve energy more efficiently than males. Their increased body fat provides better insulation from heat loss as well as increased buoyancy. Females have fewer sweat glands and sweating begins at a higher core temperature, so conserving heat. However, this increases a female's susceptibility to over heating when sitting, fully kitted up, in the sun.

Females have a lower basal metabolic rate (BMR). Overall, women are more susceptible to heat loss than men

due to an increased conductive heat loss due to their slightly higher surface area to volume ratio, their smaller muscle mass and thus less metabolically active tissue to generate heat during activity.[1] The Korean Ama (female free divers) adapted to their environment, with water temperatures as low as 10°C, and were found to have a higher BMR, higher shivering threshold, greater skin fold thickness, greater peripheral vasoconstriction and the ability to tolerate a lower rectal temperature than females from the same village who did not dive. It is noteworthy that since the introduction of wetsuits in 1976 these adaptations have been lost.[2]

TABLE 1

REPORTED DCI IN INSTRUCTORS

	Number of dives	Cases of DCI (suspected or confirmed)	Incidence
Females	44,154	10	0.023%
Males	43,126	3	0.007%

From Bangasser.[4]

Effect of physiological differences

The effect of these physiological differences on female recreational divers is small. Recreational divers are rarely required to sustain maximal aerobic endurance for prolonged periods. Both male and female divers should learn to dive well within their own physical limitations. Even if females are more susceptible to hypothermia proper equipment should ameliorate this disadvantage.

Decompression Illness

There has been a common perception that women have an increased susceptibility to decompression illness (DCI), perhaps due to their increased body fat percentage, however, reputable studies in this area are lacking.

Bassett[3] looked at the incidence of altitude DCI in the United States Air Force from 1966-1977. Of these 104 cases, 32 (31%) occurred in women. Statistically significant differences were found between males and females with DCI in weight, height and body build. A larger number of women reported a history of vascular or migraine headaches, more females experienced the onset of bends at altitude and females experienced more cutaneous manifestations than males. The author concluded females were four times more susceptible to altitude DCI than males, have more cutaneous symptoms, have more rapid onset of bends pain and have more recurrences and more lasting effects of DCI compared to males exposed to the same altitude exposure. The application of these results to hyperbaric exposures is uncertain as diving and altitude exposures result in different inert gas saturation profiles.

Bangasser[4] conducted a retrospective study of 649 female divers by questionnaire. The reported incidence of DCI in female instructors was compared with male instructors and the findings are presented in Table 1.

These results suggested a 3.3 fold greater incidence of DCI in females. However, there are several major weaknesses in this study; the study is retrospective and therefore not all conditions are known, cases with insufficient data may not have been fully scrutinised, no criteria existed to determine if DCI actually occurred and the diagnostic evaluations were based solely on the respondents' replies. It is highly unlikely that divers incapacitated by accidents or those suffering from fatalities were accounted for and later studies have also shown that if males and females are of comparable fitness the differences in rates of DCI disappears.

Zwingleberg et al.[5] compared females with males in a review of the incidence of DCI in deep diving at the Naval Diving and Salvage Training Centre (NDSTC). Deep diving was defined as 38 -86 m (125-285 ft) on air and 36-90 m (120-300 ft) on heliox. Bottom times were mostly less than 20 minutes with dive duration ranging from 8 minutes-2 hours 6 minutes. The study was in two parts, the first comparing females to males in a general review of DCI incidence on deep dives, and a second female-male buddy matched analysis on deep dives involving females.

Twenty nine female divers took part. No female divers developed DCI while 8 male divers did, 4 on air and 4 on heliox. Three of the cases were classified as Type I DCI and 5 as Type II. The overall incidence of DCI for male deep dives was 1% (1.91% for heliox and 0.6% for air). For the male-female matched pairs there was a 1.3% increased risk for DCI in males. The authors concluded women divers are at no greater risk of developing DCI under similar bounce dive exposures but caution against the extrapolation of these results to all dive exposures. United States Navy female divers have sustained DCI on long duration, experimental or saturation dive profiles as have males. In the NDSTC study dives the exposures were of short duration with only short half-time compartments becoming supersaturated. The authors concluded these results can be applied to sports diving models. Saturation and experimental dives may well be different as the "slow" compartments will have the major influence on decompression rates and the higher body fat percentage in females may well increase the risk.

DCI has been associated with altered clotting activity, specifically increased platelet aggregation.[6] Markham et al.[7] studied the behaviour of washed platelets during different phases of the menstrual cycle and the differences in aggregation of male and female washed platelets in response to decompression stress and arachidonic acid. Their study did not support the possibility that platelets aggregate differently during the menstrual cycle, however, it did support the existence of a sex difference in platelet aggregation that is not altered by external factors, e.g. decompression stress. Females compared to men do have increased sensitivity of platelets to aggregation. The importance of this increased sensitivity to the development of DCI in females is unknown.

No evidence exists in the literature of a difference in susceptibility to pulmonary barotrauma or cerebral arterial gas embolism between the sexes and the incidence of patent foramen ovale is equal in both.

Menstrual Cycle

For most healthy active women, changes associated with their menstrual cycle are negligible and cause minimal interruption to their lives. Severe "premenstrual syndrome" if disabling, may disqualify a woman from diving for this part of their cycle, however, this occurs in only a minority of females.

Brown[8] compiled a small survey on female divers "The Michigan Sea Grant Survey" and found that 89.9% of females dive whilst menstruating without any physical or psychological problems. Of those who did not dive 4.8% were menopausal or had not had the opportunity to dive whilst menstruating.

Rudge[9] examined the role of menstrual history in the development of altitude chamber decompression sickness (DCS) in USAF female personnel undertaking hypobaric exposures. Records were available for 81 personnel. This study demonstrated a clear correlation between the incidence of DCS and time since start of the last menstrual period. A higher number of subjects developed DCS (both type I and type II) earlier in the menstrual cycle (0-4 days). He concluded women were at higher risk of developing altitude DCS during menses, with the risk decreasing linearly as the time since the last menstrual period increases. This study did not address likely mechanisms for the findings and its application to female divers is unknown.

Oral contraceptive pills (OCP) are known to be associated with an increased risk of thromboembolism as they accelerate blood clotting, increase platelet aggregation and are associated with an increased blood concentration of some clotting factors.[10] There is, however, no evidence to suggest that OCPs alter a female's susceptibility to DCI and the risk of becoming pregnant is of far greater concern.

There is no evidence to suggest menstrual flow attracts sharks and no evidence of increased shark attacks on menstruating divers.

Pregnancy

Diving during pregnancy is a controversial subject. Questions such as, "Does diving increase the risk of foetal abnormality?", and, "What is the incidence of foetal DCI?" remain unanswered.

Bolton[11] surveyed 208 female divers of whom 136 had dived during pregnancy. The average depth of the dive was 13 m (42.6 ft), Twenty four had dived to 30 m (99 ft) during the first trimester. The frequency of birth defects was significantly greater in pregnancies during which females dived but this was still within the range of the normal population.

Fife et al.[12] inserted a Doppler probe around the umbilical vessel in near-term foetal sheep. Compression and decompression produced marked evidence of foetal bubbling while no evidence of DCI was noted in the mother. They concluded that bubbles were much more likely to form in the foetus than the mother. Other researchers initially confirmed these findings, however, once control experiments were performed, it became obvious that bubbling only occurred in the instrumented animals. Current opinion is that bubbles are less likely to form in the foetus than the mother. If the mother does develop DCI, bubbling in the foetus may occur. Bubbles in the foetus are likely to be more ominous than in the mother due to the differences in foetal anatomy and physiology. The lungs in an adult act as an effective bubble filter whilst in the foetus most of the blood bypasses the lungs by passing through the ductus arteriosus and patent foramen ovale. Therefore any bubble in the foetus may pass directly to the cerebral circulation as a cerebral arterial gas embolism.

Animal studies have shown an increased rate of foetal loss when the mother is exposed to a decompression insult.[13] The impression is the closer to term the higher the risk.

There are no reports in the diving literature of air embolism affecting a pregnant diver. Taylor[1] reported 15 cases, in the obstetric literature, of air embolism from sexual encounters, all occurring in young women in their second or third trimester where air was forcibly blown into the vagina. In 12 of the 15 cases there was maternal and foetal death. One patient was treated with hyperbaric oxygen for 39 hours with resultant moderate neurological defects in the mother and a stillborn infant. The conclusion was air embolism of the uteroplacental bed appears lethal.

Other points for the pregnant potential diver to consider are the effects of morning sickness and pregnancy induced gastro-oesophageal reflux, both of which are associated with an increased risk of vomiting underwater.

The fit and function of dive equipment may be compromised as the pregnancy progresses. Where does a woman secure her weight belt?

Uterine blood flow may be compromised during periods of increased demand and increased sympathetic activity, both of which occur during diving. Erratic abrupt shifts in flow dynamics are more likely to compromise uterine blood flow than a gradual increase or sustained aerobic activity. Marine envenomation carries undefined foetal toxic effects and specific antitoxins may also hold risks for the foetus.

Foetal risks of hyperbaric oxygen

A potential problem for the foetus is oxygen toxicity. Diving on compressed air exposes the foetus to an increased partial pressure of oxygen and the mainstay of treatment regimes for diving accidents is recompression on 100% oxygen. In the foetus, ductus arteriosus blood flow decreases dramatically when the oxygen tension in the pulmonary circulation increases. The foetal pulmonary bed is exquisitely sensitive to oxygen tension and responds with vasodilatation when the oxygen tension rises. There is consequently a shift from a foetal to a neonatal blood flow pattern.[1] This shift will reverse when the oxygen tension falls but it is unknown whether this has long term sequelae for the foetus. Therefore, even though pregnancy does not clearly increase the maternal incidence of DCI, the foetus may be at severe risk if a diving accident occurs.

Post-partum

There are no contraindications to women diving while breast feeding, however it is generally recommended that women do not dive until six weeks post partum to avoid intrauterine infection.

Mammary implants.

Vann et al.[14] exposed mammary implants to various simulated dive profiles followed by altitude exposures to simulate aircraft travel. The implants were observed for bubble formation and volume changes. Minimal volume changes occurred after each dive although numerous bubbles formed reaching their maximal size in 3 hours. When the implants were exposed to high altitude following a dive significant volume changes occurred. The volume changes were least for saline and greatest for gel saline implants. The authors concluded that bubble formation in breast implants might occur after shallow saturation diving but it is unlikely to result in tissue damage. However,in the unlikely event of prolonged deep saturation diving followed immediately by flying in an unpressurised aircraft at 9,100 m (30,000 ft) the resultant bubble formation might be of sufficient magnitude for tissue trauma to occur!

Summary

The assessment of fitness to dive of a fit non-pregnant female is identical to that of a fit non-pregnant male. The true risk of diving while pregnant, for both the mother and foetus, is unknown. All women divers should be advised of the potential risks of diving when pregnant. Most women will probably accept pregnancy as a 9 month self limiting condition and elect not to dive along with reducing their alcohol intake and medication usage.

References

1 Taylor MB. Women and diving. In chapter 13, *Diving medicine. 2nd ed*. Bove A and Davis JC Eds. Philadelphia; Saunders, 1990

2 Park YS. Korean woman divers revisited: current status of cold adaptation. *Undersea Biomed Res* 1981; 8 (Suppl): 25

3 Bassett B. Safe diving equals fun diving; prescriptions for diving women. *SPUMS J* 1979; 1: 9-14

4 Bangasser S. Decompression sickness in women. *Women in diving 35th UHMS Workshop* 1986: 65-71

5 Zwingelberg KM. et al. Decompression sickness in woman divers. *Undersea Biomed Res* 1987; 14 (4): 311-317

6 Edmonds C, Lowry C, and Pennefather J. Chapter 12. *Diving and subaquatic medicine 3rd ed*. Oxford: Butterworth-Heinemann, 1992

7 Markham SM, Dubin NH and Rock JA. The effect of the menstrual cycle and of decompression stress on arachidonic acid-induced platelet aggregation and on intrinsic platelet thromboxane production in women compared to men. *Am J Obstet Gynecol* 1991; 165 (6): 1821-1829

8 Brown E. The Michigan sea grant program. Women diver survey: preliminary results. *Proceedings PADI women in diving seminar* 1977.

9 Rudge FV. Relationship of menstrual history to altitude chamber decompression sickness. *Aviat Space Environ Med* 1990: 61: 657-659

10 Murad F and Kuret J. Estrogens and progestins. In *The pharmacological basis of therapeutics 8th ed*. Gilman A et al Eds. New York: Pergamon Press, 1990

11 Bolton ME. Scuba diving and fetal well being: a

Hyperbaric Technicians And Nurses Association

Two conferences At The Hotel Grand Chancellor (ex-Sheraton) Hobart

Oxygen, "Understanding the Risk" 28th and 29th August 1996

A two day Seminar on the use of medical oxygen. This will be of special interest to all who handle oxygen equipment.

Invited speakers from Australia and overseas will present on this complex topic. Subjects to include:

Oxygen cleaning
Fire hazard
Oxygen analysis
and much more.

Dr Bill Hamilton will be a speaker and due to his knowledge and background he will undoubtedly have some useful information for the technical diving community.

Details available from:
Bob Ramsay or Steve Goble,
Hyperbaric Medicine Unit,
Royal Adelaide Hospital,
Adelaide, South Australia 5000.

Phone (08)222 5514
Fax (08)232 4207

4th Scientific Conference of Diving and Hyperbaric Medicine 30th And 31st August 1996

Following on from their highly successful Melbourne Conference the HTNA is again providing a scientific meeting of growing International standing.

The Hyperbaric day is the 30th. and the diving day is the 31st.

The overseas invited speakers this year are:
Professor Dirk Bakker, University of Amsterdam
Dr Bill Hamilton
Dick Clarke, Carolina Hyperbarics

Details available from:
Sean Rubidge
Hyperbaric Unit
Royal Hobart Hospital , Hobart

Phone (002) 388 322
Fax (002) 347 684

BREATH-HOLD DIVING
AN INTERNATIONAL SYMPOSIUM
to be held on 6th and 7th of December 1996 in Paris
sponsored by the FEDERATION FRANCAISE D'ETUDES ET DE SPORTS SOUS-MARINS (FFESSM) and the WORLD UNDERWATER FEDERATION (CMAS)

The symposium is to study all forms of breath-hold diving, and the teaching and training courses for each, to increase knowledge of incidents and accidents for better prevention.

Papers are called for on the following subjects

Breath-hold diving physiology
Pathophysiology of incidents and accidents of breath-holding diving
Characteristics and constraints of underwater competitive sports: synchronised swimming, underwater fin swimming, underwater hockey, target shooting and spear fishing
Specificities of pure breath-hold diving; static test, dynamic test, constant weight, variable weight and "no-limits"; possibility of records and/or of competitions
Organisation and security for deep diving
Training and ventilation control techniques for breath-hold diving
Psychological aspects of breath-hold diving

For further information contact Dr Michel Leloup, Commission médicale FFESSM Ile-de-France, 21 rue Voltaire, 93100 Montreuil, France

survey of 208 women. *Undersea Biomed Res* 1980; 7(3): 183-189

12 Fife WP, Simmang C and Kitzman JV. Susceptibility of fetal sheep to acute decompression sickness. *Undersea Biomed Res* 1978; 5: 287-292

13 Lehner CE, Rynnning C, Bolton ME and Lanphier EH. Fetal health during pregnancy in sheep: staged and rapid decompression. *Undersea Biomed Res* 1982; 9 (Suppl): 46-47

14 Vann RD, Riefkohl R, Georgiade GS and Georgiade NG. Mammary implants, diving, and altitude exposure. *Plastic & Reconstructive Surgery* 1988; 81(2): 200-203

Key Words

Women, fitness to dive, physiology, decompression illness.

Lieutenant Commander Robyn Margaret Walker, MB BS, DipDHM, RAN, is Officer in Charge, Submarine and Underwater Medicine Unit, HMAS PENGUIN, Middle Head Road, Mosman, New South Wales 2088, Australia. Telephone (02) 9960 0315. Fax (02) 9960 4435.

MARINE MEDICINE SEMINAR '96

JUNE 29TH - JULY 7TH 1996

A Seminar on Aquatic and Hyperbaric Medicine, Marine Animal Injuries and Diver's Medical assessments will be held in Bali, Indonesia, at the Bali Padma Hotel, Legion, over 5 half days (morning sessions).

Australian and Indonesian experts in Diving and Hyperbaric Medicine will lecture at the Seminar. RACGP point allocation is pending.

Excellent fare and accommodation packages apply but places are limited due to school holidays in Eastern States.

Phone Brisbane -(07)-3376-1056 for further details

Information and application forms for courses and seminar can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road,
Jindalee, Queensland 4047.

Telephone (07) 3376 1056
Fax (07) 3376 1056

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

DIVING IS ADVENTURE:
(except in Queensland)

Bob Halstead

Diving is not amusing nor frivolous. It cannot be conducted casually nor without thought and intelligence. People dive, not for "fun", but for ADVENTURE. Nothing to be ashamed of here, this is a natural urge, but there are consequences. Pascal noted three hundred years ago that

"ALL THE TROUBLES OF PEOPLE ARE CAUSED BY ONE SINGLE THING, WHICH IS THEIR INABILITY TO STAY QUIETLY IN A ROOM"

He recognised that to seek adventure is a product of being human, but he also recognised that adventurers should anticipate "trouble". I admit he used the word "men" instead of "people". I changed it, not to be politically correct, I have nothing but contempt for that trend in intellectual fascism, but because diving is one of the few physical activities where women are equal, and often superior, to men.

As we seek adventure the trouble that should concern us is the "risk" that the adventure entails. Risk increases as soon as you close the front door and head off to the dive site, then increases even more as you get into the water and descend. The risk associated with diving changes as various physical factors change, for example risk increases with depth, if there is a current, if the water is cold and murky and so on. But increased risk does NOT necessarily imply "danger", a lack of "safety", and it is this confusion which has led to some quite incredible nonsense being promulgated by Governments and others in the name of improved "safety".

Risk is the potential for injury to occur.

Risk is calculated by considering the sum effect of the various hazards encountered when diving. The diver should not be included in the risk assessment, because, as you will see, it confuses the determination of "safety" from the individual diver's point if view. For a particular dive at a particular time the risk is thus the SAME for any diver (or even non-diver!) who attempts the dive. But the "danger" or "safety" depends on WHO is making the particular dive and how well prepared they are to overcome, or neutralise, the risk.

This is very easy to illustrate. If we imagine a dive in shallow, clear, calm, warm conditions devoid of marine life and any other hazards we would consider this a low risk dive. However the same dive would be deadly dangerous for any "diver" who did not understand the consequences of breath holding on ascent. I am going to clarify what is meant by "safety" and "danger" so you can understand what "Adventure" really is, but first I want to make it clear that adventure is NOT throwing yourself into a situation and seeing if you survive. That is "Russian Roulette", not adventure. There is also not much point in talking about safety AFTER the event (except to analyse mistakes). If you are about to participate in an adventure you need to be able to PREDICT that the adventure will be safe for you before embarking on it. That is the whole point of this analysis, we want to be able to say, individually, that the dive we are about to make is going to be "safe".

Before any and every dive, a diver should be able to say:-

"This dive will be safe <u>for me</u>"

This means "it is unlikely (but not impossible) that I will be injured on this dive".

A dive which is dangerous <u>for me</u> is one where "it is likely (but not inevitable) that I will be injured".

The reason we cannot predict perfect safety is twofold. First unpredictable events do occur, some people staying "safely" in their quiet rooms have had an aircraft land on their heads, or their homes invaded. Secondly PEOPLE MAKE MISTAKES. Mistakes are another part of being human and no amount of rules or regulation will change this, although many do imagine they can legislate perfect safety (they may safely be called fools).

Now I can define adventure:-

Adventure is the art of safely experiencing increased risk.

Isn't that beautiful ? We have admitted that risk is necessarily a part of adventure, and now we can see what needs to be done to "improve safety".

For you to be able to predict that "**This dive will be safe <u>for me</u>**"

1 Consider the hazards and calculate the risk for this particular dive.

2 Assess yourself:-
"Do I have the skills, knowledge and equipment necessary to overcome the risk?"

A safe diver :-

Is one whose skills, knowledge and equipment are sufficient to overcome the risk for the dives attempted.

A dangerous diver:-

Is one whose skills, knowledge and equipment are insufficient to overcome the risk for the dives attempted.

If you understand logic you can see that there is no such thing as a "Safe Dive" just "Safe Divers", and of course no such thing as a "Dangerous Dive" just "Dangerous Divers". You can also see how diving risk should "be managed", we neutralise it with skills, knowledge and the right equipment. We do NOT remove the risk, nor lessen it, although this may be the best approach for commercial divers, or workers in a factory, but they are not seeking adventure! By the way, for sake of brevity I am including fitness and health considerations under "skills".

Diving is not dangerous

A dive may be high risk

A dive may be low risk

The danger depends on who is making the dive

Does the diver have the skills, knowledge and equipment necessary to overcome the risk?

If the diver does:-

The dive is safe for that diver

If the diver does not:-

The dive is dangerous for that diver

This shows us how we can make diving safer. We need to concentrate our efforts on teaching divers to recognise hazards and to be able to assess the risk that the hazards present for the particular dive to be attempted. We also need to teach divers how to realistically assess their own ability to ensure that they recognise when they do have the necessary skills, knowledge and equipment, and when they do not, in which case the dive should not be attempted. Of course we also need to be able to improve the skills and knowledge of divers by better training.

Unfortunately this is not what is happening. We are told that risk must be "controlled" or "managed" but what actually happens is that the risk is being REDUCED and this is lauded as "safe practices". This is not contributing at all to the cause of improving diver safety, it contributes to the cause of ELIMINATING THE ADVENTURE. Please allow me to make this clear.

The mountaineering equivalent would be to instruct mountaineers to climb Mount Snowdon instead of Mount Everest. The motor racing equivalent would be to restrict speeds to 100 kph. The parachuting equivalent would be not to allow any jumps from higher than one metre ..... Have I made the point? YES all these instructions would REDUCE INJURIES but would they make the sports SAFER? The answer is NO because the sport no longer exists. Mountaineering is about climbing mountains not hills, motor racing is about going as fast as you can and parachuting is about jumping from heights where if your parachute fails, you die. The ADVENTURE has disappeared.

Let us go back to Pascal, he was a smart lad. People will seek adventure in their lives. In some the drive is stronger than others, but if attempts are made to frustrate, inhibit or suppress these adventures then watch out for trouble. If adventures such as diving are restricted, then people will turn to other, possibly less socially acceptable, forms of adventure.

Or they will remove themselves from the restrictions and from the opportunity to learn professionally how to be safe divers. Here is the danger created by such organisations as the Queensland Division of Workplace Health and Safety when they start sticking their nose into ADVENTURE. If I wished to start an adventure diving BUSINESS in Queensland and pass on some of the knowledge and skills I have learned, I create a "Workplace", I therefore have to conform to a "Code of Practice", created by a committee. The Code creates a "Duty of Care" which makes me liable to criminal prosecution if someone "in my care" is injured, even if the code was followed. (The recent case involving the owner of a crocodile farm in Queensland whose employee disobeyed instructions and got eaten, demonstrates the fanatic desire of the Division to prosecute, causing the innocent owner unjust expense and stress. The owner eventually won the case.) Inevitably someone "in my care" will injure themselves sooner or later because (a) stuff happens and (b) people make mistakes. The effect of the "Duty of Care" transfers the blame from where it belongs, the adventurer, to me, so now I could be taken to court and subjected to an inquisition by people who know a lot less about diving than I do. Some of them think you have to wear a snorkel all the time. I could lose my house too. Well it seems to me that the odds are too much against me so I will not be starting an adventure diving school in Queensland and those who want to learn more about the Art of Safely Experiencing Increased Risk will have to learn the hard (and dangerous) way.

Having recently dived at a very efficient and idyllic resort on the Big Barrier Reef (The world's largest partly living coral and rubble reef! By the way there were lots of turtles but they all seemed to be afflicted by plastic tag disease probably caused by an outbreak of scientists.) I can

report that indeed the Adventure is disappearing. I was restricted to a maximum depth of 18 m with a limited bottom time, and exactly the same restrictions would apply if I had just passed my first diving certification. This is the really dumb thing about limits, they do not discriminate between divers of various abilities, and, as I have pointed out, 18 m is plenty of depth for an incompetent diver to kill themselves. The Workplace legislation is such a monster that many operators dare not step out of line and the resulting experience they are forced to offer is modest. The operator and guides at the resort, all great people, have my sincere sympathy, no doubt they will soon be able to apply for stress compensation caused by Duty of Care responsibilities unfairly inflicted on them.

So instead of scuba diving I spent a lot of time snorkelling around the island, which was fantastic, however, having read the new Code of Practice for snorkelling I can see that they will have to restrict this adventure soon too, at least they will when they read the Code and realise its implications. To reinforce my point please read the snorkelling Code if you get the chance and note there is no prohibition of non-swimmers, which I cite as evidence that the real safety issues such as skills are NOT the concern of the legislation. It is difficult to reach any other conclusion than that the prime aim of the legislation is not in fact safety, but to find SOMEONE ELSE TO BLAME when something goes wrong.

Now for a bit of humour, grab this. In the SPUMS Journal of September 1995 Mr Rod Punshon, reported to be a member of the Diving Industry Workplace Health and Safety Committee, listed the measures the Pro Dive facility in Cairns, of which he is director, used to achieve their "excellent" safety record up to October 1994 (which, by the way, is nowhere near as good as mine in PNG, and we offer high risk diving). Now I see reported in Dive Log of November 1995 that his Pro Dive Facility was among 20 businesses and individuals who received (Queensland) Workplace Health and Safety Annual Best Practice Awards. The measures taken by Mr Punshon that he attributes to improving safety are listed in the SPUMS Journal as, and I quote (the italics are mine) :-

1 Strict adherence to "deepest dive first". This policy is *enforced* by a *mandatory* break of a minimum of 12 hours out of the water for *any deviation from this basic policy*.
2 A *limiting* of maximum depth for certified divers, unless *under strict supervision* or training, to 30 m.
3 *Limiting* alcohol intake and encouraging more rest. (This would not be popular with SPUMS members! B.H.) We *actively discourage* partying on board during trips.
4 We now *calculate and check* all dive profiles and *ensure compliance* with "no decompression" table *limits* on each dive.
5 Any accidental entry into decompression is *penalised* by a minimum of six hours out of the water, *depending on severity*.

Well this does not sound like fun, and it does not sound like adventure either, nor even as though the people involved might be grown ups. But such insight into human nature! And this is award winning stuff! Question:- If you want people to behave responsibly the way to do this is to treat them like ..... Please send your answers to the Queensland Government.

In the meanwhile, offered in the spirit of "Those aren't rules, THESE are rules", here are some of mine.

HALSTEAD'S GOLDEN RULES OF DIVING

1 Diving is adventure.

2 Write your will before you become a diver.

3 Never dive deeper than your IQ (Imperial units and you may add half your age for every 1,000 dives made).

4 Never dive with psychopaths.

5 Avoid the surface whenever possible.

6 Come up slow and stop in shallow water before surfacing.

7 Do not run out of breathing gas, carry a completely redundant unit.

8 Remember most "safety" devices can cause injury (particularly BCDs) simplicity is often best.

9. Do not dive dangerously. Assess the hazards, calculate the risk, know you have the skills, knowledge and equipment to overcome the risk.

10 Know yourself, know diving. The more you know, the longer you live.

11 Freedom means sometimes choosing not to dive.

12 Take the blame for whatever happens to you.

Key words.
Safety, general interest, reprint.

This paper is reprinted, with Bob Halstead's permission, from TELITA CRUISES NEWSLETTER January 1996.

Bob and Dinah Halstead's address is PO Box 141 Earlsville, Cairns, Queensland 4870, Australia.

ASPECTS OF NEUROLOGICAL DECOMPRESSION ILLNESS: A VIEW FROM BETHESDA

J R Broome

Abstract

A minority of divers with neurological decompression illness (DCI) fail to improve with recompression treatment. This is particularly seen in cases where features of severe spinal cord injury develop soon after surfacing. Haemorrhage into the spinal cord is implicated in the pathogenesis of these cases and evidence is presented that supports the view that the bleeding coincides with shrinkage of autochthonous bubbles. The role of hyperbaric oxygen therapy in the treatment of spinal cord DCI is discussed with reference to possible benefit in ischaemia-reperfusion (I-R) injury. Similarities and differences between the tissue injury of dysbaric and conventional spinal cord injury are outlined. The implications of advances in drug therapy for conventional spinal cord trauma are considered in the context of their potential application to treat neurological DCI.

Introduction

Diving medicine retains enormous challenges, and exchange service with the United States Navy in the Naval Medical Research Institute (NMRI), Bethesda, Maryland, provides a Royal Naval medical officer with unique experience in military diving medicine that is no longer available in Britain. The following account briefly summarises the author's personal view of several issues related to neurological decompression illness (DCI) and its treatment.

Most patients with DCI respond well to conventional recompression therapy, but treatment is ineffective for an important minority of cases. Clinically, these cases tend to have neurological features suggestive of spinal cord injury and are either cases with early onset of severe signs or cases where there is significant delay before initiating recompression therapy. There is no generally accepted explanation for these treatment failures.

The pathological mechanisms for dysbaric central nervous system (CNS) injury are complex and remain controversial.[1] Where the condition is due to excess gas burden (as distinct from pulmonary barotrauma with gas emboli) several mechanisms may contribute to the disease process in varying degree, depending on the circumstances. Extra-vascular (autochthonous) bubbles are most likely in short latency cases, gaseous emboli, venous infarction, and ischaemia-reperfusion effects being predominant in cases with delayed onset. However, for many years it was assumed that the main problem was of ischaemia due to the persistence of inert gas bubble emboli in blood vessels.[2,3] In refractory cases, this assumption led to therapeutic efforts to eliminate any causative bubbles as rapidly and completely as possible by recompression to greater pressures (depths), by lengthy saturation treatments, or by the breathing of exotic gas mixtures.[4] Use of these measures has been largely empirical and, unfortunately, none of them has proved convincingly effective. Indeed, there has been little real advance in the treatment of DCI since the introduction of the short oxygen recompression tables (RN Tables 61 and 62)in the late 1960s and early 1970s. Difficulty in standardising the terminology used to describe the various presentations of DCI[5] have frustrated efforts to set up appropriately randomised therapeutic trials.

The author's work in NMRI has been to develop an animal model of neurological DCI with which to evaluate treatment strategies and identify modifiable risk factors for DCI. The animal used for the model is the pig, which has many anatomical and physiological similarities to humans.[6]

The pigs undergo a simulated "dive" to a pressure equivalent to 200 feet of seawater (fsw) (61.2 msw) (612.6 kPa), for 24 minutes, in a dry compression chamber, breathing air. This pressure exposure produces a 70-75 % incidence of neurological DCI. Pigs that develop the condition are given appropriate sedation and iv. fluids, and are treated by recompression and oxygen on RN Table 62 (USN Table 6) in a manner analogous to human divers. The time from diagnosis of DCI to commencing treatment is 10-20 minutes. The success of treatment is judged 24 hours later by assessing the pigs' ability to run on a treadmill. Pigs are then anaesthetised, euthanised, and undergo a detailed pathological examination.

Haemorrhage and the pathogenesis of dysbaric spinal cord injury

In 1908, Blick reported one of the largest series of autopsies on divers with fatal DCI.[7] When describing the gross appearance of the spinal cords from these cases, he wrote: "... it looks as if one has stippled the face of the section (of the spinal cord) with a fine knife or needle ... with this condition is nearly always associated haemorrhage of greater or less extent". He goes on to recount: "... they (the haemorrhages) may range in size from mere points of blood to, as I have seen in nine cases, large haemorrhages practically cutting the cord in two". Several other authors, notably Brooks in 1907,[8] Smith,[9] and Jaminet and Clarke in the 1870s,[10,11] all describe acute haemorrhage in the cord or its membranes as a marked feature of DCI. Despite such descriptions, until recently[1] the role of CNS haemorrhage in the neuropathology of DCI in divers received little emphasis in most diving medicine texts.

During our studies of neurological DCI in pigs, it was soon noted that those pigs with early onset of neurological signs were often little improved by recompression treatment. At necropsy, these refractory cases were associated with petechial haemorrhages, grossly visible in the spinal cord (Figure 1), which appeared remarkably similar to the historical descriptions of human cases. On histological examination of the spinal cords from these pigs, microscopic haemorrhages such as those illustrated in Figure 2 were a typical finding. Pigs that responded well to recompression treatment invariably had no or minimal haemorrhage in their CNS on histopathological examination.

Further information is available: We investigated the timing of the haemorrhage in an attempt to differentiate bleeding due to mechanical disruption of micro vessels from bleeding into areas of infarction.[12] Blood was taken from each of 15 pigs and the erythrocytes were labelled with a fluorescent marker. The labelled red cells were then reinjected into the 15 pigs at different stages in the disease process; before diving in three; on DCI onset in three; immediately before recompression treatment in three; and 10 minutes after reaching treatment pressure (60 fsw) (18 msw) (183.8 kPa) in the remaining six pigs. On the day after diving, pigs were euthanised, perfusion fixed, and necropsy was performed. Frozen sections of spinal cord were made. Areas of haemorrhage were located by

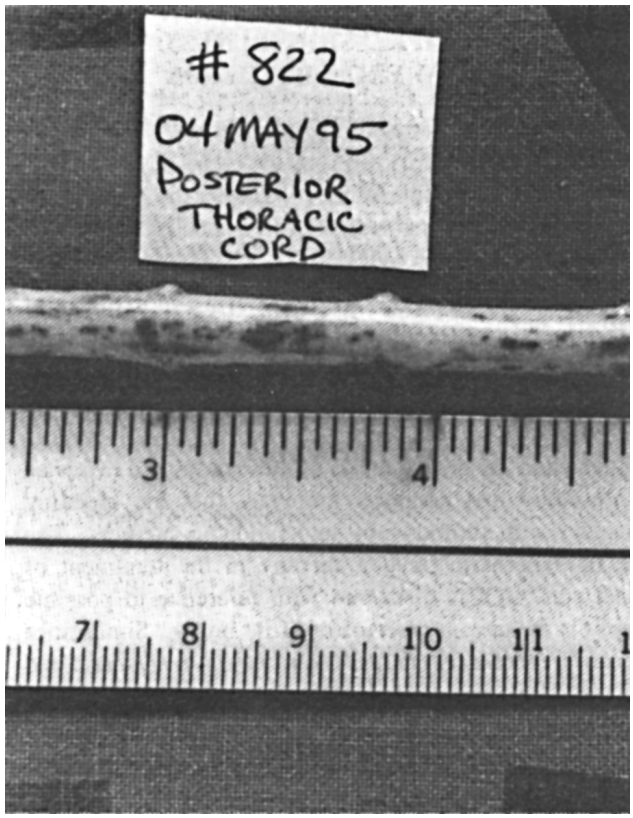


Figure 1. Spinal cord with petechial haemorrhages.

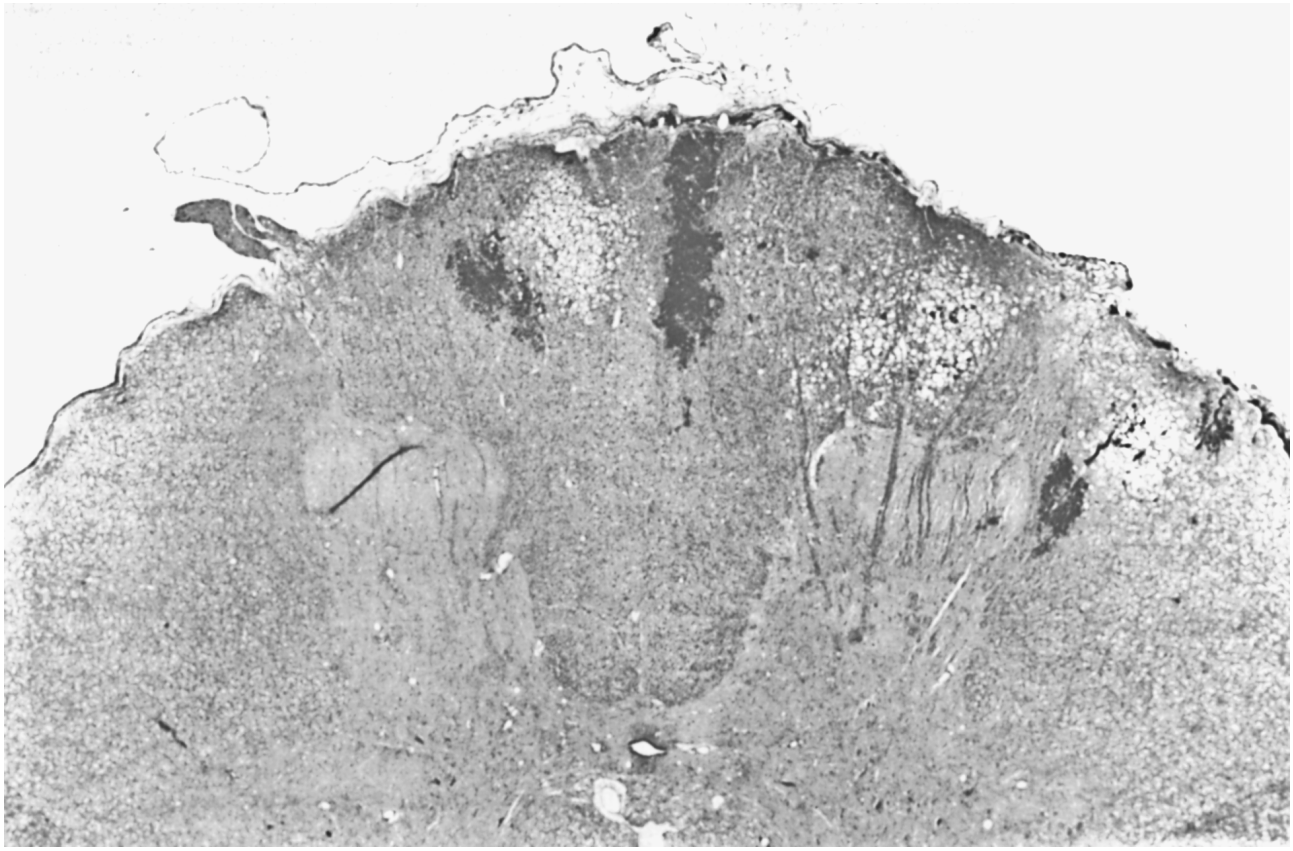


Figure 2. Cross section of spinal cord with multiple white matter haemorrhages (x25 H & E).

microscopy of frozen sections stained with haematoxylin and eosin, then the same areas of haemorrhage in adjacent, unstained, frozen sections were examined by fluorescent microscopy for the presence of labelled erythrocytes. The concept was that fluorescent cells would be absent from haemorrhages that had formed before injection of the labelled erythrocytes, but fluorescence would be present if the haemorrhages took place while labelled erythrocytes were in the pig's circulation.

Labelled red cells were found to be present in the haemorrhagic cord lesions of all nine pigs reinjected *before* recompression treatment, but were absent in the six pigs where the labelled cells were reinjected 10 minutes *after* recompression. This finding suggests that the haemorrhage coincided with recompression. A mechanism whereby autochthonous bubbles disrupt micro vessels, which then bleed when the bubbles shrink on recompression, would explain this finding. Such a mechanism can also explain why some human cases deteriorate rapidly during or soon after the start of recompression treatment. The sudden relapse seen in some cases after, or in the late stages of initially successful, recompression treatment can be explained by secondary haemorrhage. One can also speculate that, in the absence of recompression, bubbles will shrink naturally over time and in cases where micro vessels have been damaged, delayed bleeding may occur, perhaps into an area of infarction or ischaemia.

In retrospect, haemorrhagic lesions have been described in previous models of acute DCI in dogs and goats,[13,14] and a photograph of petechial haemorrhage in the cord from a human victim of severe neurological DCI, who died six days post-dive, has recently been published.[15] It seems likely that severe, short latency, human DCI shares a common pathology with the animal models; namely, the potential for acute haemorrhage into the spinal cord. However the clinical significance of this has not been clearly stated: cases where haemorrhage has occurred are likely to be resistant to standard treatment; recompression and oxygen will shrink bubbles and increase tissue oxygenation, but the consequences of haemorrhage into CNS tissue will not resolve acutely.

The above findings do not, of course, imply that we should avoid recompressing patients with early onset of neurological signs for fear of precipitating haemorrhage, but they have implications for both the management and prognosis of diving cases in certain circumstances. For instance, the understanding that CNS haemorrhage rather than just gas bubbles may contribute to the clinical condition of the patient, might alter a decision to compress a severely ill or deteriorating patient beyond the 18 msw (60 fsw) pressure of Table 62, and thereby commit them to a lengthy saturation decompression. The question that defines the clinical management rationale in such cases subtly shifts from, "Is recompression treatment good for eliminating bubbles?" for which the answer is "Yes" to the question, "Is hyperbaric oxygen good for treating nervous system dysfunction associated with haemorrhage?" to which the answer is '"Possibly, but we don't really know".

Hyperbaric oxygen

Hyperbaric oxygen (HBO) therapy, where the patient breathes oxygen in a pressurised chamber, is the main treatment modality for acute DCI. The initial beneficial effect of HBO is generally accepted: tissue oxygenation in general, and the oxygenation of injured or ischaemic tissue in particular, is improved due to simple diffusion. Arteriolar vasoconstriction lowers capillary pressure and limits oedema. Simple recompression shrinks bubbles, while the raised partial pressure of oxygen accelerates the elimination of inert gas by enhancing the diffusion gradient between tissues and blood. Animal studies suggest that physical gas bubbles are rapidly eliminated from CNS tissue, even in the absence of recompression.[1] The injury that remains when the bubbles have gone results from the mechanical compression and distortion of CNS tissue that they caused, the damage to vascular endothelium, activation of inflammatory mechanisms and the consequences of micro-vascular stasis. Histologically, the acute injury typically manifests as disruption and oedema of axons, haemorrhage, ischaemia/infarction, or a combination of these (Figure 3).[16] Repeated recompression treatments of DCI victims are carried out under the assumption that HBO is of benefit in these conditions. This is the accepted standard of care, but there have been no appropriately controlled studies to confirm the efficacy of repeat treatments. The difficulty is in distinguishing the clinical improvement that frequently occurs over time in untreated cases, from a therapeutic effect of HBO.

Despite the comments above, there is growing indirect evidence of mechanisms by which HBO could benefit the tissue injury in spinal cord DCI. Whatever the mechanism of injury, reversible microcirculatory stasis may occur and initiate a complex series of events resulting in the "no-reflow" phenomenon or ischaemia-reperfusion (I-R) injury. Our knowledge of the mechanisms of I-R injury derives from many areas of biomedical research,[17] but evidence for clinical efficacy of HBO in this condition comes mainly from work in the context of reconstructive surgery.[18,19] Leucocytes are known to adhere to damaged vascular endothelium at the site of I-R injury[20] and promote cell damage by production of oxygen free-radicals, which induce lipid peroxidation.[21] Lipid peroxidation is a process that spreads across cell membranes and disrupts the physiological ionic gradients across the membrane. It impairs the normal function of phospholipid-dependent enzymes and, if sufficiently severe, results in membrane lysis.[21] In addition to oxygen radical production, adherent leucocytes are known to

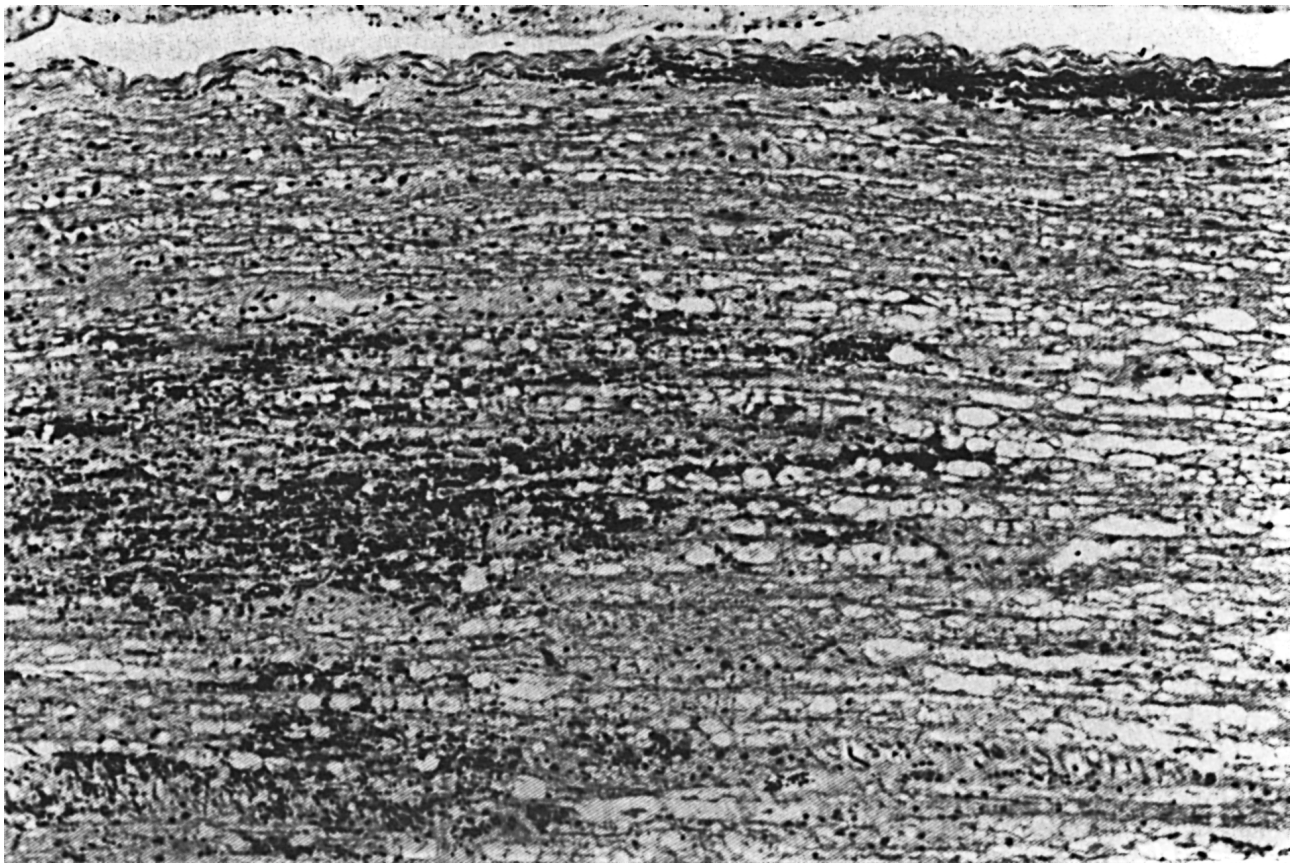


Figure 3. Long section of spinal cord showing a region with swollen axonal myelin, with erythrocytes infiltrating the white matter from a focus of haemorrhage (x250 H & E).

release a number of vasoactive substances that induce spasm in local arterioles[22] and contribute to post-injury hypoperfusion and ischaemia.

In skin flap models of I-R injury, HBO has repeatedly been shown to increase flap survival.[19,23] This may be explained by the finding that HBO can dramatically reduce leucocyte adherence at the site of injury.[22] A mechanism for this effect has been suggested by Thom[24] who showed that HBO reversibly inhibits leucocyte B2 integrin function, which is involved in the persistent adherence of leucocytes to vascular endothelium. HBO has also been shown to inhibit carbon monoxide-mediated lipid peroxidation of rat brain tissue.[25 26] If HBO produces similar beneficial effects in damaged spinal cord tissue, then any clinical efficacy in human dysbaric CNS injury could be explained.

Analogy of dysbaric injury with conventional spinal cord trauma

Where mechanisms like I-R injury and haemorrhage are involved in dysbaric spinal cord injury, comparison with the disease process of conventional spinal cord trauma is appropriate. At a cellular level, the final common path of injury is likely to be similar but, grossly, there are several obvious differences. Firstly, the diver's injury is not usually complicated by the need to stabilise vertebral fractures and manage hypovolaemic shock and other features of major trauma of which spinal cord injury is but one, albeit important, aspect. Secondly, actual traumatic section of all or part of the cord is not usually a feature in dysbaric injury. Thirdly, the disease process in DCI is typically one of multiple, small, predominantly white matter, lesions at different levels in the cord,[1] rather than the single large focus of injury common in spinal cord trauma cases. In divers, therefore, the injury is mainly axonal and the neurone cell body (which contains the biomolecular machinery for cellular repair) is largely spared. This contrasts with the injury in conventional spinal cord trauma, which often manifests as a central contusion with maximal damage to the grey matter in the centre of the cord and lesser injury to the surrounding white matter.[27] For the theoretical reasons above, the prospects for clinical recovery would seem better for divers. Anecdotally, this prediction appears to be borne out in practice,[28] although good, long term follow-up studies in divers are lacking.

Work studying conventional CNS trauma suggests remarkable functional redundancy, even in the spinal cord. Experiments in animals indicate that as few as four to six

percent of normally functioning axons in a motor tract can sustain normal motor function in the muscle they supply. Furthermore, these experiments suggest a threshold effect whereby an increase in axonal survival from less than three percent to more than six percent, in tracts passing through the site of injury, converts paralysed muscles to muscles with normal movement.[27] The implication for recovery of both dysbaric and conventional spinal cord injury is that even a small improvement in physiologic axonal survival may produce dramatic clinical benefit.

Studies of HBO treatment in conventional spinal cord trauma have been inconclusive although controlled animal studies have suggested benefit.[29] Interpretation of the human work is beset by problems of delay to treatment and the use of different partial pressures of oxygen.[30]

Future adjunctive drug therapies

Turning to the prospects for improved therapy of dysbaric CNS injury, it is likely that these will arise from improved treatments for conventional spinal cord injury. This subject has recently been concisely reviewed by Hall and Braughler.[21] Considerable progress has been made following the demonstration, in 1991, that high-dose methylprednisolone improved the outcome for human spinal trauma patients.[31] The mechanism of action of the methylprednisolone is now thought to be due, not to an effect on glucocorticoid receptors or oedema, but to a reduction in oxygen radical-mediated lipid peroxidation of injured cell membranes and inhibition of post- injury hypoperfusion. The importance of commencing treatment within eight hours of injury was highlighted by the methylprednisolone study[32] and the finding of this "therapeutic window" for drug treatment[33] may have implications for the timing of HBO treatment of dysbaric spinal injury.

A variety of other pharmacological strategies which involve anti-oxidant drugs and/or inhibitors of lipid peroxidation show great promise as future therapy of CNS injury.[20] Perhaps the most exciting potential therapeutic advance is the development of a non-glucocorticoid class of steroids, the 21-aminosteroids or "lazaroids", which are potent inhibitors of iron-catalysed lipid peroxidation.[21] This is particularly interesting because both the formation of oxygen radicals and the process of lipid peroxidation in the CNS may be catalysed by iron and haemoglobin from extravasated erythrocytes at a site of haemorrhage is an obvious source. The efficacy of one lazaroid, tirilizad mesylate, has been demonstrated in vivo in controlled, blinded studies in animals.[34] Currently, phase III human trials in head injury, subarachnoid haemorrhage and spinal cord trauma are in progress and the results are eagerly awaited. Their potential relevance to the management of dysbaric spinal cord injury is obvious.

Acknowledgments

The author is indebted to Mrs Catherine Jones, Mr Melvin Routh, and Petty Officers Sherry Burton, Cynthia Hall and Norman Wilt USN for the excellent technical assistance in the experimental work described. Thanks is also due to all veterinary and veterinary pathology staff at NMRI and to Mrs Susan Mannix for help in preparation of the manuscript.

The work was supported by US Naval Medical Research and Development Command Work Unit No M0099.01C-1053. The opinions and assertions contained herein are the private ones of the author and are not to be construed as official or reflecting the views of the United States Navy or Royal Navy.

The experiments reported herein were conducted according to the principles set forth in the *Guide for the Care and Use of Laboratory Animals,* Institute of Laboratory Animal Resources, National Research Council, DDH, Publ. No. (NIH) 85-23.

References

1 Francis TJF and Gorman DF. Pathogenesis of the decompression disorders. In *The physiology and medicine of diving, 4th Edition.* Bennett PB and Elliott DH. Eds. Philadelphia: WB Saunders, 1993; 454-480

2 Boycott AE, Damant GC and Haldane JS. Prevention of compressed air illness. *J Hyg Camb* 1908: 8: 342-443

3 Gersh I and Catchpole HR. Decompression sickness, physical factors and pathological consequences. In *Decompression sickness.* Fulton J F. Ed. Philadelphia and London: W B Saunders, 1951; 165 181

4 Moon RE and Gorman DF. Treatment of the decompression disorders. In *The physiology and medicine of diving, 4th Edition.* Bennett PB and Elliott DH. Eds. Philadelphia: WB Saunders, 1993; 506-541

5 Francis TJR and Smith DJ. Eds. *Describing Decompression Illness. Proceedings of the 42nd Undersea and Hyperbaric Medical Society Workshop.* Bethesda, Maryland: Undersea and Hyperbaric Medical Society, 1991

6 Broome JR, Dutka AJ and McNamee GA. Exercise conditioning reduces the risk of neurologic decompression illness in swine. *Undersea Hyperbaric Med* 1995; 22 (l): 73-85

7 Blick G. Notes on diver's paralysis. *Brit Med J* 1909; 2: 1796-1798

8 Brooks H. Caisson Disease. The pathological anatomy and pathogenesis with an experimental study. *Long Is Med J* 1907; 1: 149-158 and 196-208.

9 Smith AH. *The effects of high atmospheric pressure including the caisson disease.* New York: Eagle Print, 1873: 30, 49, 50.

10 Jaminet A. *The physical effects of compressed air and the cause of pathological symptoms produced on man by the increased atmospheric pressure employed for the sinking of piers in the construction of the Illinios & St. Louis Bridge over the Mississippi River at St. Louis, Missouri.* St. Louis: R and TA Ennis, 1871

11 Clark A. Effects of increased atmospheric pressure upon the human body: With a report of 35 cases brought to City Hospital from the caisson of the St. Louis and Illinios Bridge. *Med Arch St Louis* 1870; 5: 1-30

12 Broome JR, Dick EJ Jr, Axley MJ and Dutka AJ. Spinal cord hemorrhage in short-latency neurological decompression illness coincides with early recompression. *Undersea Hyperbaric Med* 1995; 22 (Suppl):

13 Hardman JM and Beckman EL. Pathogenesis of central nervous system decompression sickness. *Undersea Biomed Res* 1990; 17 (Suppl): 95-96

14 Palmer AC, Blakemore WF, Payne JE and Sillence A. Decompression sickness in the goat: nature of brain and spinal cord lesions at 48 hours. *Undersea Biomed Res* 1978; 5 (3): 275-286

15. Elliott DH and Moon RE. Manifestations of the decompression disorders. In *The physiology and medicine of diving, 4th Edition.* Bennett PB and Elliott DH. Eds. Philadelphia: WB Saunders, 1993; 487

16 Dutka AJ and Hallenbeck JM. Decompression Myelopathy. In *Handbook of Clinical Neurology, Vol 17 (61): Spinal Cord Trauma.* Frankel HL. Ed. Amsterdam: Elsevier Science Publishers bv, 1992: 215-229

17 Hallenbeck JM and Dutka AJ. Background review and current concepts of reperfusion injury. *Arch Neurol* 1 990; 47: 1245-1254

18 Russell RC, Roth AC, Kukan JO and Zook EG. Reperfusion injury and oxygen free radicals: A review. *J Reconstr Micro* 1989: 5: 79

19 Zamboni WA. The microcirculation and ischemia-reperfusion: Basic mechanisms of hyperbaric oxygen. In *Hyperbaric medicine practice.* Kindwall EP. Ed. Flagstaff, Arizona: Best Publishing, 1994: 552-64.

20 Granger DN, Benoit JN, Suzuki M and Grisham MB. Leukocyte adherence to venular endothelium during ischemia/reperfusion. *J Reconstr Micro* 1989: 257: G683

21 Hall ED and Braugler JM. Free Radicals in CNS Injury. *Res PubI Assoc Res Nerv Ment Dis* 1993; 71: 81-105

22 Zamboni WA, Roth AC, Russet RC, Graham B, Suchy H and Kukan JO. Morphological analysis of the microcirculation during reperfusion of ischemic skeletal muscle and the effect of hyperbaric oxygen. *Plast Reconstr Surg* 1993; 91: l110-1123

23 Nemiroff PM. Hyperbaric oxygen in skin grafts and flaps. In *Hyperbaric medicine practice.* Kindwall EP. Ed. Flagstaff, Arizona: Best Publishing, 1994: 566-579

24 Thom SR. Functional inhibition of leukocyte B2 integrins by hyperbaric oxygen in carbon monoxide-mediated brain injury in rats. *Toxicol Appl Pharmacol* 1993; 123: 248-256

25 Thom SR and Elbukin ME. Oxygen dependent antagonism of lipid peroxidation. *Free Radical Biol and Med* 1991; 10: 413

26 Thom SR. Xanthine dehydrogenase conversion of oxidase and lipid peroxidation in brain after CO poisoning. *J Appl Physiol* 1992; 73: 1584

27 Young W. Recovery mechanisms in spinal cord injury: Implications for regenerative therapy. In *Neural regeneration and transplantation. Frontiers of clinical neuroscience Vol 6.* Seil F. Ed. New York: Alan R Liss, 1989: 157-169

28 Linaweaver PG and Greer HD III. Paralysis in divers: The natural history of decompression sickness. In *Rehabilitation of the paralysed diver. The Thirteenth Undersea Medical Society Workshop.* Miller JN and Parmentier JL. Eds. Bethesda, Maryland: Undersea Medical Society, 1984: 7-23.

29 Narayana PA, Kudrle WA, Liu SJ, Charnov JH, Butler BD and Harris JH. Magnetic resonance imaging of hyperbaric oxygen treated rats with spinal cord injury: preliminary studies. *Magn Reson Imaging* 1991; 9(3): 423-428

30 Yeo JD. The use of hyperbaric oxygen to modify the effects of recent contusion injury to the spinal cord. *Cent Nerv Syst Trauma* 1984; 1(2): 161-165

31 Bracken MB, Shepherd MJ, Collins WF et al. A randomized controlled trial of methylprednisolone or naloxone in the treatment acute spinal cord injury. *N EngI J Med* 1990; 330 (20): 1405-1411

32 Bracken MB and Holford TR. Effects of timing of methylprednisolone or naloxone administration on recovery of segmental and long tract neurological function in NASCIS 2. *J Neurosurg* 1993; 79 (40): 500-507

33 Young W. The therapeutic window for methylprednisolone treatment of acute spinal cord injury: Implications for cell injury mechanisms. In *Molecular and cellular approaches to the treatment of neurological disease.* Waxman G, Ed. New York: Raven Press, 1993; 191-206.

34 Anderson DK, Braughler JM, Hall ED, Waters TR, McCall JM and Means ED. Effects of treatment with U-74006F on neurological recovery following

experimental spinal cord injury. *J Neurosurg* 1988; 69: 562-567

Key Words
Decompression illness, hyperbaric oxygen, physiology, reprint, treatment.

Surgeon Commander Broome is currently (Summer 1995) appointed to the Naval Medical Research Institute, Bethesda, Maryland, USA.

Address for correspondence Dr J R Broome, 3 Ellachie Gardens, Alverstoke, Gosport, Hampshire PO12 2DS, UK.

Reprinted, by kind permission of the Editor, from the Journal of the Royal Naval Medical Service 1995; 81:120-126.

ASMA-UHMS DCS WORKSHOP THOROUGH AND WELL ATTENDED

R W (Bill) Hamilton

As an example of inter-society collaboration on a topic of mutual interest, treatment of decompression illnesses, and in terms of gathering probably the best collection of experts on this subject that have ever been assembled, the Workshop was an outstanding success. In terms of achieving consensus of the experts on some key issues, success was somewhat more elusive. Since this workshop's configuration and auspices were laid out in the lead article *Pressure* [24(3), May 1995] we will not repeat them here. Its official goals, what was wanted in the way of consensus, were perhaps not so well laid out (as opposed to objectives, the mechanism for reaching those goals). Organizers and chairmen were Paul Sheffield and Richard Moon.

Basically it was hoped that uniform treatment procedures could be agreed upon that would permit defining the requirements of treatment, hence its cost. This is important to DAN, who has to pay for treatments. It is reasonable to try to set limits to how many post-treatment recompressions might be needed, for example, but by all appearances this group was not ready to sign on for this.

Lest this imply that the meeting was anything but outstanding, please note that these participants had 2 days (including an evening session) of concentrated and well-prepared presentations by the world leaders in decompression and decompression illnesses. This included everything from reviews of NASA's and DAN's experience and experience of various navies, all the way to the devastating decompression sickness not being treated at all well on the Miskuito coast of Central America. The program included most of the heavy-weights, but sitting there on the front rows were many others not on the program but with plenty of experience to share. Being with this group for a couple of days was a real treat professionally.

To begin with we got a neurological and neuropathological orientation that started us off well. Drew Dutka's chart of DCI made a lot of sense out of this slightly troublesome terminology. John Hardman's pathology states that irreversible damage begins within 10 minutes. Next the "aerospace" portion that included altitude DCS and the effects of oxygen pre-breathing as well as some provocative possibilities (by Mike Powell) to explain why astronauts are so "resistant" to DCS. Pat Kimbrell presented a crisp and well thought out table for treatment of altitude DCS. Differences showed up on treatment with Table 5 after altitude DCS; USAF uses it with few recurrences, Canadian experience says it causes too many. In fact, it seems Table 5 split the group down the middle; it will be interesting to see how the balloting went on this one. Ed Thalmann reviewed the history of treatment table development, noting that the need for decompression following treatment was not immediately recognized. Table 5 works if the rules are followed. He sees no specific benefit from helium except in the ensuing decompression. Embolism as an entity was covered, a refreshing view since the advent of "DCI" has tended to subvert the use of that term, unfortunately. The advice from Des Gorman is to treat the mechanical effects (the bubble), then the damaging effects of bubbles. A lot of embolism cases resolve spontaneously, some never do.

If this Workshop made one decision, it was that early treatment is beneficial. This was a prevailing theme throughout, and no one had any arguments against it, even if the only chamber available is a monoplace chamber, a distinctly new consensus over some older viewpoints. Yehuda Melamed reviewed the successful Israeli experience with transportable chambers in the early 1970s. Gary Beyerstein mentioned that the more enlightened commercial companies no longer have penalties for being treated, a tactic that gets divers who need it in the chamber quicker; others can learn from this success. David Elliott acknowledged that commercial diving is boring these days, since there are so few hits. Once again, the concept of recompressing first and asking questions later is endorsed.

Another feeling that seemed present was that the treatment levels that have been used are at a marginally toxic level of oxygen. The treatment with 100% oxygen at 2.8 atm is a compromise to get the maximum compression at an oxygen level that can be tolerated; it works, but the O_2 level is higher than desirable and 2.5 atm may be better.

One opinion on the oxygen toxicity front is that one might have to defend not using methyl prednisolone; a free radical scavenger. The viewpoint on use of helium was not crisp in either direction; Des Gorman is seeing the need for fewer follow-up treatments. It is hard to do a blinded study since the voice gives it away. (If it's my spine I'll take it. Ed. *Pressure*) Likewise the room seemed a bit divided on the value of the "Hawaiian spike", a fast and deep recompression to start a treatment as described by Bob Overlock. Some see merit in it, but by no means all; the case against any deep treatment, say 6 atm, did not come out strongly, however. Still another uncertainty is the benefit of lidocaine, promising to some but neurologist David Warner and others are skeptical, but say to keep studies going. Philip James hypothesized that one of the reasons fluids seem to be beneficial is that they are unsaturated and they allow a redistribution of gas. Saturation "treatments" with a nitrogen-based atmosphere are not gaining ground, in part because the nature of the disruption to an active clinical HBO facility; here the tendency is to go with something like Comex 30. Counterdiffusion is not a problem with a switch to heliox if compression is done simultaneously. Dave Youngblood, who has as much experience as anyone in the room, strongly advises against "unwarranted" saturation.

Considering that several controversial situations have developed over the use of high-tech imaging techniques, mainly due to big decisions based on preliminary data, this session moved comfortably through this. The benefit of psychological testing is not universally accepted, but techniques may be getting better developed. Sometimes imaging is used to track "tailing" techniques of giving daily treatments for days to weeks and basing the benefit on scans as well as symptoms. The suggested notion that this might work on "punch-drunk" divers is intriguing. Keith Van Meter's chamber does an average of 13 tailing treatments, but they stop immediately when it is no longer effective; Paul Harch admitted that "tincture of time" might play a role. This caused less obvious controversy than one might have expected. Dick Vann's comprehensive review of DAN data concluded that additional treatments help, but none had complete relief after 15 treatments.

Like the monoplace, in-water oxygen treatment drew less flak than it would have a couple of years ago, possibly because by now this group is firmly indoctrinated on the benefits of prompt treatment. Carl Edmonds laid out his current procedures, which are similar to those in the first two editions of his book but now require a 2-hour linear ascent from the 9-msw treatment depth. This is likely to make this approach unacceptable where the sea is rough. Among the contraindications is reluctance on the part of the diver (or team), a good point since thermal protection has to be better than that for most dives. Surface oxygen is acknowledged to be beneficial, but there were warnings that it can be abused and used to avoid proper treatment. It can be helpful after a treatment.

In an interesting coincidence, the Great Lakes UHMS chapter has planned a mini-symposium on "Different treatments for different people". This was not planned as a follow-on to this workshop, but it asks a quite cogent set of questions. And speaking of chapters, kudos to the Gulf Coast chapter for a major contribution to this Workshop, there are few better ways to put your chapter's earnings to good use.

The facilities at the Colony's conference centre were excellent, with good projection and light control (never too dark), handy coffee, and bearable chairs. This Workshop was nicely done and undoubtedly very rewarding for all those who attended. Kudos to Jane Dunne particularly for doing the intensive organising that made it happen.

Key Words

Decompression illness,hyperbaric oxygen, treatment, reprint.

Reprinted by kind permission of the Editor from PRESSURE, the Newsletter of the Undersea and Hyperbaric Medical Society (UHMS), 1995; 24 (5): 4-5.

The address of UHMS is Undersea and Hyperbaric Medical Society, 10531 Metropolitan Avenue, Kensington, Maryland 20895, USA.

The full proceedings of this meeting will be available for sale at the 1996 UHMS meeting in Anchorage, Alaska.

BS-AC GIVES THE OK TO NITROX

Chris Allen

At DOC 94, BS-AC NDO Tony Hoile announced that a Working Group had been set up to review the BSAC's position on nitrox. Here, Chris Allen, Chairman of the Working Group, describes how the review was conducted and explains the BSAC's decision to sanction the use of nitrox in Club activities and to launch its own nitrox training program.

The possible use of nitrox (oxygen-enriched air) for sport diving was first examined by a BS-AC working group set up at the end of 1991. Having examined the advantages and disadvantages in some detail, the group concluded that the use of nitrox mixes was a legitimate technique which could be carried out safely, given proper training and sensible precautions in gas mixing and testing. However,

while the use of nitrox offered some advantages in the middle depth range, it was concluded that for most sport divers on most dives the disadvantages of nitrox use outweighed the potential benefits.

The findings of the working group were published in Diver and the NDC Bulletin and were presented at the 1992 Diving Officers' Conference. Having considered the report, the BS-AC National Diving Committee decided that the use of nitrox would not be permitted during BS-AC activities. Since publication of the 1992 report, the National Diving Committee has continued to monitor progress in this and other related areas. At the end of 1994 it was decided to establish a second working group to review the BSAC's position in the light of new developments. Other members of the working group were Lizzie Bird (Southern Region Coach), Dave Crockford (NDC), Alison Farrow (Coach for North Scotland) and Dave Mitchell (First Class Diver Chief Examiner). The working group also received input from other members of the National Diving Committee and other specialists in the fields of diving medicine, decompression research and diving computer design.

As the subject of nitrox use by sports divers is still somewhat controversial and can give rise to heated debate, the working group's approach was to be as objective as possible. In particular, a conscious effort was made to obtain as much factual or statistical information as possible.

A number of initiatives were put in place to examine the five main areas of interest which were felt to be central. Firstly, to try to determine the actual size of the potential benefits from nitrox use, in terms of safety or reduced decompression requirements. Secondly, to carry out a review of accident and incident performance during the period since the first report. Thirdly, to establish as accurately as possible the number of divers using or wanting to use nitrox. Fourthly, to obtain feedback from BS-AC members on the subject of nitrox. Finally, to review recent developments in equipment and to examine the size and quality of the existing specialist nitrox training organisations.

In order to quantify the safety benefit, Dr Bill Hamilton, a decompression expert from the USA, agreed to carry out a small project to perform a Maximum Likelihood Analysis and compare the predicted DCI risk levels on air against other nitrox mixes, for a set of typical dive profiles performed using air decompression tables.

At the same time, the BSAC's own incident database was analysed to establish both the number and type of incidents which have occurred using nitrox and also whether decompression incidents were occurring on air in the depth range where nitrox can be seen to provide a benefit.

Surveys of all delegates and all diving officers present at DOC '94 were carried out to establish the degree of existing usage of nitrox and to obtain DOs' views on the subject. This was backed up with similar questionnaires distributed through the Coaching Scheme to check the validity of the DOC results. The nitrox training organisations were contacted for details of the number of certifications they had performed, and a survey of dive shops and other facilities offering nitrox training and gas sales was conducted to determine the level of current infrastructure available to support nitrox use.

In summary, the results of all these investigations showed that, while the laws of physics and physiology had obviously not changed since the time of the first report, there had been significant advances in equipment, particularly with the appearance within the last few months of nitrox dive computers and better oxygen analysers.

The appearance of nitrox dive computers is particularly important for two reasons. Firstly, they allow useful extensions of bottom time to actually be realised. Secondly, where they include an oxygen toxicity alarm, they are able to offer at least a partial solution to the increased risk of oxygen (0_2) toxicity which arises when using oxygen-enriched mixtures. Up to now the theoretical additional bottom time available when using a nitrox mixture, other than air, has not necessarily been achieved in practice.

Divers using an air dive computer have been able to match, or even exceed, the bottom time available from the common nitrox mixtures for which only tables assuming a square profile dive were available. In other words, in terms of extended bottom time, the benefit of a multi-level calculation performed by the computer was equal to, or even greater than, the benefit of using a nitrox mixture rather than air. However, a nitrox computer which can perform the same multi-level calculation, but base it on the nitrox mix being used, makes the additional bottom time a reality.

On the safety side, too, nitrox computers can offer a big advantage. One of the greatest concerns with nitrox use is the risk of oxygen toxicity through straying too deep and exceeding the partial-pressure threshold. This risk, of course, is also present with air but because the depth at which it occurs (66m+) is well outside the normal diving range of most sports divers, in practice it does not present a problem. However, with an enriched-oxygen mixture the threshold of O_2 toxicity can occur at a depth of 30 metres or less, right in the middle of the sports diving range. Computers which offer an oxygen-toxicity alarm provide at least a partial solution to this problem. Some computers, such as the Aladin Nitrox, which also monitor gas consumption are even able to adjust the oxygen clock for the diver's work rate, which can be an important factor in oxygen toxicity.

Having reviewed and analysed all the information collected, the working group has recommended that the BS-AC should permit the use of nitrox during BS-AC activities and that BS-AC courses should be developed and offered through the Coaching Scheme and BS-AC Schools. As a result, it has been decided that suitably qualified members will be permitted to use nitrox during BS-AC activities with effect from 1 May 1995. BS-AC Courses will be available from the end of 1995.

In order to be considered qualified to use nitrox from May onwards, members must have a recognised qualification from one of the nitrox training organisations and fulfil the BSAC's own minimum qualification requirements.

In order to use enriched air nitrox (EANx) 32 or EANx 36, Club members should be qualified to a minimum level of Sports Diver, with a further 20 open water dives logged since qualification, and hold either the IANTD Basic EANx Diver, TDI Nitrox Diver, or ANDI Complete Safe Air User qualifications.

In order to use nitrox mixes other than 32% or 36%, and specifically to use EANx 40 or EANx 50 as a decompression gas, members must be qualified to at least BS-AC Advanced Diver standard and EANx Diver, TDI Extended Range Diver, or ANDI Technical Safe Air Diver qualifications. Further guidance on entry requirements for BS-AC courses and equivalences between BS-AC and other qualifications will be published in due course.

The practical aspects of dive planning and organisation particularly the marshalling of mixed groups of air and nitrox divers, are of course very important. Once again, detailed guidance will be provided via the NDC Bulletin, but in summary the working group's recommendations are as follows.

As far as the use of decompression tables and computers for nitrox is concerned, the advice varies according to the reason for choosing to use nitrox. If the aim is to achieve a safety benefit, then it is recommended that nitrox mixtures are used (within their depth limit), with either the BS-AC '88 Tables or an air dive computer. If the aim is to obtain an extended bottom time or a reduced decompression requirement, then it is recommended that a nitrox dive computer is used.

For mixtures up to 40% O_2 content, normal diving equipment may be used without special precautions unless this would be contrary to the manufacturer's advice. If, during the gas mixing process, items of equipment such as cylinders are exposed to oxygen concentrations greater than 40 per cent, then extra precautions are required.

Where mixtures with an O_2 concentration greater than 40% are being used or encountered during mixing, special care is required to ensure that all equipment is suitable for oxygen service. This means that all components must be compatible with oxygen service and be oxygen clean before use.

Nitrox cylinders should be dedicated to that use, colour coded yellow with a 10 cm green band, marked clearly NITROX. They should also be prominently labelled with the mixture details.

Every diver must be responsible for the gas in their cylinder and have full knowledge of the contents. It is therefore strongly recommended that the diver should witness testing of their gas at the mixing station and that he or she should personally verify the gas analysis by re-testing immediately before use.

In the event of a diving accident or case of decompression illness in someone who has been diving with nitrox, the treatment of the casualty should be exactly the same as for an air diver. There is no reason not to administer O_2 to the casualty or to carry out a normal recompression treatment, though the details of the dive and the gas mixture being used should, as always, be given to the treatment centre.

Dive Marshals responsible for organising and marshalling a group of divers using nitrox mixtures should have a clear understanding of the implications of nitrox use. Ideally they should themselves be qualified in nitrox diving to the level appropriate to the dive being performed. As a minimum, the marshal must be aware of the percentage of oxygen in the nitrox mix and the maximum depth to which can be safely used.

The BS-AC is the first major training organisation to develop its own nitrox courses. This, we believe, underlines both the quality of BS-AC training and the fact we are the most "technical" of the major training agencies in the world. However, it also imposes upon us a responsibility to ensure that we do it safely and professionally.

The safe use of nitrox does require increased knowledge and diving discipline. If you want to be involved, you will need to invest a certain amount of time and money to obtain the proper training and equipment. Nitrox diving will not be suitable for everyone. However for those who want it, the BS-AC offers the chance to acquire the necessary knowledge and skills within the Club system.

Key Words

Equipment, nitrox, physiology, safety, training.

Reprinted, by kind permission of the Editor, from DIVER, the magazine of the British Sub-Aqua Club, 1995; 40 (5) May: 35-36.

TECHNICAL DIVING: A BEGINNER'S GUIDE

Mike Busuttili

Technical diving has arrived and is fast becoming established in the diving world. But what exactly is technical diving, and what's in it for you? Mike Busuttili explains it all here.

Sport diving, or recreational diving, can be defined as diving that is enjoyable, attainable, perhaps exciting and demanding and certainly safe.

It has been the aim of training organisations such as the BS-AC to bring people to a level of competence that should not expose them to unnecessary risk.

It is when the type of diving being undertaken begins to present new problems that new solutions and new techniques have to be evolved and in recent times diving exploration has been taking us beyond the norm, requiring a broader range of techniques and equipment in order to maintain the required level of safety.

The depth limit for air diving recommended by the BS-AC is 50 m (less by some other training organisations) and this is deeper than the vast majority of divers may ever feel able to go. But there are some who have reached this limit, and still find the need to dive deeper in safety, and it is these who have caused the so-called technical diving wave.

For many, the wish arose in the pursuit of underwater projects that took them outside the envelope of normal sport diving and into a new area of risk that needed a new set of solutions. This is an area also called extended range diving since it recognises the wish to go further, for longer, sometimes in more extreme conditions, the necessary precautions having to be taken.

It is this professional/amateur attitude, professional in approach, amateur in motivation, that is at the heart of the technical diving movement.

So what does it all involve?

Well, technical diving uses a range of special techniques and equipment to extend the safe operational range of the diver. A principle feature is the use of gas mixtures other than compressed air to reduce the problems associated with nitrogen (narcosis and decompression sickness).

For many divers the passage into technical country starts with nitrox (although nitrox will probably come to be considered a standard diving gas within a few years).

Nitrox is a mixture of nitrogen and oxygen that contains more oxygen than ordinary compressed air does. It is available in a variety of mixes and each is named after the percentage of oxygen it contains e.g. Enriched Air Nitrox 36 (EAN36), containing 36% oxygen, compared with 21% in compressed air.

The advantage of using nitrox is longer no-stop times, or shorter decompression stops or improved safety. By reducing the proportion of nitrogen in our breathing mixture, we absorb less nitrogen during the dive and thus have less nitrogen to eliminate during the ascent.

For a given mixture we can calculate, or read from a table, the Equivalent Air Depth (EAD). This is the depth at which we would have the same partial pressure if nitrogen (PPN_2) as if we were diving on air.

For example, our no-stop limit at 30 m using air on the BS-AC's '88 Table A is 20 minutes, but if we use EAN36, we will have an EAD of 24 m, so we can extend our time to 30 minutes.

As another example, if we want to spend 40 minutes at 30 m on air, we would need to make decompression stops of 1 minute at 9 m, and 9 minutes at 6 m. But by using EAN36 we would need only 1 minute at 6 m. (NB: this will only work for the first dive using current BSAC '88 Tables, as the surface interval is carried out in air, a different gas to that used during the dive).

A further possibility is to carry out a dive on compressed air, say to 40-45 m, but to use a nitrox mix (probably between 50% and 80% oxygen, or even pure oxygen) for decompression stops. This will give faster elimination of nitrogen and therefore shorter decompression times.

Yet another approach is to improve your safety margin by diving on nitrox but treating it as compressed air for decompression purposes. In other words, you breathe nitrox during the dive or the decompression stops, or both, but use your usual decompression table or air dive computer to control your decompression procedure.

The latest generation of nitrox computers enables divers to gain the benefits of reduced nitrogen intake on dives where a multi-level profile is involved. The oxygen percentage of your gas mix is simply entered into the computer, which then deduces the balance of nitrogen and calculates the decompression requirement accordingly.

It has been suggested that nitrox reduces the narcosis effect, but there is no scientific evidence to support this. Another observation is that nitrox results in a lower gas consumption rate, but this has not yet been demonstrated.

There are some problems with using nitrox. Although reducing the nitrogen content brings some advantages, increasing the oxygen content introduces some new disadvantages. We have all learned that oxygen becomes toxic when breathed at high partial pressures, and that, at 66 m, normal compressed air has reached the maximum safety limit for the partial pressure of oxygen (PPO_2) of 1.6 bar.

If we take it that the limit for sport divers is the 1.45 bar recommended by the BS-AC, then EAN32 has a maximum depth safety limit of 35 m and EAN36 is limited to 30 m, with air at 59 m.

Clearly, then, nitrox is not a gas for deep diving, and the limits should never be exceeded.

An additional oxygen problem is that it can have a toxic effect upon our lungs if we are exposed to high concentrations over a longer period. Thus, if we are diving to 30 m on EAN36 (PPO_2 = 1.44) then our maximum exposure should not exceed 120 minutes at that depth, which can be built up over a series of dives. In fact, over the course of 24 hours time spent at this PPO_2 should not exceed 180 minutes.

Nitrox computers are able to track your oxygen exposure to ensure that you do not exceed the recommended oxygen toxicity limits.

Oxygen in high concentrations also presents problems in its handling and use. Although not in itself flammable, it supports combustion and when it comes into contact with certain substances, such as hydrocarbons at high pressure, there is a risk of explosion.

Any part of our diving equipment that stands a chance of coming into direct contact with high concentrations of oxygen must therefore be scrupulously clean and free from any such contamination.

Firstly, the diving cylinder, along with its valve, must be cleaned for oxygen use, known as “in oxygen service”, because the procedure for filling it with nitrox could start by introducing a quantity of pure oxygen into the cylinder. The standard air regulator is normally suitable for use with nitrox mixes containing up to 40% oxygen, but for higher concentrations it, too, must be cleaned and never then used with normal air.

That is nitrox diving, with its various limitations.

In order to achieve greater depths than normal, we need to reduce the oxygen content in the mix to avoid oxygen toxicity and reduce the nitrogen content to avoid narcosis problems. This is where we enter the true realm of technical diving.

To reduce the amount of nitrogen it is obviously necessary to add another inert gas to make up the difference.

Helium is the gas usually used and such a mixture is called Trimix.

Trimix diving allows divers to achieve great depths, well beyond 65 m, but needs exceptionally careful planning (usually involving using desktop computers) and complex stage decompression.

This means that divers must carry various different supplies of gas, each in its own cylinder, and also be sure which mouthpiece is which, switching at the required time and depth.

The well organised “tekkie” will carry a travel mix suitable for breathing from the surface down to a certain depth, will then switch to a bottom mix (lower in oxygen content), and during the return to the surface will switch back to the travel mix, with the additional possibility of a decompression mix which will have a high oxygen content to give optimal nitrogen elimination.

Trimix divers are seen entering the water with four or more cylinders, and this is true technical diving, suitable for only the minority.

For even deeper diving still, it is possible further to reduce the oxygen content in the mix and to replace all of the nitrogen with helium. Called Heliox, the use of this is beyond the scope of almost all divers.

Clearly, techniques have had to be developed to ensure safe diving in extreme underwater situations, such as the penetration of wrecks like the *Lusitania*, deep underground cave systems, and diving under ice, all grouped under the description overhead environments.

Diving in a situation where you cannot make a direct ascent to the surface presents a host of additional problems and puts great emphasis on managing your gas supply. There is, too, the question of navigating your way in and out!

Other techniques have been evolved to allow extended in-water decompression procedures to be carried out in greater safety and with greater efficiency.

Helium, incidentally, has two major disadvantages: It is horribly expensive and it cools the diver down faster than when breathing air.

What about rebreathers?

The problem of carrying large amounts of gas on a dive can be a real one. An obvious solution is to use the

gas more efficiently, rebreathing it so that the oxygen content is used more fully. A rebreather using a nitrox mix containing, say, 32 or 36 percent oxygen will typically be equipped with a much smaller reservoir cylinder than an open-circuit aqualung set.

Among other benefits, such as compactness and low bubble production (in the case of semi-closed-circuit versions) or no bubble production (in the case of fully-closed units), rebreathers achieve major reductions in gas consumption. Against this must be weighed their currently high initial cost, and their need for considerably more care and attention than open-circuit equipment.

One significant handicap to progress is the fact that dive centres equipped to fill your dedicated nitrox cylinder with the appropriate gas mixture are still few and far between. This is only likely to change as a result of increased demand, because a fully-equipped mixed gas blending system requires a major investment on the part of the filling station.

What training is available for nitrox and technical diving?

If you are a BS-AC member, you will soon be offered skill development courses leading to BS-AC Nitrox Diver and BS-AC Advanced Nitrox Diver qualifications. An extended range diver course is also in hand.

An alternative is to go to one of the specialist agencies: IANTD (International Association of Nitrox and Technical Divers): TDI: (Technical Diving International); or ANDI (American Nitrox Divers International). They all offer a path to nitrox and trimix diving, eventually with rebreathers.

A good starting point for those interested is *An Introduction to Technical Diving*, by Rob Palmer, available from dive shops and the *DIVER* Bookshop at £17.95.

CMAS (the World Underwater Federation) should soon have international equivalents available which will allow member organisations to apply for equivalents to their certificates for issuing to their members.

Glossary

ANDI	American Nitrox Divers International.
EAD	Equivalent Air Depth.
EAN	Enriched Air Nitrox.
Heliox	A breathing gas mixture containing oxygen and helium.
IANTD	International Association of Nitrox and Technical Divers.
Nitrox	Any gas mixture (including air) containing nitrogen and oxygen, but commonly used to describe one where the oxygen content exceeds that of normal air. Known as Oxygen Enriched Air, Safe Air (registered to ANDI), and EAN.
PPO_2	Partial pressure of oxygen in the mixture breathed. The BS-AC recommended limit for this is 1.45 bar for in-water use.
PPN_2	Partial pressure of nitrogen in mixture breathed.
TDI	Technical Diving International.
Trimix	A breathing gas mixture containing oxygen, nitrogen and helium.

Key Words
Equipment, mixed gas, nitrox, physiology, reprint, safety, technical diving.

Reprinted, with some editing, by kind permission of the Editor, from DIVER, the magazine of the British Sub-Aqua Club, 1995; 40 (10) October: 41-43

KILLER FACTORS IN TECHNICAL DIVING

Killer factors in technical diving are complacency, attitude, oxygen toxicity, exceeding personal limits, ignorance and complexity.

Key Words
Reprint, safety, techical diving.

Reprinted, by kind permission of the Editor, from DIVER, the magazine of the British Sub-Aqua Club, 1996; 41 (1) January: 58

COULD DO BETTER! BRITISH SUB-AQUA CLUB INCIDENTS IN 1995

Tim Parish

Nineteen-ninety-five was a good year for diving. Great weather, a 2,000 increase in BSAC membership and, probably as a result of both these factors, a rise in the number of "man-dives" carried out, estimated at over 3,000,000, half a million more than in 1994.

Even better was the drop in the number of incidents, from 389 in 1994, to 351 in 1995, despite the large increase in the number of dives carried out. This reduction has also

TABLE 1

BS-AC INCIDENTS IN 1995

Overseas	33
Miscellaneous	2
Equipment	22
Technique	14
Ascent	10
Boat or surface	113
Injury	28
Decompression illness	111
Fatalities	18

(Constructed from a bar graph with numbers)

TABLE 2

DEATHS AND BS-AC MEMBERSHIP

Year	Deaths		BS-AC
	BS-AC	Other	Membership
1986	5	8	34,000
1987	5	5	34,000
1988	10	7	33,000
1989	4	8	34,000
1990	3	7	36,000
1991	9	9	43,000
1992	9	8	45,626
1993	3	7	50,000
1994	6	8	50,000
1995	9	9	52,364

(Compiled from a small scale bar graph and the text of the article)

been reflected in the data available from The Coastguard Agency, Diving Diseases Research Centre (DDRC) and the Royal National Lifeboat Institution (RNLI). Furthermore, the increased detail available from the Coastguard (our largest source of information) has enabled us to analyse more of the incidents more fully, leading to some surprising changes to some of our previous conclusions.

The breakdown of the data does not, however, bring any surprises. The number of fatalities in Britain rose to 18 last year, in 15 separate incidents. Nine involved BSAC members (despite the BSAC having an estimated two-thirds of the diving population as members). This is a 50 percent increase over 1994 and double the 1993 total, but before we get too despondent it is worth bearing in mind that statistically there is still only a 0.005 percent chance (or 1 in 200,000) of becoming involved in a fatal incident!

To put this further into perspective our records reveal that, while 1995 was not a particularly good year compared to 1994 and 1993, the picture is not as bad as it has been painted in the press. With a continuously expanding BSAC membership (52,364 on Nov 1 1995) and with half a million more "man dives", fatalities were no worse than in 1992. In that year there were 17 fatalities, 9 of which were BSAC members, with BSAC membership at 45,626.

1995 was also far better, statistically speaking, than 1988 (16 fatalities, 10 BSAC, with only 33,000 members). When you compare the ratios of number of members to fatalities, there was a 50 percent decrease in the relative number of fatalities in 1995 over 1988. While that should make us slightly more positive about the number of fatalities, this is obviously still too high.

Decompression incidents showed a welcome drop, with 111 incidents in 1995, compared to the 149 in 1994. Unfortunately, the British Hyperbaric Association's recompression data was not made available to us last year. Had their figures been available, they would have boosted our total decompressions to around 130, but still a welcome reduction. It is also important to realise that the BHA information is sparse and its loss did not invalidate any of our analyses.

Injury and illness also showed a reduction, only 28 incidents compared to 47 in 1994, but boating and surface incidents apparently increased, though only by one! In reality, I believe that this category has probably actually reduced, for 1994 was the first year of the revised Coastguard reporting procedures and they could supply only 9 months of data. Last year we received the full 12 months' information and could reasonably expect that difference to affect the figures.

Nevertheless, given that this was an area we were trying to target, the figures are relatively disappointing.

TABLE 3

FACTORS PRESENT IN 15 DIVING DEATH INCIDENTS

Separation	7
Buoyancy	4
Deep Dives	4
Narcosis	4
Panic	3
Free flow	2
Incorrect controlled buoyant lift	2
Out of air	1
Solo dive	1

(Table constructed from a pie chart)

The number of divers missing on the surface has remained at 51 and, with engine failures at 46, these two categories make up the greatest proportion of the 113 incidents recorded.

Ascents accounted for 10 incidents, technique for 14, equipment problems for 22 and 2 were categorised as miscellaneous.

Last year, for the first time, we categorised the 33 overseas incidents separately and did not include them in our statistics. This was because the overseas figures we produce each year have always before been quoted as UK figures and this has been misleading. Overseas incidents are still included in the Incident Report which has been sent to every Branch Membership Secretary.

The main contributory factor in last year's fatal incidents was separation under water. Seven incidents stated this as a major cause and, when you consider that we have 3 incidents where the detail of the dive is not known, separation was involved in over half the UK fatalities. This is an area that we should be able to control and must improve.

Together with depth, and very much interrelated, buoyancy and narcosis were the second most common factors, each showing themselves as causes in 4 incidents (not all the same ones). Depth continued to be a major contributor in all incidents. Last year we saw 18 incidents occurring at depths below 50 metres, compared to 9 in 1994. I do not believe that it has become any more dangerous to dive to such depths than it was in 1994, therefore the conclusion we must draw is that *more* deep dives are being carried out.

One of the factors that must be borne in mind is that all the hype surrounding "technical diving" has made such depths appear to be far more normal than they used to be. That has, in part, led to people attempting dives that are far beyond their training, experience and capability. As a typical example, 2 divers died in 1994 attempting to break their own depth records, to get below 75 m on air! Their bodies were recovered by an ROV some days later, from nearly 90 m.

Panic, both underwater and on the surface, was the major cause in 3 incidents and free-flows and incorrectly handled controlled buoyant lifts contributed to 2 each.

The incorrectly controlled buoyant lifts were particularly disappointing. Both started with the casualty still alive, but the rescuers tried to carry out the lift using their own buoyancy instead of that of the casualties. On both occasions contact was lost during the ascent, with the result that the rescuer arrived on the surface out of control and the casualty sank to the seabed! The moral is clear: it must be the casualty who is made buoyant, not the rescuer, and if that means releasing the weightbelt then so be it. Get casualties to the surface by any means. They can be treated up there; they can't be helped on the bottom.

Moving to decompression, there is a better picture, with a drop in people treated, down to 111 from last year's 149. There have been significant areas of change. This has mostly been caused by the analysis of the more detailed Coastguard data, and it marks a significant improvement in our knowledge of what happened prior to the incidents. For instance, repeat diving leapt up last year, occurring in nearly 20 percent of incidents. The incidence of divers missing deco stops also increased. The most important and interesting changes, however, were the number of incidents within the tables or computer algorithms, and the number of rapid ascents.

For many years, the number of decompression incidents occurring within the tables has hovered around the 37-40 percent mark, calculated from the information available to us. Last year, that figure was only 20 percent, but the figures also show that rapid ascents apparently increased from 25 percent in 1994 to nearly 40 percent in 1995. An almost direct reversal in perentage occurrence.

If these figures are a truer reflection of what is happening, it gives us a far better chance to reduce the incident rate by ensuring that ascent rates can be controlled properly. This is a breakthrough. We could now be looking at a skill that we can train people properly to carry out and to practice. In contrast, DCI within the limits is relatively uncontrollable. I will be keeping an eye on this area to see if this is a real change, not just an anomaly. To do so properly, I do need the information from yourselves, DDRC and the Coastguard, so please keep providing it.

There are three main areas we need to address in 1996. The first is the Easter hump. Incident rates in April are always on a par with the busiest months, July and August, and the vast majority of April incidents occur over Easter. Most of these incidents are easily avoidable, so:

i We need to realise that the early season, particularly Easter, is an Incident Hot-spot.
ii We must take steps to ensure that we are practised in our core diving skills and that work-up dives are carried out.
iii One of the easiest actions is to make sure that all equipment, including club boats and engines, has been serviced and properly tested in safe conditions before the Easter dive trip.
iv Rescue skills should be practised regularly. It is no use learning the skill and then expecting to be able to carry it out properly for the first time several years later.
v Lastly, try to ensure that early dives are marshalled by your more experienced dive marshals. This part of the season can set the standard for the remainder of the year. Make sure the right example is set.

The second area I want to tackle is deeper diving. I have been tracking the trend towards deeper "average" dives and an analysis of 4 years of data shows that the trend is growing. There are two worrying factors. The first is that many divers carrying out these dives do not have the training necessary. The second is that their equipment is not always adequate. There are several things we can do to improve this:

i Less experienced divers must be educated about the realities of deep diving in British waters, and dismiss the myths regarding depth records and 80 m-plus air dives. Yes, they have been done, but not by recreational divers on a weekend trip.

ii Divers need to be trained in the techniques and practices required to carry out deep dives, the deployment of decompression stations, use of bottom lines, etc.

iii Inert gas narcosis is always a factor in incidents occurring on deeper dives and is probably a significant cause of underwater separation. Divers can work up their inert gas tolerance and, together with proper education, be taught how to deal with narcosis. By paying proper attention to their buddies, divers could also decrease the incidence of separation. Separation drills should always be part of the dive plan and we must ensure that divers realise the importance of following them. It only requires self discipline.

iv The equipment used on a deep dive is significantly different from that used on most club dives. Equipment redundancy and equipment layout are very important and the knowledge and experience in this area is generally gained from practice.

v Planning and marshalling deep dives is far more important than on shallower dives. Detailed plans need to be formulated for separation procedures, finding the shotline, air requirements, etc. The Dive Marshal must be aware of your dive plan so that he or she can effect an immediate rescue if required. When an incident happens at depth, time is critical and discipline is a key factor if safety is to be maintained.

vi Lastly, all these skills should be practised in safe conditions before they are tried for real. It is worth remembering that most of these skills can be learnt and practised on the BSAC Extended Range Diving skill development course.

Analysing incident reports throughout the year, several other things that could do with improvement came to my notice.

i The lack of buoyancy skills is a consistent offender in the incident database. We have taken steps to increase these skills in early training, but we must correct bad buoyancy in later diving as well. With the apparent increase in incidents caused by rapid ascents, this becomes even more important.

ii Dive planning and marshalling seem to be getting worse rather than better. The latest computer can still not plan your dive for you, nor tell the marshal what you are planning to do. These are critical skills and disciplines and we cannot afford to let them go to SEEDS, if you will excuse the pun.

iii Several incidents this year have ended up with casualties dead on the bottom, with weightbelts still firmly strapped around their waists. We need to reinforce the teaching of weightbelt dumping, a basic skill that can be a life-saver. It needs to be included in the buddy check before every dive, not taken for granted.

iv There has also been an increase in the number of near-incidents where controlled buoyant lifts have not been too successful, due to air leaking from the neck seal of the casualty's dry suit. We must make clear in our buddy checks how we can be lifted in an emergency. Make sure your buddy knows to use the BCD to gain adequate buoyancy in such a case.

v Generally it would seem that boatmanship and surface cover skills are in need of improvement in order to minimise the number of divers who end up missing on the surface. Accordingly, we must carry detection aids with us. The Coastguard reports are full of cases where divers could have been located hours earlier had they carried flares, strobes or even a torch. Orange smoke flares are available in waterproof versions and are excellent signalling aids.

vi Finally, let's make sure that both our equipment and ourselves are ready to go into the water.

Have a safe and happy 1996.

Key Words

Deaths, decompression illness, incidents, reprint, safety.

Tim Parish is British Sub-Aqua Club Incidents Advisor.

Reprinted, by kind permission of the Editor, from DIVER, the magazine of the British Sub-Aqua Club, 1996; 41 (1) January: 37-38

The offices of the British Sub-Aqua Club are at Telford's Quay, Ellesmere Port, South Wirral, Cheshire L65 4FY, United Kingdom.

DIVER is published by Eaton Publications, 55 High Street, Teddington, Middlesex TW11 8HA, United Kingdom. The annual subscription is £ 30.00 which must be paid in English pounds.

RESCUE FOR REAL

Keith Waugh

For a long time now I have been concerned about the rescue procedures in the Scottish Sub Aqua Club. Whilst they are very good if they are taught properly, (and be honest, how many Branches of any diving organisation can say that they teach rescue skills really thoroughly ?), the Rescue skills are not really very realistic. The underwater bit is not too bad. Bearing in mind that there are still many divers who dive in wetsuits, we need to be sure that positive buoyancy can be obtained in the casualty quickly, by either inflating the Stab/ABLJ or by ditching the weight belt.

In the dry suit diver we would normally inflate the casualty's suit, but what do we do once we have arrived at the surface? The casualty may still be unable to help him/herself and the rescuer may be somewhat tired or very anxious about the situation. So then what happens? The rescuer, in a state of concern and anxiety, sets about dragging a helpless casualty, including all that person's very unstreamlined equipment, as well as his/her own very unstreamlined equipment, towards safety in a sea which is rarely flat, calm and current free. Sooner or later, but probably sooner, the rescuer is also going to need to be rescued, so now, hey presto!, two for the price of one.

What went wrong? Was the rescuer not fit? Was the equipment at fault? Did the rescuer go around in circles? Was the finning technique poor? The answer to these questions might indeed be YES, but the real answer, I suggest, is that the rescue procedure was totally out of touch with the reality of the open water situation. The rescuer, having established positive buoyancy in the casualty at the surface by injecting air into the casualty's drysuit or Stab/ABLJ, should then have ditched the casualty's weight belt and aqualung and then possibly have ditched his/her own (the rescuer's) aqualung. Pretty drastic stuff, all that gear at the bottom of the sea, but hold on here, we are talking about saving a life, correction, two lives. I hope I can dispel all outcries by saying: equipment is replaceable, lives are not.

So, perhaps we should be getting to grips with reality and teach realistic rescue in the pool and open water training environment. Teaching in the pool would present no problems, but ditching equipment in the sea would, of course, require a bit of organisation to avoid losing equipment.

I would suggest a site approximately 10 m deep with a firm, sandy bottom. Rig 2 shot lines, approximately 6 m apart. The diving party would consist of the Trainee, a "casualty", an Instructor and 2 divers acting as equipment gatherers. It seems like a large number of divers in the water in a small space but it should be quite possible. Only the training party need be between the shot-lines.

So the idea is that the exercise is conducted between the shot-lines. The dropped equipment will land on the seabed between or very near the shot-lines. Certainly the weight belts will. It is an easy task to dive and recover the equipment.

Another advantage of this procedure is that it will teach Branch Dive Organisation and Search and Recovery techniques. However, the main objective will be to demonstrate that a realistic approach to rescue is much more efficient, much less tiring, much less dangerous and much more likely to result in two live divers.

So what do you think? At least give it a try and then inundate the Editor with your observations.

Key words

Rescue, reprints, training.

Reprinted, with minor editing, by kind permission of the Editors, from SCOTTISH DIVER 1995; 34 (3): 55-56. The address of SCOTTISH DIVER is Scottish Sub-Aqua Club, The Cockburn Centre, 40 Bogmoor Place, Glasgow G51 4TQ, United Kingdom.

DIVING MEDICAL CENTRE
SCUBA DIVING MEDICAL EXAMINER'S COURSES

Courses for doctors on diving medicine, sufficient to meet the Queensland Government requirements for recreational scuba diver assessment (AS4005.1).will be held by the Diving Medical Centre in 1996 at:

Royal North Shore Hospital,
Sydney, New South Wales,
8th-10th June 1996 (Queen's Birthday Long Weekend)

Melbourne, Victoria
2nd-4th November 1996
Melbourne Cup Weekend

Previous courses have been endorsed by the RACGP (QA&CE) for 3 Cat A CME Points per hour (total 69).

Information and application forms for courses can be obtained from

Dr Bob Thomas
Diving Medical Centre
132 Yallambee Road,
Jindalee, Queensland 4047.
Telephone (07) 3376 1056 Fax (07) 3376 1056

ROYAL NEW ZEALAND NAVY DIVING MEDICINE COURSE

A 5 day course in Diving and Hyperbaric Medicine is offered by the RNZN Naval Health Services on an annual basis.

The program is aimed at medical practitioners and other health professionals with a special interest in diving medicine as well as diving instructors and dive boat operators.

The course introduces candidates to many of the principles of diving and hyperbaric medicine, but is primarily directed at the assessment of individual's fitness for diving and hyperbaric exposures and the first aid of the common diving illnesses.

The course is recognised by the NZ Department of Labour and the UK Health & Safety Executive.

Venue
RNZN Hospital, Calliope Road, Devonport, Auckland.
Date June 3rd to 7th 1996 inclusive
Fees $NZ 500 (inclusive of GST) which includes a complete set of course notes, morning and afternoon tea/ coffee and a light lunch.

A deposit of $NZ 100 is required to reserve a place. Cheques should be made payable to Diving and Hyperbaric Medicine Unit, RNZNH.

For further information contact

Mrs Anne Powell, RNZN Hospital, Private Bag 32901, Naval Base, Devonport, Auckland 1309, New Zealand. Telephone (64) 09 445 5972. Fax (64) 09 445 5973.

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

Basic Course in Diving Medicine

Content	Concentrates on the assessment of fitness of candidates for diving. HSE-approved course.
Dates	Monday 28/10/96 to Friday 1/11/96
Cost	$A 500.00

Advanced Course in Diving and Hyperbaric Medicine

Content	Discusses the diving-related, and other emergency indications for hyperbaric therapy.
Dates	Monday 4/11/96 to Friday 8/11/96
Cost	$A 500.00

$A 800.00 for both courses

For further information or to enrol contact

Professor John Williamson, Director, HMU,
Royal Adelaide Hospital, North Terrace
South Australia, 5000.

Telephone	Australia	(08) 224 5116
	Overseas	61 8 224 5116
Fax	Australia	(08) 232 4207
	Overseas	61 8 232 4207

ROYAL AUSTRALIAN NAVY

MEDICAL OFFICERS UNDERWATER MEDICINE COURSE 1996

Monday 11/11/96 to Friday 22/11/96

Apply directly to
The Officer in Charge
Submarine and Underwater Medicine Unit
HMAS PENGUIN
Middle Head Road
Mosman, New South Wales 2088
Telephone (02) 9960 0572
Fax (02) 9960 4435

ROYAL ADELAIDE HOSPITAL HYPERBARIC MEDICINE UNIT

Diving Medical Technicians Course

Unit 1 St John Ambulance Occupational First Aid Course. Cost approximately $A 500
Unit 2 Diving Medicine Lectures. Cost $A 500
Unit 3 Casualty Paramedical Training. Cost $A 300

Dates

July 1996

Unit 1	1/7/96	to	5/7/96
Unit 2	8/7/96	to	12/7/96
Unit 3	1/7/96	to	19/7/96

October/November 1996

Unit 1	14/10/96	to	18/10/96
Unit 2	21/10/96	to	25/10/96
Unit 3	14/10/96	to	1/11/96

Diver Medical Technician Refresher Courses

Dates
8/7/96-12/7/96
21/10/96-25/10/96
Cost $A 350

For further information or to enrol contact

Professor John Williamson, Director, HMU,
Royal Adelaide Hospital, North Terrace
South Australia, 5000.

Telephone	Australia	(08) 224 5116
	Overseas	61 8 224 5116
Fax	Australia	(08) 232 4207
	Overseas	61 8 232 4207