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JOURNAL

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South Pacific Underwater Medicine Society Inc.

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ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS,
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ARE DETAILED ON THE BACK COVER

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

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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

President	Dr Des Gorman	Hyperbaric Unit, RNZN Hospital , Naval Base, Auckland, New Zealand
Past President	Surgeon Commodore Tony Slark	19 Stanley Point Road, Devonport, Auckland 9, New Zealand.
Secretary	Dr John Robinson	Science Centre Foundation, Private Bag No 1, Darlinghurst, New South Wales 2010.
Treasurer	Dr Grahame Barry	P.O. Box 268, Newport Beach, New South Wales 2106.
Editor	Dr John Knight	SPUMS Journal, 80 Wellington Parade, East Melbourne, Victoria 3002.
Public Officer	Dr John Knight	34 College Street, Hawthorn, Victoria 3122.
Committee Members	Dr Chris Acott	Hyperbaric Medicine Unit, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000.
	Dr Darryl Wallner	114 Vasey Crescent, Australian Capital Territory, 2601.
	Dr John Williamson	Hyperbaric Medicine Unit, Royal Adelaide Hospital, North Terrace, Adelaide, South Australia 5000.
Education Officer (Co-opted)	Dr David Davies	Suite 6, Killowen House, St Anne's Hospital, Ellesmere Road, Mount Lawley, Western Australia 6050.

New Zealand Chapter

Chairman	Dr Peter Chapman-Smith	67 Maunu Road, Whangarei.
Secretary	Dr Rees Jones	C/o Northland Pathology Laboratory, PO Box 349, Whangarei.

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 \$A50.00 and for Associate Members is \$A30.00.

The Society's financial year is the Australian financial year which runs from July 1st to June 30th.

Anyone interested in joining SPUMS should write to
Dr John Robinson,
Secretary of SPUMS,
Science Centre Foundation,
Private Bag No 1,
Darlinghurst,
New South Wales 2010, Australia.

AIR MAIL DELIVERY

The *SPUMS Journal* can be airmailed at the following annual extra costs.

Zone 1	e.g. Papua New Guinea & the South Pacific	\$ 10.00
Zone 2	e.g. Indonesia and Malaysia.	\$ 12.00
Zone 3	e.g. India and Japan.	\$ 15.00
Zone 4	e.g. U.S.A. and Israel	\$ 18.00
Zone 5	e.g. Europe, Africa & Sth America	\$ 20.00

Those interested in having their copies of the *SPUMS Journal* airmailed should write to
The Editor,
SPUMS Journal
80 Wellington Parade,
East Melbourne,
Victoria 3002, Australia.

INSTRUCTIONS TO AUTHORS

Contributions should be typed in double spacing, with wide margins, on one side of the paper. Figures, graphs and photographs should be on separate sheets of paper, clearly marked with the appropriate figure numbers and captions. Figures and graphs should be in a form suitable for direct photographic reproduction. Photographs should be glossy black and white prints at least 150mm by 200 mm. The author's name and address should accompany any contribution even if it is not for publication.

The preferred format for contributions is the Vancouver style (*Br Med J* 1982; **284**: 1766-70 [12th June]). In this Uniform Requirements for Manuscripts Submitted to Biomedical Journals references appear in the text as superscript numbers.¹⁻² The references are numbered in order of quoting. The format of references at the end of the paper is that used by *The Lancet*, the *British Medical Journal* and *The Medical Journal of Australia*. Examples of the format for 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, Bugg S. The diving emergency handbook. Melbourne: J.L.Publications, 1985

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 sickness (DCS) can thereafter be referred to as DCS.

Measurements should be in SI units. Non-SI measurements can follow in brackets if desired.

Illustrations should be good quality black and white photographs. If the subject requires colour illustrations 35mm colour slides should be provided for reproduction. Always send a copy, not the original.

REPRINTING OF ARTICLES

Permission to reprint original articles will be granted by the Editor, whose address appears on the inside of the front cover, subject to the author's agreement, provided that an acknowledgement, giving the original date of publication in the *SPUMS Journal*, is printed with the article. Where the author has claimed copyright at the end of the article requests for permission to reprint should be addressed to the author, whose address appears at the end of the article.

Papers that have been reprinted from another journal, which have been printed with an acknowledgement, require permission from the Editor of the original publication before they can be reprinted. This being the condition for publication in the *SPUMS Journal*.

SPUMS JOURNAL BACK NUMBERS

Some copies of past issues are available to members at \$ 5.00 each, which includes airmail postage.

The available issues are:-

1984 Vol. 14, No. 2. (1 copy)

This contains papers presented at the SPUMS- RAN Meeting in August 1983 and at the ANZICS -SPUMS Meeting in Rockhampton in October 1983.

1985 Vol. 15, No. 4. (4 copies)

This contains papers from the 1985 Annual Scientific Meeting and from the New Zealand Chapter of SPUMS Meeting in November 1985, including an account of the formation of the New Zealand Chapter.

1986 Vol 16, No 4. (8 copies)

This contains papers from the 1985 Annual Scientific Meeting and from the 1986 Annual Scientific Meeting

1987 Vol. 17, No. 2. (10 copies)

This contains papers from the 1986 Annual Scientific Meeting.

1987 Vol. 17, No. 3. (1 copy)

This contains papers from the 1986 and 1987 Annual Scientific Meetings and papers assessing dive decompression computers.

1987 Vol 17, No 4. (11 copies)

This contains papers from the 1987 Annual Scientific Meeting.

1988 Vol 18, No 1. (28 copies)

This contains case reports of two unusual diving accidents.

1988 Vol 18, No 2. (13 copies)

This contains a report on Australian diving fatalities.

1988 Vol 18, No 3 (31 copies)

This contains papers from the 1988 Annual Scientific Meeting .

1988 Vol 18, No 4 (23 copies)

This contains papers from the 1988 Annual Scientific Meeting .

1989 Vol 19, No 1 (8 copies)

This contains papers on buoyancy control and dive computers

1990 Vol 20, No 1 (3 copies)

Orders, with payment, should be sent to

The Editor,
SPUMS Journal,
80 Wellington Parade,
East Melbourne,
Victoria, 3002,
Australia.

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

Lung function reference values for FEV ₁ , FEV ₁ /FVC ratio and FEF ₇₅₋₈₅ derived from the results of screening 3788 Royal Navy submariners and submariner candidates by spirometry	G.J.Brooks, R.J.Pethybridge and R.R.Pearson	187
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THE MARINE STINGER HOTLINE

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For advice about the treatment of marine stinger injuries dial **008-079-909**.

DIVER EMERGENCY SERVICE 008-088200

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For access to the same service from outside Australia ring ISD 61-8-223-2855.

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.

ADVERTISING IN THE SPUMS JOURNAL

Advertising space is available in the SPUMS Journal. Rates are available on application to the Editor, SPUMS Journal, 80 Wellington Parade, East Melbourne, Victoria 3002.

Deadlines are February 14th, May 14th, August 14th and November 14th.

EDITORIAL

In this, my first editorial, I wish to pay tribute to the hard, effective and often unrecognised work that my predecessor, Dr Douglas Walker, has devoted to SPUMS and the diving community in general. He took over as editor of the SPUMS Newsletter early in 1975 and built it up so effectively by soliciting contributions that in by early 1977 it became the SPUMS Journal/Newsletter, with over 40 pages. This title continued until December 1979 when the title became the SPUMS Journal and production was moved to Melbourne. In the early days of his editorship he hand addressed over 200 envelopes for each issue. In his editorials and papers Douglas Walker has pushed the need for logical thought in diving safety based on knowledge, of the types of accidents, of the equipment used, of the faults in equipment, behaviour and training, before acting on theories of causation. His many years of work, starting with a report on the 1973 diving deaths and continuing to a paper in this issue, elucidating the causes and patterns of Australian diving-related deaths, popularly known as Project Stickybeak, is unique in its depth of focus. Others publish bigger series, but the information is far less detailed and so less informative when it comes to providing a basis for effective action. It is hoped that, in conjunction with the Australian Underwater Federation, the collected works (1973-1988) of Project Stickybeak will soon be published in book form.

The diving medical is a topic of considerable interest and disagreement. It was raised in a paper given at the 1990 Annual Scientific Meeting and the Annual General Meeting requested that the Society provide a position paper on the topic. The Executive Committee delegated this task to the Education Officer, Dr David Davies, whose paper appears below as a guest editorial

John Knight

DIVING MEDICAL EXAMINATIONS

During the last Annual General Meeting held in Palau, a long discussion was held about the Society's attitude to, and policy concerning, diving medical examinations. This Society has long been concerned that some sections of the community believe that a Diving Medical Examination (DME) prior to the commencement of a diving course is an unnecessary imposition, a waste of time, and merely an excuse for doctors to rip off potential divers. It comes at a time when the trainee knows very little of what lies ahead and when he is paying out large quantities of cash for what he is told is essential equipment.

Such attitudes are found, not only among divers and potential divers, but also within the diving instructor community itself. The medical profession too is not guiltless, as neither the State Medical Registration Boards nor the AMA recognize Diving Medicine as a speciality in its own right. Approaches to these organizations by the Society have been met with thunderous apathy in the past.

Although for the healthy, fit, sensible individual diving is a safe pastime, should anyone of these elements be missing, diving can become quite hazardous and potentially lethal. For this reason alone, all potential divers should be medically screened to eliminate possible life-threatening pre-existing medical conditions.

Diving has often been compared with other outdoor adventure pastimes such as rock climbing, hang gliding, parachuting and caving, none of which require a medical examination. Unlike these, diving is conducted in a dense,

non-breathable medium which is subject to tides, currents and wave action. Further more, small changes in depth result in relatively large changes in pressure with alterations in volume and density or gases.

This Society has long advocated that Medical examinations for all divers be conducted by doctors who have a complete understanding of the physical and physiological effects of diving. To obtain such an understanding, it is strongly recommended that the doctor undergo appropriate training and there is a number of well conducted courses being run at regular intervals. In the past doctors with the required training have been few, widely and unevenly distributed and often not well known. With the educational facilities now available there is little excuse for what Dr Carl Edmonds once called "the Mickey Mouse Medical".

It must also be acknowledged that the training course in itself is not sufficient to ensure adequate knowledge and skill in the field. This initial infusion needs to be regularly supplemented with experience, reading, and attendance at educational meetings. The SPUMS Journal is the ideal medium for finding the latest information on the various aspects of diving medicine.

The correct reasons for having a diving medical examination need to be explained to all trainee divers prior to their commencing a diving course. This would help to eliminate many of the misconceptions. It is appreciated that in some areas of the country, financial pressures encourage training agencies to cut corners and perhaps recruit trainees

who may prove unfit for diving. It is known that such agencies even coach their trainees on how to disguise a medical disability, such as asthma, in order to pass the medical examination. Such an attitude is to be abhorred and actively discouraged.

The role of the medical practitioner in the DME is that of adviser. There are many conditions which are not absolute contra-indications to diving. Even with the absolute contra-indications, there is often some difference of opinion between various authorities. The examining physician must have the knowledge and be in the position to advise the trainee as to his medical suitability to undertake the course. He must be able to provide reasons, logical argument and suitable explanation if the trainee does not meet the fitness criteria.

As man is not an aquatic mammal, it follows that he is not automatically fit to dive. The examining doctor can only determine whether or not the person he examines conforms to the standards at that time. In all divers, fitness varies from day to day, month to month and year to year. For this reason, a medical examination last week may give no

indication of fitness to dive next week, next month or next year. Any person who suffers a debilitating disease should undergo a further medical examination after recovery, prior to resuming his diving career.

The Federal Government has seen fit to exclude diving medical examinations from its Medical Benefits Schedule so that divers are obliged to cover the entire cost themselves. Despite submissions by this Society to the Government, it seems unlikely that this situation will alter in the foreseeable future.

As a segment of the overall cost of a diving course including the cost of tuition and equipment, the proportion made up by the medical examination is very small and divers should be encouraged to seek quality of service rather than a bargain price. Such a service should include lung function tests, audiometry and tympanometry and cardiography if indicated. Many diving doctors also require a chest X-Ray.

David Davies
Education Officer SPUMS

ADDRESSES FOR SPUMS CORRESPONDENCE

Following decisions taken at the 1990 Annual General Meeting the Society has arranged with the Science Centre Foundation to undertake the routine office work for SPUMS.

All correspondence for the **Secretary** (Dr John Robinson) should be addressed to

**The Science Centre Foundation
Private Bag No 1
Darlinghurst, New South Wales 2010.
Australia**

Subscriptions and correspondence for the **Treasurer** (Dr Grahame Barry) should be addressed to

**P.O.Box 268
Newport Beach, New South Wales 2106
Australia**

All matters relating to the **SPUMS Journal** should be addressed to

**Dr John Knight,
Editor, SPUMS Journal
80 Wellington Parade
East Melbourne, Victoria 3002
Australia**

SPUMS NOTICES

THE MINUTES OF THE ANNUAL GENERAL MEETING HELD ON THURSDAY, JUNE 7, 1990 AT THE PALAU PACIFIC HOTEL, PALAU AT 1620

Present

All members attending the Scientific Conference

Apologies

Drs Hurst, Refisch, Williamson and Bridger

Business

1 MINUTES OF THE PREVIOUS MEETING

These have been published and were taken as read.

Moved that the minutes be accepted as true record. Dr C Lourey, seconded Dr D Davies. Carried.

2 BUSINESS ARISING FROM THE MINUTES

Dr Davies spoke on the incorporation of the Society.

This had been underway in the previous two years and it was explained that this was necessary due to threatened litigation. Whilst the threatened litigation did not eventuate, it came to this Society's attention that if we are not incorporated, each member is individually responsible for any expenses and costs pursuant to litigation. Once incorporated, the Society is liable and only the Society's assets are liable. Dr John Knight had been involved in extensive negotiations with lawyers and an acceptable constitution having been arrived at, this will be distributed to members shortly.

No further business was raised.

3 REPORTS

3.1 Reports were presented by the President and the Secretary and are published in this issue of the Journal.

Moved that the reports be accepted. Dr C Lourey, seconded Dr J McKee. Carried.

3.2 The Treasurer (Dr G Barry) presented his report. A financial statement to April 30, 1990, was read and copies were available. The Treasurer stated the Society's financial position was sound and that annual subscription need not be raised from its present \$50.00 for members and \$30.00 for associates. It was noted that Dr P Chapman-Smith had presented \$3,000.00 arrears from New Zealand members.

Moved that the Treasurer's report be accepted. Dr D Davies, seconded Dr J Robinson. Carried.

The President, Dr Slark, announced that, following an Executive decision, in future New Zealand Chapter subscriptions will be directly to the Treasurer on an individual basis. The same subscription will apply to both New Zealand and Australian members.

4 EXECUTIVE COMMITTEE:

Two nominations were received for the post of President and a ballot had been held. The assistance of the Science Centre Foundation was sought to count the votes. The successful candidate was Dr Des Gorman. There were no other contested positions. There was no nomination for Editor and the Committee had nominated Dr J Knight. The Committee is

President:	Dr D Gorman
Immediate past President:	Dr A Slark
Secretary:	Dr J Robinson
Treasurer:	Dr G Barry
Editor:	Dr J Knight
Public Officer:	Dr J Knight
Committee:	Dr C Acott
	Dr D Wallner
	Dr J Williamson
Education Officer (co-opted)	Dr D Davies

A motion was moved by Dr Slark, expressing the thanks of the Society to the outgoing Editor (Dr D Walker) and noting his many years of hard and selfless work in the publication of the Journal. The Executive also wished to express its pleasure in the appointment of Dr J Knight as the incoming Editor. The motion was seconded by Dr D Davies. Carried.

Dr A Slark stated the outgoing Secretary has been co-opted to assist the Committee as Education Officer. At the next Annual General Meeting there will be a motion put to include the position of Education Officer in the Society's Executive.

5 SCIENCE CENTRE FOUNDATION

Dr A Slark informed the meeting that the Executive Committee recommended that the Science Centre Foundation (SCF) take over routine administrative work from the Secretary. It was noted that the expenses incurred were moderate and not necessarily committed beyond the joining and annual fees. The President hoped that the Meeting would endorse this view unanimously. Dr Barry stated that the Society suffered from a lack of a permanent fixed address. Moved that the Committee's recommendation be adopted. Dr M Logan, seconded Dr J Lloyd. Carried.

A point of order was raised by Dr Sutherland. He expressed a wish that Mrs Ruth Inall of the SCF leave the room so that free and democratic discussions on this proposal could occur. Some argument ensued as to technicalities of whether this was a motion or not, but finally, Mrs Inall voluntarily left the room. A discussion followed in which Dr Logan noted the difficulty in getting people to work on the Executive Committee and felt the meeting should not

oppose this motion. Dr Douglas wondered if there was some alternative to the SCF. He also wondered about the ultimate cost. Dr Barry commented that a full-time secretary was not a proposition, given the financial status of the Society. He also indicated a fee increase was not automatic and that the cost of the Science Foundation will depend on use. At the moment, he noted the commitment was \$25.00 joining fee.

Finally, the motion to use the Science Centre Foundation's facilities was again put by the original proposer and seconded by the original seconder, and this was carried unanimously.

6 ANNUAL GENERAL MEETING 1991

There was discussion on a number of points with regard to venue. It was noted by the incoming President (Dr Des Gorman) that there were many options available and a presentation was received by Mr Skinner of Allways Travel on the various advantages and disadvantages of these resorts. The President noted that the Executive would like to have the site of AGM's known in advance so continuity of meetings could be maintained and we could match speakers to meeting, bearing in mind the cost of having speakers. With this in mind, it was felt that the 1991 meeting should be held in the Maldives and this was discussed at an Executive meeting following the AGM and confirmed as the site. The actual time will be subject to the availability of the hotel. Mr Skinner will be investigating the feasibility of other sites favourably commented on by the meeting for the next few years and will report to the Executive.

7 ANNUAL SCIENTIFIC MEETINGS

Dr Gorman made a statement

7.1 He had requested the written transcripts of presentations at the meeting to be provided prior to the meeting. He was pleased that all but one speaker had complied. This is of great assistance to the Editor.

7.2 He had investigated the financing of the meetings and was able to report that no member's conference fees, nor any other payments made, have been used to subsidise trips by any other member.

7.3 He found that there was no evidence that conveners had been paid for their job and that all had done it without payment. He also found that there were no subsidies or kick-backs, to any member of the Executive.

7.4 He explained why it was possible for the New Zealand contingent to travel at considerably less expense than the Australian contingent. The fundamental reason was the size of the group, there being no discounts available for a group of 150.

8 AUDITOR

Moved that David Porter FCA continue as auditor. Proposer Dr Slark, seconded Dr Barry. Carried.

9 THE JOURNAL

Dr Knight spoke acknowledging the outstanding job Dr Douglas Walker had done over many years. He thanked

the speakers for the excellent material from the Hobart and Vanuatu meeting and the scripts from the Palau meeting. With the addition of theses from diploma students under Dr Gorman's encouragement, he felt the Journal had a satisfactory forward bank of articles. The price of the back issues of the Journal is \$5.00, if the price was higher the Society would lose Australia Post Category B publication status. It was requested that all members forward suggestions of any material which is useful for the Journal, including letters, and anything which might rightly appear under the heading of "Gleanings". The Editor however reserved the right to edit contributions.

10 LIFE MEMBER

In the President's address it had been mentioned that Dr Lourey was recommended by the Executive for election by the A.G.M. to life membership. This motion as carried by acclamation.

Dr Lourey spoke thanking the members and all the Executive members he had had the pleasure of working with. He also offered congratulations to the new President.

11 OTHER BUSINESS

Dr John Archdeacon of Cairns made a statement. He felt a considerable number of divers had arrived in ICU as a result of misleading the doctor at their diving medical. He wished the Executive to prepare a policy statement regarding diving medical examinations with the intention of encouraging a normal patient-doctor relationship and, in particular, removing any incentive for patients to mislead their diving medical doctor.

The President noted this matter should more correctly have come forward as "Business with notice". Nevertheless, the matter was allowed to be debated and a number of statements were made resulting in a motion. "This meeting calls upon the Executive to prepare a policy statement regarding diving medical examinations". Moved Dr G Davis, seconded Dr A Slark. Carried.

There being no further business, the meeting closed at 1825.

AUDIT REPORT

I have conducted various tests and checks as I believe are necessary considering the size and nature of the Society and having so examined the books and records of The South Pacific Underwater Medicine Society for the year ended 30 April, 1990 and report that the accompanying Statement of Receipts and Payments has been properly drawn up from the records of the Society and gives a true and fair view of the financial activities for the year then ended.

David S. Porter,
F.C.A., Chartered Accountant
(Registered under the Public Accountants Act, 1946, as amended).

16 May, 1990, Newport Beach, NSW 2106.

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
Statement of Receipts and Payments for Year Ending 30 April 1990

	1990	1989	1988
Opening Balance			
Standard Chartered Finance Ltd.	1,000	1,000	1,000
National Mutual Royal Bank	4,228	5,177	7,361
National Australia Bank	1,503	789	403
Cash on hand	10	22	26
	6,741	6,988	8,790
Income			
Subscriptions	33,001	24,074	27,475
Interest	2,260	1,161	1,787
Journal sales and advertising	1,845	-	-
	37,106	25,235	29,262
	\$43,847	\$32,223	\$38,052
Expenditure			
Secretarial	3,016	4,143	3,735
Stationery	570	576	198
Journal	14,739	7,310	10,487
Postage	6,017	4,047	4,198
Travel	5,774	7,775	10,335
Equipment, see note	2,454	372	850
Miscellaneous	550	122	358
Bank charges	311	232	286
Returned cheques	32	25	25
Audit	175	200	200
Legal expenses	-	180	392
Donation to D.E.S.	-	500	-
Uncashed cheques	351	-	-
	33,989	25,482	31,064
Closing Balances			
Standard Chartered Finance Ltd.	1,000	1,000	1,000
National Mutual Royal Bank	8,305	4,228	5,177
National Australia Bank	441	1,503	789
Cash on hand and stamps	112	10	22
	9,858	6,741	6,988
	\$43,847	\$32,223	\$38,052

Note : Equipment is written off as purchased.

PRESIDENT'S REPORT

It is a great pleasure to be at yet another very successful meeting of SPUMS. This year has seen us all able to meet at yet another exotic and fascinating venue, and in even greater numbers than last year. The meeting at Vanuatu was particularly successful in my view combining good diving and an interesting program. We had two excellent speakers, Jimmy How from Singapore, and Mike Davis from Christchurch. You will be able to see their papers printed in the journal, but this scarcely does justice to the skill they both have in presentation. As well as providing good diving, we had catering of a high standard, related perhaps to the French influence in the country. The dive at Hat Island I rate as one of my most memorable.

A week later we had the UHMS Meeting in Hawaii, and I was pleased to see how many of this society were able to attend. This was as well organized as usual, and we had a very enjoyable and interesting meeting of high academic standard. However I was pleased that we had not followed up on earlier suggestions to hold our two meetings conjointly. It would have been very difficult to manage within the time frame, and we would have had little opportunity to get any diving. The pattern of our meetings is quite different, and our interests would have been submerged by the larger organization.

There are a number of changes on the Executive Committee. Our Secretary, David Davies has decided, regrettable, to retire from this position. I am sorry that he has made this decision as he has been a very hard worker for the Society, and a great help to me and to other presidents as we have come to office. I trust that my successor has the good fortune to have such industrious help from his successor. However, I hope that he will continue as a member of the Executive Committee. I would like to pay tribute to the sterling service of our Treasurer, Grahame Barry, over many years of handling the Society's finances, a task he is happy to continue. Peter McCartney has not stood for re-election to the committee and I regret that we will not have the benefit of his wide experience and advice. This year Chris Lourey has decided to leave the Executive Committee after many years of consistent work for the Society during which he has steered it through many difficulties. He has been our President and acted as Convener for many AGMs, and in doing this he has given us the opportunity to enjoy visiting such places as this. I know that you will join with me in thanking him for all the time he has given the Society. The Executive Committee has decided to recommend that this meeting confers Life Membership of the Society on Chris Lourey in recognition of his outstanding service to the Society. Life Membership is a high honour as the Society can only have five Life Members. Douglas Walker (last year's Life Member) has not nominated for the position of Editor, breaking a connection with the Journal which took it from a newsletter sent to a relatively small number of

members to the polished production of today with a distribution of over 900 copies. The Society owes a lot to the years of work that Douglas has given so freely and unstintingly both to the Journal and to the cause of diving safety and the elucidation of the causes of diving accidents.

It was with great regret that I learnt of the tragic death of Anthony Newly from injury by a stingray. Those of us who have attended Annual Scientific Meetings from 1977 to 1988 have attended meetings where the travel arrangements, hotel and diving had been organised by Anthony. His cheerful competence and untiring efforts helped make these meetings the successes they were. He was a friend to all of us. The Society has sent condolences to Linda and the children.

The journal presents our public face and this has in my view improved very greatly in the last year. Although we may regret the passing of humour from the cover, I am sure you will agree that what is after all a serious educational venture should not put on comic clothing, and the cartoon would be better on the inner pages somewhere, and hope that the editors may arrange this for the future. I have also been pleased to see the development of the letters section and hope that we may see some provocative arguments therein.

I must make some comments on the advance of the academic status of the Society. Our censor, Des Gorman, has achieved a great deal in this position, ensuring that the standards for those offered our diploma under the "grandfather" clauses have fulfilled the criteria which your executive agreed to (he refused to accept the "Oh yes I know him, he's O.K." evaluation), and has insisted that other applicants fulfilled the time involvement, course attendance, and paper presentation that we wished for. There have been some very interesting papers presented that I hope will be published in the journal. The discipline of making some effort of this nature is very good for us all. There have also been excellent courses in the subject run in Adelaide and Christchurch, and of course the work of the School of Underwater Medicine at HMAS PENGUIN has continued.

The executive has not met in the last year, for a variety of reasons. It is difficult to get together from such diverse parts of the area, but this has resulted in worthwhile saving for our Treasurer. Communication has been improved however by the use of fax and if we had this facility for all our correspondence such meetings would be less necessary in the future. Our attempts to investigate the possibility of a joint meeting in Indonesia with their Underwater Society have been made somewhat difficult by the unreliability and sloth of postal communication, and may preclude further progress with this suggestion.

Hyperbaric Medicine is making great advances in the region. Western Australia now has a unit, associated with the Emergency and Intensive Care Departments of Fremantle Hospital. At the other side of our area building is

advanced for the installation of a new chamber complex at the RNZN Hospital in Devonport. It is anticipated that this will operate by the end of the year.

I have much enjoyed my time as President of the Society, and feel pleased leaving the position with the Society in such evident strength. It is particularly pleasing to me to hand over the Presidency to Des Gorman. I know that further advances will take place under his guidance.

Tony Slack

SECRETARY'S REPORT 1990

It is my pleasure to present this, my fifth, Annual Report to the Society.

1 Incorporation

You will recall that we have been working towards incorporation now for a couple of years to reduce the liability of ordinary members to legal action as a result of activities of other members of the Society. The Corporate Affairs Commission of Victoria finally saw fit to approve our application on April 11th, 1990.

As Public Officer, Dr John Knight has been overseeing this process and has ensured that the Constitution and Rules of the Association are set out to our requirements rather than those of the Victorian Public Service.

The general membership of the Society will probably not notice a great difference but there will be some administrative changes and an increase in bureaucratic paper shuffling.

2 Executive Committee

This meeting sees the departure from the Executive of two long standing members, Dr Chris Lourey and Dr Peter McCartney. Dr Lourey has been on the Executive since the mid 70s and has served in just about every capacity on the Committee. He was instrumental in organising the highly successful combined meeting with the Republic of Singapore Navy in Singapore in 1980 which put SPUMS on the international scene in diving medicine. The Society has benefited significantly from his efforts over the years and I am grateful for his help, advice, and recall of significant events.

Dr McCartney served on the Committee for about 5 years and is an active teacher and researcher in Hobart. He has been a constant source of ideas. As a result of his work, the medical undergraduates in Hobart are the only ones in Australia that receive formal tuition in diving medicine.

To both these men, we extend our thanks for their service to the Society and wish them well in the future. Their positions will be taken by Dr John Williamson and Dr Darryl Wallner.

3 Legislation

1989 saw the introduction in Queensland of the new Workplace Health and Safety Act of which there is a whole section about diving operations, both professional and recreational. Of note is Regulation 260(2)(b) which states that a student engaged in an entry level certification course has been certified as fit to dive by a legally qualified medical practitioner with experience in diving medicine. For divers doing non-certification resort courses, the diver must complete the recognised medical questionnaire and be assessed as fit for diving by the diving instructor.

I see this as a start but the regulations could have stated that the medical practitioner must have completed a recognised course in diving medicine. Even so, there will be a rush of doctors wanting to attend such courses. Already Dr Bob Thomas of Brisbane has completed one course and is organising further courses later in the year. From the Society's point of view this is an ideal opportunity to recruit new members. I have written to all those who did Dr Thomas' first course and have had one join up as a result. In addition, I have written to Dr Thomas requesting that he sends the Secretary a list of all those doctors who complete his courses so that the Society can maintain a register of doctors who have training, whether or not they are members of the Society.

4 Education

One of the prime aims of the Society is education of divers, doctors and other interested parties such as government departments. Apart from Tasmania, no Australian medical undergraduate receives formal tuition in diving and hyperbaric medicine. In recent communication from a member of the University of Newcastle Medical School I was told that if SPUMS made approaches with an appropriate syllabus then it would be seriously considered. This will be a job for the incoming executive to consider. Along the same lines, it has been suggested, and your Executive is currently investigating the feasibility, that SPUMS makes one, or a series, of video tapes on various aspects of diving medicine.

Education of doctors remains a priority. Courses continue to be run in Adelaide, Dr Thomas is now holding basic courses in Queensland, and the Fremantle Hospital is planning a course for later in the year. For those members in other states, it is just a matter of one person with enough time and energy to get a group of trained people together and coordinate a course. All these should be self funding.

5 Secretariat

In the last Annual Report I suggested that the Society was becoming too big to be run by a part-time amateur. To this end I have held discussions with the Science Centre Foundation which is located in Sydney. You will have heard Mrs Inall's presentation this week and will agree that employment of such a group will enable your Executive to achieve the aims of the Society rather than remain bogged down with routine administration.

6 Obituary

I wish to record the sorrow we all felt with the news of the tragic death of Anthony Newly in November 1989. Anthony was swimming with giant rays at Waya Island when he struck in the leg. This injury, which divided his femoral artery, resulted in his death from blood loss before he could be evacuated to medical help.

Anthony was a great friend to the Society and was a driving force in making these annual meetings the success that they have become. His water skills were excellent and it was always a pleasure to dive with him. The Society owes him a great debt and he will be sorely missed by those who knew him.

7 Acknowledgements

In conclusion I wish to acknowledge the work done by the Executive members over the past year and the help that they have given me. Dr Gorman's move from Adelaide to Auckland has made quite a difference in that he is much more difficult to contact in a hurry, but it is always worthwhile in the end. I have mentioned the help I have received from the President, Dr Slark and the Public Officer, Dr Knight. Dr Barry continues to keep a close eye on the finances and I am pleased that he is prepared to continue in that position. I have thoroughly enjoyed my time as Honorary Secretary of the Society, but it is now time for fresh blood to take over and I wish Dr John Robinson all the best in this position.

David Davies

MINUTES OF THE EXECUTIVE MEETING HELD IN THE BOARD ROOM AT PALAU PACIFIC HOTEL ON THE 1ST JUNE 1990 at 1600

Present

Drs Slark, Gorman, Davies, Barry, Knight, Acott, Wallner, Adkisson by invitation.

Apologies

Drs Williamson, Chapman-Smith, McCartney, Walker.

Business

1 MINUTES OF THE PREVIOUS MEETING:

Having been distributed and published these were taken as read.

2 BUSINESS ARISING FROM THE MINUTES:

2.1 Incorporation. Dr Knight reported that the Society is now incorporated as from the 12th of April, 1990. Copies of the new constitution were circulated and will be distributed to the membership in due course. (This was posted with the SPUMS Journal Vol 20 No 2)

2.2 Standards.

2.2.1 Committee sitting to determine a standard for Scientific Divers has been disbanded. The Secretary will contact Dr C Lowry informing him of the fact.

2.2.2 Dr Knight presented a full report on the deliberations of the committee considering the entry level recreational diver standard. The problem of costs for diving medical examinations was discussed. It was agreed that there is a need to improve communications with training organisations on what is needed from a diving medical examination. Dr Davies moved that the report be accepted. Seconded Dr Barry. Accepted

3 EXECUTIVE COMMITTEE 1990/91

3.1 Nominations have been received for:

President:	Dr D Gorman Dr D Davies
Hon Secretary:	Dr J Robinson
Treasurer:	Dr G Barry
Editor:	No formal nomination
Public Officer:	Dr J Knight
Committee:	Dr C Acott Dr D Wallner Dr J Williamson

3.2 A postal ballot had been initiated by the Secretary and conducted with the Science Centre Foundation for the position of President. Dr Gorman was the successful candidate.

3.3 It was noted that there were no formal nominations for the position of Editor. Although Dr Walker had indicated his willingness to continue no nomination had been received by the Secretary. Dr Knight was then proposed by Dr Lourey, seconded by Dr Davies for that position. Dr Walker's role with the Society was discussed and his contribution acknowledged. The Secretary will write to inform him of the decision and the Society's appreciation.

3.4 The Committee noted the retirement of Dr Lourey and expressed its appreciation for his efforts in helping to keep the Society together during a difficult time and acting as Convener of the Annual Scientific Meeting on several occasions. The Committee unanimously approved his nomination as Life Member of the Society.

3.5 In view of Dr Davies' work as Secretary of the Society for the past five years and his working knowledge of the Society he would be co-opted onto the new Executive as Education Officer. A formal motion will be placed before the next AGM that the position of Education Officer becomes a permanent Executive post.

4 GENERAL BUSINESS:

4.1 Travel Costs.

4.1.1 In response to letters from members of the Society suggesting cheaper fares could be obtained for travel to the meeting, the Executive received a report from Allways Travel. It was noted that, despite popular opinion, large tour groups do not

have greater bargaining power than small ones either with airlines or hotels. A detailed costing of the conference fee was supplied by Allways.

4.1.2 Both Dr Gorman and Dr Davies had made separate enquiries with independent travel agents and Continental Airlines and agreed that the costing supplied by Allways was very competitive.

4.1.3 It was agreed that selected letters received by the Secretary be published in the Journal along with a reply unanimously supported by the Executive. Allways would also be invited to reply.

4.1.4 The incoming President (Dr Gorman) will make a report to the AGM later in the week.

4.2 Society Spokesman.

4.2.1 It was reiterated that the only members entitled to speak on behalf of the Society are the President, the Secretary and any other person specifically appointed by the Executive to speak on a particular subject.

4.2.2 The Committee agreed that the NZ Chapter is not a separate Society but remains part of the parent body. The NZ Executive is not entitled to enter into public debate without ratification by the Executive Committee of SPUMS.

4.3 New Zealand Chapter.

4.3.1 The NZ Chapter is constituted to run local affairs only and to conduct its own meetings. Its responsibilities to the parent body have been neglected over recent times.

It was moved by the Treasurer and seconded by Dr Gorman that "Control of the finances in NZ will revert to the Treasurer who will receive all subscriptions directly from members. All Journals will be mailed directly to members by the Editor. Secretarial and other expenses will be paid by the Treasurer on receipt of a detailed account". Passed.

4.4 Science Centre Foundation.

The Secretary reported on his negotiations with the Executive Director of the Foundation. It was noted that the routine administration of the Society is occupying in excess of 12 hours per week. The membership currently stands at about 800 members of whom 300 are not Australian residents.

Employment of a secretarial group such as the Foundation to deal with routine administration will allow the Secretary time to better manage Society affairs. It was moved by Dr Knight seconded by Dr Barry that the Society join the Foundation with a view to engaging their services as and when required.

4.5 SPUMS Video Enterprises.

The Secretary reported that as part of an overseas aid project by AIDAB he had been approached as to the feasibility of making a video teaching program on various aspects of diving medicine. A long discussion followed on the cost effectiveness of this method of information dissemination. It was suggested that time constraints precluded it from this year's budget.

4.6 Annual General Meeting 1991.

4.6.1 There has been no response from Indonesia to the Secretary's last letter. The Secretary will write to Dr Prayitno again suggesting that time is now too short to organise a combined meeting in Jakarta in 1991 and perhaps it should be delayed. Dr Slark suggested that the NZ Military Liaison Officer in Jakarta may be recruited to assist with negotiations.

4.6.2 Discussion was held about Guest Speakers. The final choice will depend on the venue. A number of alternatives were discussed and the Executive will investigate further before a decision is made.

4.6.3 Dr Gorman offered his services as Convener for the next Annual Scientific Meeting. Site inspections will be conducted by Executive members available at the time.

5 NEXT MEETING:

It was agreed to hold another meeting later in the week to complete any outstanding business.

Meeting closed 2100.

THE MINUTES OF THE SPUMS EXECUTIVE MEETING HELD ON FRIDAY, JUNE 8, 1990 AT THE PALAU PACIFIC HOTEL, PALAU

The Meeting commenced at 1010.

Present

Drs Gorman, Acott, Chapman-Smith, Davies, Knight, Robinson, Slark, Sutherland and Wallner.

Business

1 NEW ZEALAND MEMBERS

An explanation as the delay in collection of New Zealand subscriptions was presented by Dr Chapman-Smith. He explained that the situation had now been rectified and their accounts were in order. Dr Gorman noted that the New Zealand fees are to be paid directly as from now to the Treasurer. The New Zealand Executive is to invoice Dr Barry for any costs incurred.

2 MEETINGS

Dr Gorman indicated that, in future, meetings should be simplified by the introduction of teleconference with fax facilities. These have been proven to be extremely efficient in the past. The Secretary noted that in order to conduct such conferences successfully, a disciplined and precise format will need to be adhered to and instructions seeking co-operation from each executive member concerning this will be issued shortly. Dr Knight suggested a fax be provided by the Executive for the Secretary and this was agreed to. Dr Davies noted that a teleconference costs \$12.00 per minute.

Dr Gorman noted that the New Zealand Chapter must be at all times represented on the Executive and be notified of all meetings.

Dr Knight felt a new letter head was now required that we are incorporated.

3 ANNUAL SCIENTIFIC MEETINGS

Discussion was entered into and it was agreed that the next year meeting will be in the Maldives. The subject is "Diving equipment and diving accidents". Invited speakers will be Peter Bennett and Glen Egstrom. The conference time could not yet be decided as Geoff Skinner needs to enquire regarding the resort's availability. However, this should be settled in a matter of days.

4 OTHER MATTERS

Dr Gorman noted that the Society must at all times be totally separate from UHMS. Further, SPUMS is not a regional alternative to UHMS but a body dealing specifically with sports diving medicine and as such holds a unique position. It was noted that the Society is expanding rapidly and Dr Gorman indicated support for Chapters in any part of the world provided the initiative came from those concerned. Dr Raymond Rogers had already expressed an interest in forming a North American Chapter and it was agreed that this would be an excellent development. The Secretary was instructed to pass this information to Dr Rogers and subsequent to the Meeting, has done so and Dr Rogers will be in contact when and if further developments occur.

There being no other business, the President concluded by stating that it will be necessary for an executive meeting to occur in about 8 weeks' time. In particular, discussion is required regarding:

- a Speakers and themes for future conferences and this should be planned several years in advance in order to maximise the worth of the conference and;
- b Meeting venues, and again, this should be planned well in advance so that a match can be made between speakers and venues in order to achieve the most cost effective way of importing speakers.

At this point Dr Knight had to leave the meeting

6 SCIENCE CENTRE FOUNDATION

Mrs Ruth J Inall of the Science Centre Foundation (SCF) was invited to address the Executive privately. She requested a letter from the Secretary to join the Foundation plus \$75.00 fee. This is to be presented to the Governors in August.

After discussion as to who will be responsible for liaising with the SCF, it was decided this would be only through one member of the Executive. Consequently, the Executive authorised all discussions to be channelled through the Secretary only. No other person has authority of the Executive to communicate business relating to the Society. Mrs Inall informed the meeting that Liz Swaby will be

delegated as first point of contact for the Society.

Mrs Inall recommended that

6.1 The address of the SCF is used as that of the SPUMS for purpose of contact and an entry in the telephone directory for SPUMS using the SCF telephone number.

6.2 The letterhead should indicate that correspondence goes to the SCF and that SPUMS is an incorporated body.

6.3 After discussion regarding records of membership, it was decided to convert these to computer records and it was decided a database should be drawn up, both in floppy and hard disk. This is to prevent erasing of information. It was decided that David Davies be delegated to decide what sort of information should go into the database. After discussion, he is to give this database to Des Ireland for suggestion as to improvements or otherwise, and this is then to be discussed at the next Executive meeting.

7 SUBSCRIPTION NOTICES

It was decided these would be continue to be handled by Dr Graeme Barry.

8 EDUCATION OFFICER

Some discussion followed concerning the office of Education Officer.

8.1 It was pointed out by Ruth Inall that Government assistance is available in some circumstances to help developing countries. She felt that there is a possibility that SPUMS may be able to be assisted in the expenses of recommended speakers if they were seen to be of benefit to the developing country. This matter is to be discussed at a later date.

8.2 It was felt that the availability of money to make a video was not appropriate as a video is difficult and time consuming to produce and rarely addressed the major problems of the Third World, in particular as diving medicine seems to be the least of their problems, and that no member of the current Executive had the time to put into the development of such a video. The President spoke suggesting that money available for a video might be better used on a series of symposia in the Third World. There was some doubt as to whether money was available from AIDAB for this purpose, but Dr Gorman indicated he will write to AIDAB requesting their view whether aid will be available for the meeting in 12 months.

9 OTHER MATTERS:

Mrs Inall suggested that the Society look at its budget. She stated that here is no forward planning of the budget nor is there any ongoing data available to suggest how the budget is performing. She suggested a monthly review of budget projection should be instituted and a formal budget sheet be drawn up. She stated that although the finances appeared healthy, the budget could be easily exhausted by a shift in previous spending patterns.

In the absence of Graeme Barry, the Secretary took it upon himself to write, requesting a budget and clarification of the financial statement to be presented at the next meeting of the Executive. (This has subsequently been carried out).

There being no other business, the meeting closed at 1154.

SPUMS DIPLOMA OF DIVING AND HYPERBARIC MEDICINE

Owing to clerical errors four names were left off the list of diploma holders published on page 97 in the last issue. The full list, in alphabetical order, of those awarded the diploma up to June 1990 is below.

Dr C Acott	Dr C Macfarlane
Dr J Anderson	Dr S Maheson
Dr T Anderson	Dr H Mahdi
Dr N Barnes	Dr P McCartney
Dr G Barry	Dr B McKenzie
Dr G Bayliss	Dr I Millar
Dr R Capps	Dr R Moffitt
Dr P Chapman-Smith	Dr J Monigatti
Dr J Chuah	Dr W Murtha
Dr N Cooper	Dr J Orton
Dr D Davies	Dr M Osborne
Dr M Davis	Dr H Oxer
Dr G Dawson	Dr A G Robertson
Dr C Dillon	Dr P Robinson
Dr C Edmonds	Dr R Schedlich
Dr M Fraundorfer	Dr A Slark
Dr A Gebbie	Dr D Smart
Dr J Gilligan	Dr R Stevens
Dr D Gorman	Dr C Strack
Dr R Gray	Dr P Sullivan
Dr D Griffiths	Dr A Sutherland
Dr J How	Dr R Thomas
Dr C Kenny	Dr B Turner
Dr D Kerr	Dr D Tuxen
Dr J Knight	Dr I Unsworth
Dr P Laverick	Dr A Veale
Dr G Lovell	Dr R Webb
Dr C Lowry	Dr J Williamson
Dr M Loxton	Dr R Wong
	Dr T Wong

BOARD OF CENSORS

Following the election of the present committee the Board of Censors has new membership. It now consists of the President, (Dr Des Gorman), the Education Officer, (Dr David Davies), and the Director of an Hyperbaric Medical Unit (Dr John Williamson).

NOTICE OF INTENTION TO ALTER THE RULES OF THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY INCORPORATED

The Executive Committee of SPUMS intends to move two motions to alter the rules of the Society at the 1991 Annual General Meeting. The motions will then have to be passed by a three fourths majority of the full members and life members in a postal ballot. This rather cumbersome arrangement is necessary because the Corporate Affairs Office, Victoria, requires motions to alter the rules to be presented to a meeting.

The motions seek to add an Education Officer to the Committee and to enable members resident outside the South Pacific, specifically North America, to form a local branch of SPUMS.

Motion 1.

“That rule 22 (a) be altered by inserting the words “an Education Officer,” after the words “the Editor of the Journal,” and inserting the word “the” before the words “South Pacific Underwater Medicine Society” and inserting the word “Incorporated” after these words.”

Rule 22 (a) would then read (with the added wording in **bold type**)

“The Committee shall consist of a President, Immediate Past President, a Secretary, a Treasurer, Public Officer, the Editor of the Journal, **an Education Officer**, a representative appointed by the New Zealand Chapter of **the** South Pacific Underwater Medicine Society **Incorporated** and three other members of the Association entitled to vote.”

Motion 2.

“That rule 41 (b) be altered by deleting the words “in the South Pacific area”.”

Rule 41 (b) would then read

“A regional branch of the Association may be established at any place to further the objects of the Association in that place.”

NEW ZEALAND CHAPTER OF SPUMS CONFERENCE AND AGM Friday April 12th to Sunday April 14th 1991 Whitianga and the Mercury Islands

There is a wide range of accommodation available and plenty of alternative activities, sailing, fishing, golf and sightseeing. The cost is expected to be about NZ\$ 475.00 for accommodation, meals and diving.

The conference organisers are Dr E.Allen, 6 Pacific Drive, Tairua and Dr C.McAllum, “Denbora”, Te Tiki Street, Coromandel, to whom enquiries should be addressed.

ORIGINAL PAPERS

PROVISIONAL REPORT ON AUSTRALIAN DIVING-RELATED DEATHS IN 1988

Douglas Walker

Summary

There were nine diving-related fatalities identified in this year, two being breathhold divers, four using scuba, and three with hookah air supply. In each tragedy there may be found some factor or factors which adversely influenced the course taken by events. One breathhold diver demonstrated the need for exhaling during ascent after taking a breath of air from a friend who was using scuba. The other died while spearfishing, the result of a post-hyperventilation blackout. The scuba divers died from a diverse series of causes, one being medical in nature, another an illustration of the danger of believing one is really an advanced diver after 9 dives, whatever a certificate may state. There is an important lesson to be found from examination of the three hookah diving fatalities, that many commercial diving operations continue to be performed in a "she'll do" manner not conducive to safety.

Case Notes

Case BH 88/1

The victim was an experienced spearfisherman who was on holiday with some friends. On this day he was with two others, one remaining in the boat while he and the third man spearfished. They remained initially within 9 m of each other so as to act as buddies but later became separated after the buddy noticed the current had moved them to an area where the water was deeper, which the victim liked but the buddy disliked. The buddy returned to shallower waters, expecting that his friend would join him but failed to check he had done so. It was 20 minutes before he realised he had not seen his friend for some time and approached the boat to find that the man left there was now similarly worried, till then having assumed the divers had been together. The body was located next day lying on the sea bed, weight belt on and a fired speargun nearby. This is a scenario typical of a post-hyperventilation blackout in a diver intent on hunting a fish. The only unusual fact was that in this incident the fish appears to have escaped the hunter's spear just before the latter lost consciousness. There was no medical reason for him to lose consciousness other than hypoxia.

EXPERIENCED SPEARFISHERMAN. SEPARATION. FOUND WITH WEIGHT BELT ON. HAD FIRED SPEARGUN. POST-HYPERVENTILATION BLACKOUT TYPE FATALITY.

Case BH 88/2

Of this trio of divers two were intending to breathhold spearfish while the other was to scuba dive. On their way to the dive they visited a dive shop and while the scuba diver was hiring scuba gear the victim bought an additional two weights. The victim was keen to try using scuba and it was agreed that he would dive down and join the scuba diver when the latter reached 4-5 m depth. There they were to share the use of the regulator. This they managed to perform successfully, and indeed ascended together buddy breathing to the surface. The victim was enthusiastic with this experience and asked to be allowed to dive using the equipment. This was denied but after they had lunched he was allowed to repeat the morning's dive-down-and-share adventure, the other friend declining an offer to try the same experience. The scuba diver advised the victim of the need to breath out when ascending before the morning dive and the successful dive must have confirmed in his mind a belief that there was no danger in this sharing.

The victim had carried a camera for the morning dive and had taken a photo of his scuba buddy before they ascended. On the afternoon dive he brought his speargun. They came together as arranged at 4.5 m depth but after five exchanges the victim saw a fish a little below them and dived after it. As he began making his ascent the buddy tried to indicate to him the need to exhale, but it is unknown whether he saw or understood these signals. The buddy thought he could see his friend reach the surface but could not see clearly because of his ascending bubbles and the somewhat poor visibility. He therefore assumed the victim was spearfishing when he was unable to see him after he surfaced.

Nobody saw the victim reach the surface, though there is no proof that he failed to reach it. The first awareness of anything untoward was when two divers returning to shore happened to look down and saw the victim lying on the sea bed, his weight belt on. It was back to front so the quick release was out of his reach, although it is not known whether it had slipped round or he had chosen to wear it thus. The body floated up after the weights were dropped. The autopsy confirmed the diagnosis of air embolism which clinical grounds suggest. Surgical emphysema was found both in the neck and mediastinum and air was noted in the aortic arch. Despite this clear evidence of pulmonary barotrauma with probable cerebral arterial gas embolism, the Coroner decided that the cause of death was "anoxia when he accidentally drowned".

BREATHHOLD DIVER SHARED SCUBA BUDDY'S REGULATOR. UNTRAINED IN USE OF SCUBA. SPEARFISHING. SOLO ASCENT. WEIGHT BELT ON. PULMONARY BAROTRAUMA. AIR EMBOLISM.

AUTOPSY EVIDENCE OF SURGICAL EMPHYSEMA IN NECK AND MEDIASTINUM PLUS AIR IN AORTA IGNORED IN CORONIAL VERDICT.

CASE SC 88/1

This case illustrates the danger which can result if a diver on completion of a course of instruction fails to realise that without the maturing effect of experience he is a novice and likely to find it difficult to translate the information recently acquired into appropriate responses to new situations. The victim and his buddy had recently completed, one after another, a basic scuba course and then an Advanced Diver course. Indeed it was probably the first unsupervised dive this victim had made and his buddy was possibly making his second such dive. But they could show the documentation (card) to confirm their being Advanced Divers, which was associated with conversation seemingly confirming their being also experienced. Unfortunately nobody thought to check this.

The two men were visiting another State and obtained an introduction to a diver there. When they hired equipment their certification was checked at the dive shop but there was no check of their experience, in particular whether they had made any dives to significant depths. Had the dive shop been running the dive it would have been routine to ask such questions but in this case, as a private person had hired the boat and boatman, there was no such questioning, though an additional hire charge was considered to be appropriate for the boat.

There were six divers in all, the others being locals and experienced divers. The two visitors were paired together for the dive, their talk of wreck diving leading the others to believe they were experienced. The wreck was broken into pieces and lying on the sea bed at 43 metres depth. Although visibility was called good the victim and his buddy were unable to find the anchor when they became low on air and decided to ascend. So they had to start an open water ascent. As was apparently their practice during the courses they had taken, the buddy came up a little ahead of the victim at this time but he was aware of the ascending bubbles which were evidence to him that the latter was close. When the buddy reached about half way to the surface he realised that he was very nearly out of air so inflated his vest and started finning to assist his ascent rate. He was in some distress when he surfaced so was very quickly got aboard the dive boat, placed in the head low position, and given oxygen. The victim failed to surface, which indicated to the others that he had died. When another boat responded to their calls for assistance the boatman and a diver from this boat dived using equipment from the second boat as the dive boat's only extra tank had been hung on the anchor line and used by the divers for their decompression stop. They soon located the victim lying on the sea bed, all equipment in place, buoyancy vest part inflated. There was no further inflation on pressing

the vest inflation button but on ditching the weight belt the body became buoyant. They allowed it to ascend while they made a precautionary decompression stop.

Autopsy confirmed that death resulted from drowning, but the presence of bubbles in the tissues led the pathologist to diagnose early decomposition changes which appears to indicate that his understanding of diving medicine was limited. The tank was shown to contain a little air but the pressure was inadequate for inflating the buoyancy vest at this depth and probably had seemed to the victim to be an out of air situation. It was shown on test of the vest in a chamber at this depth that it inflated slowly and quietly even when connected to a full tank so a wearer would not gain any rapid effective buoyancy and would readily believe there had been an inflation failure. It is reasonable to suppose that a very inexperienced diver, alone, low on air, unable inflate his vest and excessively weighted (he probably carried 15.5 kg (34 lbs), affected to some degree by nitrogen narcosis and cold, might panic, forget to drop his weight belt and perhaps hold his breath as he tried to swim up. Unconsciousness from anoxia due to exertion using his available oxygen, or from cerebral arterial gas embolism (CAGE) would cause the regulator fall from his mouth. Drowning would be very likely to occur.

JUST TRAINED "ADVANCED DIVER". FIRST UNSUPERVISED DIVE. BUDDY SAME STATUS. DEEP DIVE. CERTIFICATION CHECKED BUT NOT EXPERIENCE. UNABLE LOCATE ANCHOR LINE. LOW AIR SO OPEN SEA ASCENT. SEPARATION AT START OF ASCENT. BUDDY WAS NEAR. DEVELOPED NO-AIR STATUS WHEN HE INFLATED BUOYANCY VEST AND MADE FINNING EMERGENCY ASCENT. QUICK SURFACE RESPONSE TO MISSED DECOMPRESSION. VICTIM HAD PART INFLATED VEST. NO/LOW AIR STATUS. EXCESS WEIGHTS. FAILED TO DROP WEIGHT BELT. INFLATION OF VEST VERY SLOW AND QUIET AT DEPTH. NITROGEN NARCOSIS. GROSS INEXPERIENCE. PROBABLY PANIC. ANOXIA DUE TO EXERTION OF FINNING ASCENT POSSIBLY LED TO UNCONSCIOUSNESS AND DEATH. CAGE ALSO POSSIBLE.

Case SC 88/2

The dive shop on this resort island ran Resort Courses for visitors and organised boat dives for these new divers and those who wished to snorkel or were certificated scuba divers. This day the dive location was a bay on a nearby island. The instructor, on the boat to take the "resort diver" group, checked that all those wishing to scuba dive had been trained. They chose to dive as one group of five but shortly after descending the victim was seen to leave the group and ascend. The group leader followed him and saw him reach the boat, then he descended again and rejoined the three others and they made their planned dive. The victim, an underwater photographer carrying a camera, was an experi-

PROVISIONAL REPORT ON AUSTRALIAN

Case	Age	Training/Experience		Dive Group	Dive Purpose	Water Depth m	Incident Depth m	Weight Belt		Contents Gauge
		Victim	Buddy					On	lbs	
BH1	25	Trained Not stated Experienced	Trained Not stated Experienced	Separation Buddy	Spearfishing	18	Not stated	On	Not stated	Not applicable
BH2	16	Trained Not Stated Some experience	Trained Some experience	Separation Buddy	Recreation	6	4.5	On	Not stated	Not applicable
SC1	43	Trained 9 dives	Trained 9 dives	Separation Buddy	Recreation	43	24	On	34	Yes
SC2	52	Trained Experienced	Not stated	Not stated	Recreation	15	3	On	Not stated	Yes
SC3	26	Trained Inexperienced	Trained Inexperienced	Separation Buddy	Recreation	12	Not stated	On	Not stated	Yes
SC4	20	Trained Inexperienced	Trained Experienced	Separation Buddy	Recreation	45	13.5	On	Not stated	Yes
H 1	46	Trained Experienced	Not applicable	Solo	Work	7.5	3	On	Not stated	Not applicable
H 2	25	Trained Inexperienced	Not applicable	Solo	Work	15	15	Off	32	Not applicable
H 3	54	Trained	Not applicable	Solo	Work	24	24	On	35	Not applicable

enced scuba diver but was wearing a new backpack and his tank had come loose. To assist him the instructor gave him one of the shop units in exchange for his backpack unit and indicated where bubbles showed the location of the others. However poor visibility prevented him from finding them even after a second attempt to dive on the bubbles. He chose to continue solo and only surfaced when frustrated by malfunction of his camera. The instructor admonished him for this behaviour.

There was a second dive, in the afternoon, and on this occasion no separations occurred. When one of the group noted that he had acquired a decompression requirement according to his decompression computer the group leader buddied him with the victim and dispatched them both to make a 14 minute stop at 3 m, this to be made in open water as there was no line. The other three surfaced a short time later and were picked up by the surface cover inflatable. The

driver of this boat returned them to the dive boat and then returned to the place where he expected the remaining pair to surface after their decompression time was completed. But only one diver surfaced.

The buddy described how they had been floating close to and facing each other for 12 minutes, then the buddy turned his head to look at his gauges. When he looked up the victim had gone. He turned around to check all directions, then surfaced and looked in all directions without seeing any sign of his companion. There were waves but the man in the boat was certain that he would have seen the diver if he had been at the surface. After he had picked up the buddy and made a local surface search he took the buddy to the dive boat and returned to make a wider search. They were in a bay and he noticed something floating near the headland, this he recognised was a backpack. He drove the boat towards this object but the engine swamped when he was round the point

DIVING-RELATED FATALITIES OF 1988

Remaining Air	Vest	Equipment Check	Equipment Owner	Significant Factors
Not applicable	No	Not applicable	Own	Spearfishing, separated, post-hyperventilation blackout.
Not applicable	No	Not applicable	Own	Breathhold dived then shared buddy's scuba. Solo ascent. CAGE.
Low	Partly inflated	Satisfactory	Hired	Separation started open water ascent. Buddy no/low air "Advanced Divers".
None	Partly inflated	Some adverse	Own	Shop backpack. Decompression stop separation. Faulty contents gauge. Acute Myocarditis. CAGE?. No Inquest.
Yes	Not inflated	Satisfactory	Dive Shop	First post-course dive. Separation but continued dive. History of epileptic fit.
Low	Not inflated	Equipment lost	Own	No Inquest. Body not found. Low air. Failed buddy-breathing. Panic, became unconscious.
Had air	No	Yes	Employer	Part obstructed grid caused unsuspected strong current. Previous refusal to dive ignored.
Lost air	Not stated	Some adverse	Employer	Unsafe/unorthodox cylinder change method. Supply hose had faulty non-return valve. Backup system failed. CAGE.
Had air	Not inflated	Some adverse	Employer	Unconscious while trying to ditch equipment. Tied to water-jet gun by belt sucked into casing. No tender. No effective leader.

and his signals for assistance were only seen when he drifted back around the point and into view of the dive boat. He resumed his journey, having restarted the engine, before another boat reached him after receiving a radio call for assistance sent from the dive boat. By this time he had found that the object was the part inflated vest of the backpack and that it was supporting the victim, unconscious, in a vertical position, his head forwards and face underwater. His first action was to raise the victim's head out of the water, then he commenced giving EAR. However he was not able to pull the victim into the boat unaided. When help arrived, the victim was got aboard and taken back to the dive boat, resuscitation being continued throughout.

The victim was brought back to the resort, then flown to the mainland. There he was pronounced dead. The autopsy showed no evidence of pulmonary barotrauma or air embolism but there was histological evidence of an acute

viral myocarditis and a chronic hepatitis. The contents gauge needle was found to be loose and it showed 3 bar (45 psi) when the tank was empty. Whether he ran out of air, or was aware that this was imminent, then attempted to inflate his buoyancy vest, cannot be known. As the coroner considered there to be no need for an Inquest there was never any official discussion of the reasons for his death. It is possible that he had suffered from an air embolism, or his diseased heart may have suddenly gone into failure when stressed by the low/no air situation. The delay before the body was seen at the surface may be because it floated lower than a surfaced diver would and so remained unobserved till it floated into the calmer waters outside the bay.

EXPERIENCED DIVER. SEPARATION AT DE-COMPRESSION STOP 3 M DEPTH. DELAY BEFORE SEEN AT SURFACE. PART INFLATED BUOYANCY VEST. VERTICAL IN WATER WITH FACE SUB-

MERGED. WEIGHT BELT ON. CONTENTS GAUGE FAULTY SO PROBABLY LOW-AIR BECAME OUT-OF-AIR. NO AUTOPSY EVIDENCE OF CAGE. EVIDENCE OF ACUTE VIRAL MYOCARDITIS PLUS CHRONIC HEPATITIS. NO INQUEST.

Case SC 88/3

The diving course had been conducted from a schooner from which the two divers had made their required four dives. They had successfully completed the course despite poor visibility and now were making their first unsupervised dive. The boat was lying off a reef, unanchored because there was no current. The buddy pair of novices entered the water together but then one regulator started to free flow and when this was resolved it was found that the victim was no longer at the surface. Despite the strong advice in their course that separated divers should resurface he did not do so. The buddy was directed to where bubbles were seen breaking at the surface and dived, but was unable to locate him so returned to the boat. After 80 minutes it was realised that he must have used all his air and could no longer be considered to be diving.

A search was made but was unsuccessful. The body was found the next day lying on the sea bed, all equipment in place. A check showed he had plenty of air remaining and his buoyancy vest inflated efficiently. Information from a friend revealed the most probable reason for this fatality, that he recently suffered a fit of some type but had declined further investigation because there was this planned and desired holiday trip due.

JUST TRAINED. FIRST POST COURSE DIVE. SURFACE SEPARATION. PROBABLY HAD EPILEPTIC FIT WHILE DIVING SOLO.

Case SC 88/4

This diver was so keen to view the Barrier Reef that she took a scuba course and this was her first unsupervised dive, indeed her first dive since completing her course. The boat which took them out to the reef carried a diving instructor who checked that all those intending to scuba dive were trained, though he did not check their actual diving experience. He paired the victim to a diver who had been trained for three years, unaware she had only made 12 dives in this time. The dive platform was a pontoon and the reef was reached by following a rope down from a nearby buoy. The instructor told them that the best coral was in the shallow areas rather than below 15 m depth, but if they chose to dive deeper a decompression stop was advised.

There was some current but this was against them for their outward swim along the reef. They surfaced once to check on their position as the rope guide ceased where it reached the reef and they felt unsure of whether they were going in the correct direction. When their contents gauges

each showed 100 bar, half full, the return swim was commenced in accord with the instructor's advice. They had been swimming at 12-13 metres depth for about 10 minutes and then the victim showed her contents gauge to her buddy: it was indicating 50 bar and the buddy had very little more so they knew they should start to ascend. The buddy realised her companion was starting to ascend too rapidly and restrained her gently. However she then appeared to become agitated, let the regulator fall loose from her mouth, and signalled that she was not able to breathe. The buddy gave her her second (octopus) regulator, which she guided to her mouth but failed to purge. Her buddy offered it back, purged twice, but it was spat out and the victim then kept her mouth very firmly closed. She then began to struggle and clutched her buddy, who naturally began to panic as they began to sink deeper.

The buddy attempted to inflate the victim's buoyancy vest and drop her weight belt but failed. She managed to get free and saw that the victim had become quiet, probably unconscious, and was sinking. She became breathless from overbreathing, panicky, and drained of energy so ascended rapidly, recognising that she was in real danger herself. At first her yells and waving arms failed to attract attention, then a snorkeller on the reef noticed her and he managed to alert staff on the pontoon. It was recognised that her rapid ascent without any decompression stop placed her at risk of decompression sickness so she was given oxygen and was put in a head low position after return to the pontoon. Search of the locality was unsuccessful and this was expected as there was some outflow current and a deep channel where they had stopped to make their ascent. The body was never recovered so it is unlikely that the buoyancy vest was inflated.

The sequence of events as described is suggestive of an inexperienced diver becoming anxious in a mid water situation, believing an out-of-air situation was occurring (which could be a consequence of anxiety effecting the respiratory rhythm), followed by a flustered management of the offered regulator, leading to the final panic, aspiration of water and sudden or rapid death.

One problem which emerged from this tragedy was that of management of a diving emergency in the presence of non-divers, as occurred here. The boat which took these divers to the pontoon also carried visitors for a glass-bottomed boat view of the reef. Though these people were aware that some accident had taken place they were told nothing and mistook the attempts made by the staff to shelter them from involvement for lack of a feeling of urgency in responding. They saw the boats and planes searching but failed to experience an awareness of the intense activity around them or feel involved. This is obviously a public relations problem which requires consideration by those involved in commercial boat trips to the Barrier Reef.

TRAINED. GROSS INEXPERIENCE. FIRST

POST-COURSE DIVE. BUDDY HAD LIMITED EXPERIENCE BUT ACTED VALIANTLY. DIVE BOAT CHECK OF CERTIFICATION BUT NOT OF EXPERIENCE. FAILED TO DROP WEIGHT BELT. FAILED TO INFLATE BUOYANCY VEST. ANXIETY/PANIC AIR HUNGER SYNDROME. BODY NOT RECOVERED.

Case H 88/1

The commercial diver who died in this accident was a victim of a set of circumstances whose potential danger to divers went unrecognised till too late. Although another diver had noted the adverse diving conditions and declined to dive, there is no reason to believe he anticipated the true degree of danger posed by this job, the clearing of a trash grid in a concrete canal carrying the water supply into a power plant. This task was usually performed by a machine but it was out of action and being repaired. Failure of communication between diving supervisor and works engineer was a critical factor in the chain of events.

The diver entered the water from a ladder a little upstream of the grid, wearing hookah, and descended. A short time later, when his tender realised there was no response to line calls, there was a sudden realisation that all was not well. The second diver then kitted up and entered the water, almost immediately finding he was torn from the ladder and onto the victim on the grid. It was with difficulty that he was able to regain the surface after the water flow had been stopped. The water had forced the victim so rapidly onto the grid he would have been stunned, and even had he retained his regulator the water power would have prevented him making any respiratory excursion. The second diver was fortunate to survive.

Although the speed of the water flow was far greater than on other occasions this diving company had worked here, there was a more important and less obvious factor involved. Because of the build up of weed on the grid the channel was reduced to a small available cross section at the grid. This greatly increased the power and flow rate in the immediate vicinity of the clear part of the grid while the flow was slow and weak in front of the remainder of the grid. The diver was suddenly caught in a powerful (and unexpected) current and flung onto the grid.

This tragedy, however, arose in the first instance out of a failure to follow recognised safe diving procedures, probably in an effort to please the client. Although the water flow should have been stopped before the divers entered the channel it appears that this would have required taking two generators out of action at a time when others were not ready to take over. The consequent loss of power generation would have adverse consequences on the public conception of the Authority involved, and the diving company would lose a powerful client. Such thoughts would occur to those making decisions on the spot even if not actually spoken or discussed.

SOLO. HOOKAH. PARTLY OBSTRUCTED GRID NARROWED EFFECTIVE WATER CHANNEL SO THERE WAS A RAPID AND POWERFUL CURRENT. DIVER FORCED AGAINST GRID. RESCUE DIVER ALSO AFFECTED BUT SURVIVED. FAILURE STOP WATER FLOW BEFORE ENTRY. POSSIBLE REASONS FOR BREACHES OF SAFE DIVING PRACTICES.

Case H 88/2

The circumstances of this tragedy may appear to many as beyond belief in a world where commercial diving is apparently subject to over cautious regulations overseen by strict Government Inspectors much more interested in the wording than the spirit of such regulations. The victim was a newcomer to commercial diving, an experienced scuba diver (49 dives) who has never undertaken a hookah dive till he joined this firm two weeks before. Since then he had dived three times using hookah to familiarise himself with an unusual method they employed when changing to a new air cylinder. When the diver was warned there was to be a change over he turned on his bail-out bottle and used this while the empty cylinder was removed and the fresh one connected.

The job was to survey an old pipe, marking its course at intervals with floats. The dive boat was 1 km from shore, the water depth was 15 metres. The victim swam to the float which indicated how far the previous diver had travelled, then descended. His rate of descent was slow as he had difficulty equalising his ears, this being known because he was wearing a Kirby-Morgan band mask which had direct communication with the surface. He appeared to be calm and normal when advised that a cylinder change was to take place. Delay occurred after the cylinder was turned off and the line had been vented, disconnection requiring the use of a shifting spanner before this was accomplished and the fresh cylinder connected. It was a surprise to everyone when the victim was seen to surface as there was no warning of his intention to do so. His mask was off, held in his hands. He appeared to say "I thought I was dead", then lost consciousness and floated on his back at the surface. He may have been wearing his weight belt when he surfaced and dropped it as he lost consciousness.

He was brought onto the dive boat and everyone there realised that he had suffered an air embolism. He was placed in a left lateral head-down position, oxygen commenced, and a radio call made to shore for an ambulance to meet their arrival. He appeared to have difficulty with breathing, it being rasping and irregular, his pulse was weak and rapid, and jaw was clamped tightly shut. He then started fitting, was agitated and thrashed about so that they were unable to maintain him in the approved lateral position. The ambulance took him to the local hospital and there there was some discussion as to who should transport him to which centre capable of providing recompression treatment. It was decided to transport him by air and before he left the hospital

for the airport he was apparently sufficiently recovered to understand and answer simple questions. However his condition deteriorated again and he had to be taken to a hospital on the route. There he was put onto a drip of dextran and a transportable recompression chamber reached him. It was now 4 1/2 hours from the time he surfaced. Although he did reach the hyperbaric treatment unit alive he died four days later from the cerebral damage he had suffered.

It is not known why the victim failed to communicate his intention to ascend but it is reasonable to suggest that this was because he was making an instinctive response to an emergency situation, tearing the mask off his face indicating his feeling of being suffocated. Examination of the equipment showed it was very possible that the non-return valve in the air hose might not have worked reliably. The bail-out bottle was shown to contain air and to be turned on. It was suggested that when the air lines vented, prior to removing the in-use cylinder, the faulty non-return valve allowed the venting of his mask to the surface and the air supply from the get-home tank may have been insufficient to maintain air in his mask. In a diver grossly inexperienced with hookah diving, as this man was, the loss of mask air (not merely cessation of the air supply) would be a panic creating situation. It would require any diver to make an emergency ascent in order to survive.

There was no legal requirement for any recompression chamber to be on site, and though there was a chamber ashore which could have been used it would have taken several hours to make it ready for use. It is uncertain whether there was anyone available with training in its use. Naturally the delay before commencing a recompression of the victim greatly reduced his survival chances, which were probably minimal from the time he surfaced.

SCUBA EXPERIENCED. UNTRAINED (FOURTH USE OF HOOKAH) AS "COMMERCIAL DIVER". UNUSUAL TOPSIDE CYLINDER CHANGE METHOD REQUIRED USE OF BAIL-OUT BOTTLE. POSSIBLE FAILURE OF NON-RETURN VALVE IN AIR HOSE CAUSED VENTING OF KIRBY-MORGAN MASK TO SURFACE. EMERGENCY PANIC ASCENT. PLACED IN TRANSPORTABLE RCC 4 1/2 HOURS AFTER HE SURFACED. LOCAL RCC NOT AVAILABLE FOR IMMEDIATE USE. CEREBRAL ARTERIAL GAS EMBOLISM. DEATH DELAYED 4 DAYS.

Case H 88/3

The task was to remove the concrete cladding round a pipe to enable it to be inspected. There were four divers working from the dive boat, a democratic group who shared tasks, the diving supervisor taking his turn with the rest to dive and none of them formally acting as tender or backup diver. Indeed each was quite free to decide whether to make a decompression stop or not. There was one other person aboard, the mechanic whose responsibility was the pump

supplying the water jet gun.

The inlet of the water pump became blocked so it had to be put on idle while this was cleared. In order to utilise the diver's available no-decompression dive time fully the supervisor decided to bring up the diver until the water-jet was functioning again. It was at this time that it was noticed that there were no bubbles ascending, and a line call went unanswered. The air supply had not failed as the air compressor was separate from the water-jet pump. The supervisor descended to discover what had happened, choosing to use the second air compressor. He found the diver was lying on the sea bed with his equipment partly ditched. The mouth piece of the regulator was out of his mouth (he was not using any retaining strap) and no air was escaping. The rescuer tried first to bring the victim up by orally inflating his vest (no reason is given for not attempting to use the hose supply inflation system) but this failed. He then noticed that the heavy water jet gun lay across the diver's thigh and was retained attached to the body by the fact that the buoyancy vest belt had been sucked into the jet gun's retro nozzle casing. After it was cut free the body floated up to the surface. Other divers had noticed the annoying habit of loose portions of equipment to be sucked into the casing but this had been treated as a nuisance, not a danger.

There are several unanswered questions in connection with this fatality. The air supply for the diver was said to have been uninterrupted so there was no reason for him to attempt this ditching of equipment. He was an experienced diver who had a bail out bottle and buoyancy vest, so there is no obvious reason why he could not have calmly cut himself free. Possibly he drowned while trying to recover his regulator mouthpiece, suffering sudden groin pain when stretching for it. This could occur because he had been jumping into the water from the upper part of the boat during the mid day meal break and had suffered some groin pain following one of his water entries. The mid day break served two purposes, being a surface interval period between the morning and afternoon dives and also when the tide change made the visibility very low. It is not known why he attempted to ditch his equipment rather than cut the strap connecting him to the jet-gun. He was experienced in the ditch and recovery of equipment because he was a well known local diving instructor. Indeed his knife was used to cut the strap.

Resuscitation attempts were unavailing: very probably he was dead when located. None of the three divers knew about EAR but the engineer who controlled the water pump was trained and he performed the resuscitation attempt. Death was due to drowning.

EXPERIENCED. UNREGISTERED COMMERCIAL DIVER. ATTEMPTED TO DITCH BACK PACK BEFORE HE DIED. NO DIVE TENDER. FAILURE TO DEFINE TOPSIDE DUTIES. FAILED TO DROP WEIGHT BELT. FAILED TO INFLATE BUOYANCY VEST. VEST

STRAP SUCKED INTO WATER JET-GUN'S CASING.
NO REPORTED DEFICIENCY OF AIR SUPPLY. DI-
VERS IGNORANT OF RESUSCITATION METHODS.

Discussion

The death of an experienced breathhold spear fisherman is commonly a consequence of pre-dive hyperventilation and this is the almost certain reason for the death of the diver in the first case described. The second death was unusual as the practice here described of a breathhold diver descending to join a scuba diver and then buddy breathing is probably rare nowadays. Although this tragic example of the reason for exhaling during ascent after the breathing of air at a higher ambient pressure is both instructive and predictable it is apparent that the scuba diver had failed to adequately appreciate the potential danger of the sharing his air with a breath hold diver.

There were a number of critical factors which had an influence on the scuba diver fatalities, with the most significant one being the factor of inexperience. Any just-certificated diver who thinks her or she is more than in a novice category has obviously been inadequately instructed in their skill status, and the use of the term "Advanced Diver" for someone who has performed only nine dives, all under supervision, should cease immediately. This is in part because such people may well believe they have acquired a diving ability rather than a diving certification. Possibly there should be a probationary period of a minimal number of supervised and logged dives before this certification is confirmed. There is also a warning to those who operate dive trips that it is wise to make a routine check of the experience as well as certification when signing up clients for dive trips.

An examination of Case SC 88/2 shows several factors worth consideration. The faulty contents gauge (probably owned by the victim but this is not directly stated) played a part in this incident if the victim was unaware that it was unreliable, however it was not stated whether the gauge was likely to have given some reading when greater pressure was in the line or whether it would have been obvious that it was faulty. Special significance should be paid to the basic reason for this fatality, the need for one of the group to make a decompression stop. That an experienced diver should accumulate a decompression requirement of 14 minutes taken at 3 m depth in open water and the matter be treated as not being noteworthy may indicate an attitude to decompression safety which is leading to so many cases for the hyperbaric units to treat. In this case visibility was poor and the divers were neither holding onto a line or onto each other. They were at double jeopardy, from separation (which occurred) and failure to maintain a correct and constant depth during the decompression stop. It is probable that the victim, who did not require the decompression stop, became very short of air and ascended rapidly, using the last of his air in an attempt to inflate his buoyancy

vest. The partially inflated vest unfortunately allowed his head to fall forwards and be submerged. This is a failing of modern buoyancy control devices (ADV's) which have little buoyancy in front on the surface and do not tilt unconscious divers onto their backs. The described events support the suggestion that he suffered from a cerebral air embolism during his 10 feet ascent rather than the postulated cardiac attack.

Two fatalities occurred in divers making their first dive following completion of their basic scuba training. One case involved an epileptic who possibly declined to accept that he had such a condition and required investigation and medication. He ignored the advice given to dive with a buddy and surface if separated. There was therefore nobody near to rescue him when he (it is assumed) suffered a fit. A similar event is known which ended more happily because of the presence of an instructor. Epileptics should not dive. In the other case there was panic which the buddy attempted valiantly to reduce, placing herself at real risk in the process.

The remarkable fact concerning the commercial diving fatalities is that in these seemingly over-regulated days so many ill managed diving operations continue. This is more a matter for education than regulation, though the latter response is far more likely. Reading the incident resumes will indicate which critical adverse factors were most important in each case. In the first of the cases described there was a failure to insist that the water flow be stopped, or at least monitored, before the diver entered the channel. The significance of another diver's refusal to dive here was not appreciated by the engineers and possibly unknown to this diving company. The "Can Do" tradition of commercial divers must surely be given some of the blame for this fatality.

The power of flowing water is easy to underestimate, or fail to consider in advance. This is particularly important in calm or slow moving water where there is a restricted outlet such as a pipe. In this case the partial obstruction of the grid was a cause of the channel suffering sudden reduction in cross section. This was not visible from the surface appearance of the water and neither diver was aware of this till in front of the unobstructed portion of the rubbish grid, when the flow was irresistible.

While a change over from one air cylinder to another while the diver is underwater is an entirely reasonable practice, a method which requires venting of the supply lines and a removal of one cylinder before a fresh one can be connected up is unusual and unsafe. That such a method had been apparently followed for some time without significant misadventure gave no guarantee that it was safe, and this victim's gross inexperience proved too great an additional factor when the non-return valve failed while the air line was venting to the surface. The air supply rate from his get-home bottle was insufficient to offset this venting so he made an out-of-air ascent, unfortunately suffering air embolism.

The third case is hard to understand because in this

incident the diver was experienced in ditch-and-recovery from his work as a scuba instructor. If the evidence is correct that there was no failure of his air supply, there must have been another, not identified, reason for his actions. While there was entrapment of his buoyancy vest's belt in the water jet-gun this machine had been turned off about the time he died. Possibly he misjudged the danger of his situation and inhaled water while he was attempting to ditch his equipment.

Acknowledgements

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Project Stickybeak Divedata Databank

The objective of this on-going project is to identify factors which influence the safety of divers, whether recreational or commercial divers. Reports are requested concerning incidents of all types and severity, particularly where there has been a successful outcome. MEDICAL CONFIDENTIALITY is given to every communication received. Address for correspondence:-

Dr D Walker
P O Box 120, Narrabeen
New South Wales 2101
Australia.

DEVELOPMENT AND USE OF THE OXYGEN-BREATHING MINIMAL-RECOMPRESSION TREATMENT OF DECOMPRESSION SICKNESS

Geoffrey Gordon

Introduction

When man ventures into a hyperbaric or hypobaric environment, his excursions are occasionally complicated by the development of decompression sickness (DCS) and arterial gas embolism (AGE). To treat the occurrence of these illnesses, recompression therapy is used either according to a standard treatment table or to a depth that brings relief. This approach to treatment is not based on experimental evidence, but on the empirical application of theoretical concepts.^{1,2} Old protocols were superseded when it was felt that the results were unsatisfactory, rather than when case analysis indicated poor treatment outcomes.

Analysis of the effectiveness of treatment regimens needs to be conducted if, in any meaningful way, we are going to be able to improve our effective treatment opinions. That a solution will be developed to benefit every case is a naive idea, but have the current "minimal recompression oxygen breathing tables", developed in 1965, filled a void, or are they just another attempt at treating a disease process that is still incompletely understood?

The history of treatment tables

It was not until 1847 that Pol and Wattle³ first recognised that there was a relationship between the onset of DCS symptoms and the depth, bottom time and rate of ascent. This was 28 years after Siebe developed the first practical deep-sea diving outfit and 6 years after the production of the first large capacity compressors that permitted large numbers of men to work at raised ambient pressures. Although this work established recompression as the primary treatment modality, the manner of its application was unclear. It was not until 1878 that Bert⁴ demonstrated that liberation of nitrogen in the form of bubbles was the cause of DCS. He also recommended recompression and went on to expound that treatment with oxygen should be effective. No decompression rates were specified, and as pure oxygen was both scarce and very expensive it was little used. In 1897 Zuntz⁵ utilised oxygen in conjunction with recompression to increase the gradient for nitrogen elimination and hasten bubble resolution. However, due to the development of oxygen toxicity, the use of this adjunct proved unpopular and was not widely used again for many years. Air recompression therapy for DCS was subsequently developed.

In 1937 Behnke and Shaw⁶ re-investigated the use of oxygen in the treatment of DCS. They hoped to utilise the increased gradient for nitrogen elimination to improve treatment outcomes. In experiments using a dog DCS model, they observed that severe cardiopulmonary DCS responded well to recompression to 30 msw regardless of whether air or oxygen was breathed. On subsequent decompression however, those treated on air had recurrences of cardiopulmonary DCS of pretreatment severity. Those treated with oxygen showed a minimal return of symptoms, indicating better inert gas clearance when an oxygen atmosphere was breathed. Yarbrough and Behnke⁷ two years later, documented a 50% recurrence of symptoms in divers treated by recompression to depth of relief plus 10 metres of seawater, the procedures published in the US Navy (USN) Diving Manual of 1924. This protocol probably did not achieve resolution of all the gas in bubble form despite the pressure applied. In an attempt to achieve complete bubble elimination, they empirically developed guidelines limiting recompression depth to 50 msw with a minimum time at this depth of 30 minutes. In modifying the Haldanian type decompression, 100% oxygen was breathed from 18 msw to the surface. The process of gas diffusion from bubbles and tissues was thought to be slow, and so administration of

oxygen at increased pressure was used to accelerate this process. Initial results were encouraging, and attributed to the elimination of gas mixtures during the ascent from 18 msw.

By 1945 the performance of these new treatment tables "appeared" no better than the results obtained using the US Navy 1924 protocol. As a result of this "apparent" failure, a series of experiments conducted by Van der Aue et al.⁸ led to the development of formal treatment tables embodying the Yarbrough and Behnke principles. These tables were subsequently promulgated as USN Tables 1, 2, 3 and 4 in the USN Diving Manual of 1945 and remained in use becoming standard in the USN, several foreign Navies and many commercial worldwide for the next 20 years.² It is interesting to note that these widely accepted treatment procedures were based upon a study involving only 33 subjects and that some of the individual treatment tables were based on as few as 6 subjects. The Royal Navy (RN) developed their equivalents in the RN Tables 52, 53, 54 and 55 a few years later.

Problems with the current air treatment tables

In 1947, Van der Aue and his colleagues analysed 113 cases treated with these "new" air tables and found a first treatment failure rate of 5.3%.⁹ It was clear that these air treatment tables showed promise. Unfortunately these tables were not subjected to further outcome analysis until 1962 when Slark¹⁰ retrospectively reviewed the treatment of 137 cases. In this study the overall first treatment failure rate had risen to 21.5%. Slark considered this unacceptable, and further pondered on the likelihood that the nitrogen uptake occurring during treatment contributed to the observed failure rate. He did see something positive in the air tables however, postulating that the prolonged 9 msw soak was the strength of USN tables 3 and 4. In recommending the development of new tables, Slark foresaw these having a longer time at lower pressures, and further suggested incorporating an even release of pressure during the ascent.

In 1964 Rivera¹¹ reviewed the treatment of 935 cases of DCS, spanning the period 1947 to 1963. The failure rate of tables 1 and 2 during initial treatment was 5.6% and considered satisfactory. These two tables were used to treat pain only DCS. The treatment of serious DCS with tables 3 and 4 had a failure rate during initial treatment of 25%. This overall figure is perhaps a little misleading as the failure rate rose dramatically over the review period, from 16.1% in 1947 to 46% in 1963. This increase was paralleled by an escalation in the number of civilian divers being treated and a concomitant increase in divers presenting following marked departures from accepted diving practice. In 1963, some 66% of the divers treated had developed DCS following such non-standard dives, and this sub-group accounted for almost all of the 46% initial treatment failures. There were no failures using USN tables 3 and 4 on Navy divers, who

invariably received early recompression, unlike the civilian divers, who usually presented following long delays.

Goodman and Workman¹² reviewed the cases treated with air tables during 1964, and noted a similar overall failure rate of 25%, with USN tables 3 and 4 again having an initial failure rate of 47%. This group similarly comprised civilian divers with long delays to treatment following non-standard exposures with omitted decompression.

The construction industry has traditionally utilised air recompression in cases of DCS occurring at the workplace, and have developed their own code of practice. Analysis of the effectiveness of these procedures in the various large projects has not been undertaken in this review, but air recompression has been used in two recent large projects in Hong Kong and Singapore with acceptable results.¹³

These figures, and the increasing number of civilian casualties, necessitated a fresh look at the problem of treatment DCS and the development of more effective recompression tables for what promised to be an epidemic of DCS in the 1970s.

Development of the minimal recompression oxygen tables

Goodman¹² in 1964 defined what he felt to be the fundamental route to DCS symptom remission viz:-

- 1 Compression to reduce bubble volume and radius in order that the intensity of the tissue reaction be reduced.
- 2 Termination of the focal ischaemia brought about by the endothelial irritation.

The latter, he surmised, could best be achieved by exploiting the collateral blood supply after hyper-oxygenating the patient, effectively establishing a metabolic detour around the occluded vessels. Hyper-oxygenation would also provide the maximum gradient for the elimination of inert gas from within the bubbles and tissues. Compression has a limited ability to RESOLVE bubbles, so bubble compression was relegated to a position of less than first order significance. The risk of developing DCS from treatment was also essentially eliminated with oxygen therapy.¹²

Goodman and Workman, from the USN Experimental Diving Unit, were given the task of revising the treatment of DCS, and in late 1965 their landmark report¹⁴ was released.

Schedule development

Noting, as did Slark¹⁰, that the 9 msw soak was probably the strength of the USN tables 3 and 4, Goodman and Workman conducted a number of trials at this shallower depth. The first provisional format was empirical in its design. Patients were taken to 10 msw breathing 100% oxygen and assessed after 10 minutes. If all symptoms had been relieved, then treatment was continued at this depth as indicated by the dotted line in Figure 1. If relief was not complete in this time, then the patient was taken to 18 msw and decompressed according to the 18 msw schedule shown in Figure 1.

In a few instances, the decompression from 18 msw was interrupted by spending 30 minutes at 9 msw before continuing the decompression to the surface. There were no failures with this provisional format.

Following analysis of the cases so treated, patients were subsequently compressed directly to 18 msw, eliminating the 10 msw trial. This resulted in treatment times of between 100 and 130 minutes.

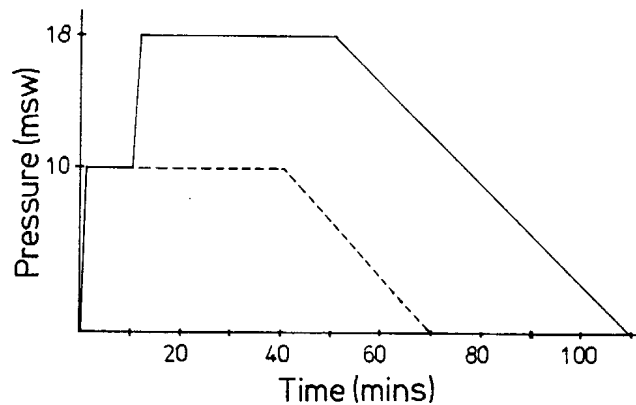
Retrospective analysis showed that both the full treatment depth and the actual time breathing oxygen, were significantly related to treatment adequacy, leading Goodman and Workman to define a Minimum Adequate Time of 30 minutes oxygen breathing at 18 msw with a total time breathing oxygen of 90 minutes. New schedules were developed reflecting treatment durations of 1.5 and 3 times this minimum adequate time and designated the 2nd provisional format (Figure 2). If relief occurred within 10 minutes at 18 msw, 130 minutes of oxygen breathing was administered (Figure 2 upper). If relief was not complete however, then 285 minutes was spent on oxygen (Figure 2 lower).

This second format also proved very effective, but was further refined to reduce the risk of acute oxygen toxicity by the inclusion of air breaks interrupting the periods of oxygen breathing. The resulting tables are the familiar USN tables 5 and 6 (RN tables 61 and 62).

In the cases receiving adequate treatment, 50 cases in all, there was a 2% failure of initial recompression which compared favourably with the overall air tables initial failure rate of 15%. Of these 50 cases 28 (56%) met the criteria for treatment with USN tables 3 or 4 (serious cases) and these had an initial failure rate of 3.6%, a marked improvement over tables 3 and 4, which historically accounted for 27% of all treatments and had a failure rate of up to 47%. Better results were thus achieved with sicker divers. Oxygen toxicity was not a problem as it had been with Bert in 1878.

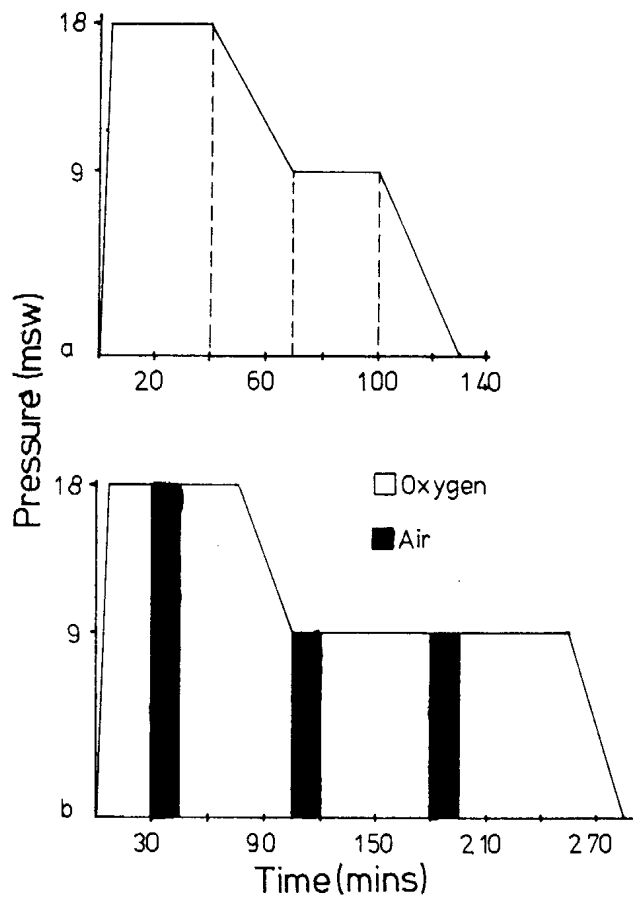
In attempting to convince physicians that deeper was not always better they noted that the law of diminishing returns becomes relevant for bubble diameter before it does so for bubble volume (Figure 3). Thus in going from 20 msw to 50 msw little is achieved in diameter reduction, but there is an obligation of additional inert gas uptake as gas mixtures

FIGURE 1



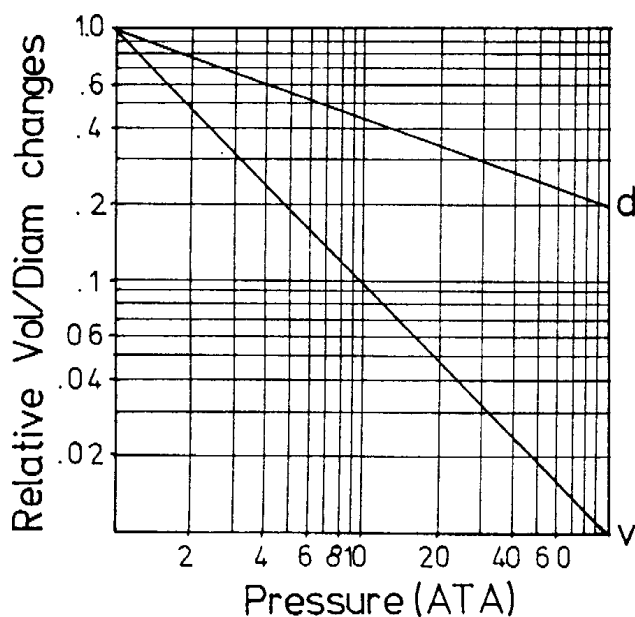
First Provisional Format
(Drawn from data by Goodman and Workman¹⁴)

FIGURE 2



Second Provisional Format
(Drawn from data by Goodman and Workman¹⁴)

FIGURE 3



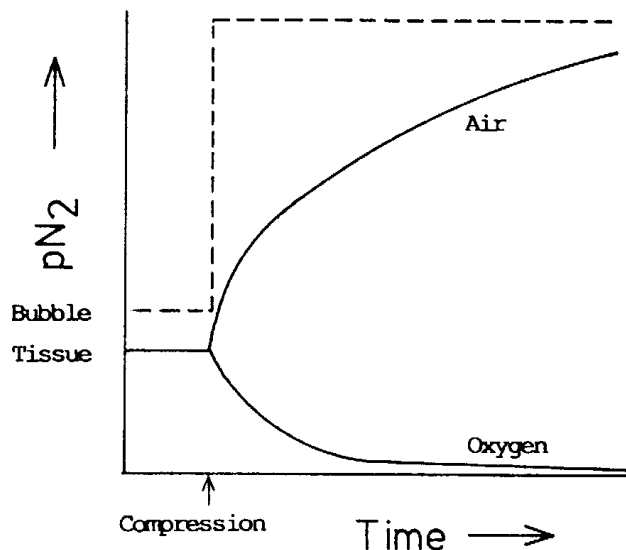
Volume and diameter changes under varying overpressures

must be breathed below 20 msw.

The question as to whether the treatment failures occurring after recompression on the air tables was due to a relapse of DCS or whether they represented freshly provoked DCS due to additional inert gas uptake, has never been satisfactorily answered. What is known though, is that the gradient for inert gas (nitrogen) elimination while the patient is breathing oxygen at the maximum safe pressure is large. At 18 msw the partial pressure (pp) of nitrogen in the bubble is approximately 2,105 mm Hg while the pp of nitrogen in the blood of a patient breathing 100% oxygen will be 0 mm Hg, creating a massive gradient for nitrogen egress and avoiding the potential problems of bubble growth due to additional inert gas uptake. Breathing 100% oxygen results in a nitrogen elimination curve with the partial pressure gradient of nitrogen between the tissues and the bubbles increasing with time as the oxygen is breathed, driving the nitrogen out of the bubbles, reducing their size until they collapse. In contrast, during treatment with air, the gradient for nitrogen elimination decreases with time and there is additional nitrogen uptake, nitrogen that sooner or later must be off-loaded (Figure 4).

Goodman and Workman¹⁴ saw benefit in the even release of pressure during decompression, rather than the staged ascent of the air tables. This avoidance of sudden pressure reductions was seen to be an important adjunct and had been previously recommended.¹⁰ The time savings of the oxygen tables were of major significance. A USN table 6 (RN table 62) takes 4 hours 45 minutes to complete, while

FIGURE 4



Effect of compression on the pN₂ gradient from a bubble using air and 100% oxygen (Redrawn from Pilmanis²⁷)

a USN table 4 takes 38 hours 11 minutes. This time saving, combined with a better treatment efficacy, had considerable equipment and personnel advantages, changing what was often a marathon treatment effort into a tolerable and effective therapy.

These new tables met with instant favour when first released in November 1965. But have they stood the test of time, and are they as effective as the first 50 cases treated would suggest?

Effectiveness of the oxygen tables

Following the inclusion of these treatment tables into the USN Diving Manual in 1967, Workman¹⁵ reported on his experience of using these in 150 cases, 40 of whom were civilian divers. First treatment failures in this series was 15%, falling to 5% after the second treatment. In the military divers, initial failure was 7%. Consistently good treatment results were documented and at a reduced time obligation and staff commitment. Workman did concede that these tables would not benefit 100% of divers, especially in view of the increasingly inadequate decompression seen in the civilian divers developing DCS. However, his initial treatment relief with the oxygen tables of 85%, compared with 55% on USN tables 3 and 4, was indeed encouraging.

Three divers not improving at 18 msw in this series, were further compressed to 50 msw with no additional benefit.¹⁵ Bornman¹⁶ in the same year stated that although an increase in pressure could relieve the pain associated with mechanical pressure and ischaemic obstruction, pressure per se would not aid bubble elimination. He concluded that

the use of oxygen in the new tables was very important.

Lambertson¹⁷ in noting the initial success with these new tables, concluded that extension of oxygen tolerance at pressure would provide the ultimate improvement in the therapy of DCS. His initial studies showed that the total tolerated oxygen dose at 20 msw could be extended to 20 hours by periodic respite from exposure to these toxic oxygen levels.

In other series, Erde and Edmonds¹⁸ noted an initial treatment failure of 13-15% if the delay was greater than 3 hours while Davis¹⁹ noted a 10% failure rate, many of these occurring following considerable delays before treatment. Melamed and Ohry²⁰ in 35 patients documented a 10% failure of initial therapy, while Hunt²¹ recorded 100% success in the initial treatment of 18 divers (78% civilian) with DCS, the best figures achieved in any study. Bornman's²² experience in treating Navy divers was a 3.6% failure following the institution of early recompression.

Kizer²³ reviewed 50 cases of DCS treated following delays in excess of 12 hours, again most of these divers having marked departures from accepted diving practice. Forty-seven of these patients were treated with USN table 6 (RN 62) half of these treatments being extended. The standard table had an initial failure rate of 20%. In the severe cases, when the table was extended, the failure rate rose to 37%. Overall though, Kizer felt that 92% had complete or substantial recovery following initial treatment. These results are similar to those achieved in the other series, but Kizer noted that the oxygen dose frequently needed to be increased to achieve this result.

Green and Leitch²⁴, reviewing 20 years' experience analysed 179 cases with severe DCS. Fifty-six (30%) were treated with air tables and 123 (70%) with the short oxygen tables. Overall, the oxygen tables performed better, but particularly if the delay to treatment was greater than 12 hours. Some cases presented at between 10 and 17 days after symptom onset and made full recoveries following treatment (Table 1). The USN¹⁶ found the oxygen tables to be superior if the delay to treatment exceeded as little as 5 hours. Looking at the results achieved with the oxygen tables as a separate group, RN table 61 (USN 5) had an 18% relapse rate following initial treatment, while the RN table 62 (USN 6) had a relapse rate of 3%.²⁴ Green and Leitch²⁴ restated that it is inappropriate to treat severe cases on an RN table 61, as this treatment proved ineffective in severe cases and had a high relapse rate. With the oxygen tables firmly established as the mainstay of treatment, they concluded that grounds exist for the removal of the RN table 61 (USN 5) as a treatment option, relying instead on the longer oxygen table. This would reduce treatment errors and improve outcomes, a feeling that has been expressed by others.²⁵

The same authors, but in a different study²⁶, reviewed the treatment of severe DCS that was not responding at 18 msw. In the 24 cases, almost universal ineffectiveness was

TABLE 1

PER CENTAGE CURE RATES

	< 12 hrs delay	> 12 hrs delay	Overall
Air Tables	50%	7%	32%
Oxygen Tables	58%	58%	46%

(from data by Green and Leitch²⁴)

documented following further compression, usually to 50 msw. Those showing some response had already demonstrated some improvement at 18 msw. They concluded that serious cases of DCS are not materially improved by further compression to a greater depth. Further, 6 cases still deteriorating at 50 msw were no better at 70 msw, reinforcing the belief that compression to a greater depth does not necessarily halt or prevent deterioration. The animal data also supports the doubtful efficacy of increasing the pressure to greater than 18 msw in the treatment of serious DCS.^{1,27}

While most commonly seen in divers and caisson workers, DCS also occurs in aviators during their exposure to sub-atmospheric pressure. In a review of 145 cases of aviators bends, Davis et al²⁸ documented a recurrence rate of 22% in those treated with the air tables, while in contrast, only 1% of those treated with the oxygen tables relapsed. The USAF used modified tables 5 and 6, in that they continued the 20 min oxygen/5 min air sequence at 9 msw, rather than the 60 min oxygen/15 min air as promulgated in the USN tables for this depth. The total time on oxygen remained unaltered. As with diver DCS, the treatment of stricken aviators with the oxygen tables was very successful, even following delays of many hours.

Studies have suggested that treatment at 18 msw for AGE may also be effective especially if a concomitant decompression debt had been incurred. Traditionally, patients at 18 msw on oxygen not showing improvement have been further compressed to 50 msw on air. This makes it difficult to make a true assessment as the effectiveness of RN 62/USN 6 in treating AGE. From the data^{2,15,21,29} it is not possible to say that 50 msw is better than 18 msw. It seems likely that most cases of AGE will do equally as well at 18 msw on oxygen as they will at 50 msw on air. If early recovery does not occur, time seems to be the major factor, regardless of the pressure at which treatment occurs, hence extension at 18 msw is indicated. In the studies reviewed, USN 6A/RN 63 showed the best performance. However following a delay before treatment of greater than 6 hours, proceeding to 50 msw was no more effective than staying at 18 msw. One omission that does stand out is that maximum use was not made of extending the tables at 18 msw as is

permitted.^{30,31}

Conclusion

Human beings did not evolve for a marine existence, but they continue to venture into this and other alien realms with attendant morbidity and mortality. Before the 1950s, the divers presenting for treatment of compressed air illness were primarily military or commercial. They presented early and with predominantly "pain only" DCS. This has been entirely reversed since the late 1960s by the large number of less disciplined and unsupervised sport divers who not only present later, but have a preponderance of the more sinister neurological manifestations of DCS.^{11,15,24,26} The treatment protocols used to treat this civilian population in the 1980s have been satisfactory, but it must be realised that they were designed in the military for a totally different population.

Because the therapeutic value of compression therapy is so self evident controlled studies in man have not been done. Davis in 1935 further commended that "No one who has seen the victim of compressed air illness, gravely ill or unconscious, put back into a chamber and brought back to life by the application of air pressure, will forget the extraordinary efficiency of recompression or will be backward in applying it to a subsequent case of illness".³² In contrast to this, Saumarez and his colleagues³³, who were without recompression facilities, successfully treated a case of severe neurological DCS with oxygen at the surface.

Pressure and oxygen have become the cornerstones of therapy and studies have been directed at identifying the optimum pressure, oxygen dose and time that these variables should be applied to achieve maximum effectiveness. Some centres believe that pressure is the all important variable, but the Boyle's Law effect is not nearly as dramatic when viewed from the standpoint of the bubble as opposed to the more usual bubble volume (Figure 3). In an animal model of spinal cord DCS treated with a constant 2.0 bar pp oxygen, recovery was not significantly altered by recompressing deeper than 20 msw.¹ This, and other studies, support the belief that going deeper brings little further benefit. In a similar study, pressure was held constant at 5.0 bar and the pp of oxygen was varied between 1.0 and 3.0 bar.³⁴ Treatment with a pp of oxygen of 2.0 bar (10 msw) achieved the best results suggesting that the optimum treatment of DCS was at 10 msw on 100% oxygen. DCS has been reported following treatment with hyperbaric oxygen³⁵, so potentially at least, oxygen as well as inert gas, can cause a DCS like syndrome. The use of oxygen and pressure in the manner empirically derived by Goodman and Workman¹⁴ is gradually being supported by work from the laboratory. The time over which this "dose of pressure" and "dose of oxygen" needs to be applied is, as yet, less clear. What is clear though, is that oxygen treatment at lower pressures is superior to deeper recompression on air. Although the current oxygen tables have a failure rate of between 4 and 15%, this was achieved in much more seriously afflicted patients than

those previously treated on the air tables.

The advent of computer technology has seen the development of much remarkable investigative equipment, and with the realisation that DCS is primarily a disease of the nervous system, follow up investigation has been directed towards assessing the neurologic sequelae of DCS. Gorman, Edwards and Parsons³⁶ in treating 87 cases of DCS achieved a 96% resolution rate at discharge. At 1 week follow up, 47% had abnormal EEGs and 20% had abnormal CT scans. So, is our treatment of DCS as effective as we might think, or has improving technology just reinforced our fears that we really know little about this disease and how to treat it effectively?

The future

Current research is being directed at alternative approaches to the treatment of the difficult cases, i.e. those in which the victim deteriorates while at treatment depth, or those with significant deficits not improving at treatment depth. Previously, further compression to 30 or 50 msw has been advocated. Mixtures of oxygen and an inert gas (helium or nitrogen) are breathed and a saturation type final decompression is adopted for the return to the surface.^{1,23,26,27} The use of helium has been favoured recently because of the existence of safe decompression tables for heliox diving and because of the decreased incidence of serious DCS noted in heliox divers.^{37,38} Heliox diving however is usually performed by professional divers in saturation, and the relevance of these studies to the treatment of no-decompression sport divers is tenuous. Isolated case reports with sports divers demonstrate that these techniques are occasionally effective, but then failures are not usually reported.^{2,32,37,38}

In contrast, in 1984 the USN began pursuing the option of being able to remain at 18 msw indefinitely when the diver was not responding to standard therapy. Advantages were seen in this, as no additional gas mixtures would be required thus simplifying therapy. This investigation saw fruition in 1989 when treatment table 7 was promulgated in the USN Diving Manual.³⁹ Table 7 is a "heroic measure" for the treatment of the seriously ill diver and is essentially an extension of Table 6 at 18 msw with a saturation type decompression to the surface over 36 hours. The minimal time at 18 msw is 12 hours with solid evidence of continuing benefit required for stays of longer than 18 hours. A maximum of 24 oxygen breathing periods are allowed for. This gives a table length of 48+ hours. The usefulness of this technique is yet to be verified!

In the meantime we shall all continue to search for a solution to the treatment of the non-responding neurological DCS patient.

Conclusions

The minimal recompression, oxygen breathing ta-

bles have remained effective in the treatment of a sports diver population that is presenting with increasingly more severe disease. However, as our investigative tools become more sophisticated, previously covert disease will be demonstrated. When the efficacy of these tables is tested against our ability to demonstrate pathology in this minimally affected group of DCS patients they may, as have the air tables in the past, be found wanting. Continued development of investigative tools and the on-going analysis of results is warranted.

References

- 1 Leitch DR and Hallenbeck JM. Pressure in the treatment of spinal cord decompression sickness. *Undersea Biomed Res* 1985; 12(3): 291-305
- 2 Shilling CW, Carlston CB and Mathias RA. *The Physician's guide to diving medicine*. New York, Plenum Press, 1984; 286
- 3 Pol B and Watelle TJJ. Memoire sur les effets de la compression de l'air. *Annal d'hygiene publique et de medicine legale (Paris)*, 1854; 1: 241-279
- 4 Bert P. *Barometric pressure*. 1878. (1943 Translation to English, republished by Undersea Medical Society, Bethesda 1978)
- 5 Zuntz N. Zur Pathogenese und Therapie der Durch Rashe Luftdruckanderungen Erzeugten Krankheiten. *Forschr. d. Med Berlin* 1897; 15: 632-639
- 6 Behnke A and Shaw L. The use of oxygen in the treatment of compressed air illness. *US Naval Medical Bulletin* 1937; 35(1): 61-73
- 7 Yarbrough O and Behnke A. Treatment of compressed air illness utilising oxygen. *J Indust Hygiene Toxicol*, 1939; 21(6): 213-218
- 8 Van der Aue OE, White WA, Hayter R, Brinton ES, Kellar JR and Behnke A. *Physiologic factors underlying the prevention and treatment of decompression sickness*. USN Experimental Diving Unit, Report 1, Washington DC: USN, 1945
- 9 Van der Aue OE, Duffner GJ and Behnke AR. The treatment of decompression sickness: an analysis of 113 cases. *J Indust Hygiene Toxicol* 1947; 29: 359-366
- 10 Slark AG. *Treatment of 137 cases of decompression sickness*. RNPL report 8/62 August 1962
- 11 Rivera JC. Decompression sickness among divers: an analysis of 935 cases. *Milit Med* 1964; 129: 314-334
- 12 Goodman MW. Decompression sickness treated with compression to 2-6 atmospheres absolute. *Aerospace Med* 1964; 35: 1204-1212
- 13 How JYC. Medical support of compressed air tunneling in the Singapore Mass Rapid Transit project. *SPUMS J* 1989; 19(4): 155-172
- 14 Goodman MW and Workman RD. *Minimal recompression oxygen breathing approach to treatment of decompression sickness in divers and aviators*. USN Experimental Diving Unit Research Report 5-65, Washington DC: USN, November 1965
- 15 Workman RD. Treatment of bends with oxygen at high pressure. *Aerospace Med* 1968; 39: 1076-1083
- 16 Bornman RC. Limitations in the treatment of diving and aviation bends by increasing ambient pressure. *Aerospace Med* 1968; 39(10): 1070-1076
- 17 Lambertsen CJ. Concepts for advances in therapy of the bends in undersea and aerospace activity. *Aerospace Med* 1968; 39(10): 1086-1093
- 18 Erde A and Edmonds C. Decompression sickness: a clinical series. *J Occup Med* 1975; 17(5): 324-328
- 19 Davis JC (Editor). *Treatment of decompression accidents among sport scuba divers with delay between onset and compression*. UMS Workshop Report No. 34, Bethesda, Maryland: Undersea Medical Society, 1979
- 20 Melamed Y and Ohry A. The treatment and the neurological aspects of diving accidents in Israel. *Paraplegia* 1980; 18: 127-132
- 21 Hart GB. Treatment of decompression illness and air embolism with hyperbaric oxygen. *Aerospace Med* 1974; 45(10): 1190-1193
- 22 Bornman RC. *Experience with minimal recompression oxygen breathing treatment of decompression sickness and air embolism*. USN Experimental Diving Unit Report 2/67, Washington DC: USN, 1967
- 23 Kizer KW. Delayed treatment of dysbarism. *JAMA* 1982; 247(18): 2555-2558
- 24 Green RD and Leitch DR. Twenty years of treating decompression sickness. *Aviat Space, Environ Med* 1987; 58: 362-363
- 25 Pilmanis AA. Treatment for air embolism and decompression sickness. *SPUMS J* 1987; 17(1): 27-32
- 26 Leitch DR and Green RD. Additional pressurisation for the treatment of non-responding cases of serious decompression sickness. *Aerospace and Environ Med* 1985; 56(12): 1139-1143
- 27 Pilmanis AA. Hyperbaric oxygen therapy rationale for the treatment of diving accidents. *SPUMS J* 1987; 17(1): 20-23
- 28 Davis JC et. al. Altitude decompression sickness: Hyperbaric therapy results in 145 cases. *Aviat Environ Med* 1977; 48(8): 722-730
- 29 Leitch DR and Green RD. Pulmonary barotrauma in divers and the treatment of CAGE. *Aviat Space, Environ Med* 1986; 57(10): 931-938
- 30 *USN Diving Manual* 1985. Best Publishing Co., 1985; section 8: 21-50
- 31 *RN Diving Manual*. BR 2806 HMSO: London, 1987; section 5: 15-26
- 32 Edwards C, Lowry C and Pennefather J. *Diving and subaquatic medicine*. 2nd edition. Sydney, Diving Medical Centre, 1981; 160-161
- 33 Saumarez C, Bolt JF and Gregory RJ. Neurological decompression sickness treated without recompression. *Brit Med J* 1973; 2: 151-152
- 34 Leitch DR and Hallenbeck JM. Oxygen in the treatment of spinal cord decompression sickness. *Under-*

sea Biomed Res 1985; 12(3): 269-289

- 35 Donald KW. Oxygen bends. *J Appl Physiol* 1955; 7: 639-644
- 36 Gorman DF, Edwards CW and Parsons DW. Neurologic Sequelae of DCS: A clinical report. *Underwater and Hyperbaric Physiol IX*. 1987; UHMS: 993-998
- 37 McIver NKL. Treatment of compressed air decompression accidents. *J R Soc Med* 1989; 82(2): 74-79
- 38 Douglas JDM and Robinson C. Heliox treatment for spinal DCS following air dives. *Undersea Biomed Res* 1988; 15: 315-319
- 39 *USN Diving manual* 1989. Washington DC: US government printing office, Section 8: 29-39

TABLE 1

DIVERS IN TASMANIA

Registered by the Department of Sea Fisheries

Non Commercial Diving Licence	3100
Commercial Abalone Licence	125
Commercial Diving Licence (including police divers)	229

Unregistered divers (estimates)

Unlicenced Amateur Divers	3000
Sea Urchin and Periwinkle Divers	400
Aquaculture divers	50

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**HIGH RISK DIVING
TASMANIA'S AQUACULTURE INDUSTRY**

David Smart and Peter McCartney

Despite its cool climate and waters Tasmania has a large population of commercial and sports divers working in, and enjoying the waters of its 3,200 km coast line. Of a total population of 446,500 (1986 figures)¹, over one percent are active divers (Table 1).

Diving accidents in Tasmania requiring hospital admission or recompression therapy occur on average fifteen to twenty times per year. These patients are treated at the Royal Hobart Hospital (RHH) Recompression Chamber by staff of the Hyperbaric Unit, which is linked to the Department and Anaesthesia. There has been little change in the number of accidents over the years 1985 to 1988. The 1985-1988 population treated at RHH consisted mostly of abalone divers and a small number of recreational divers. In 1988 the first of a new population of divers presented for treatment. These divers were employed in the aquaculture industry. From April 1988 to October 1989, eighteen divers from all sources were admitted to hospital for diving related illness and twelve were treated in the recompression chamber. Sixteen of the eighteen divers had been using hookah apparatus and nine divers were employed in the aquaculture industry.

This industry is now a major export earner for the state of Tasmania and by October 1989 according to the Department of Sea Fisheries more than 270 people were employed at 35 separate ventures. At the time of writing

approximately 40-50 were divers. During 1990 further growth is expected in the industry to over 400 employees. From its humble beginnings in 1986 the industry now produces Atlantic Salmon of world export quality. The salmon are "farmed" from smolt (50 g size) to adult size in floating circular pens approximately 20 m in diameter (Plate 1, page 151) supporting a cylindrical net approximately 8 m deep. Feed is released automatically at regular intervals to the fish. There can be as many as 5,000 fish per pen. Larger operations manage 40 pens or more. Surrounding the inner pens is a coarser mesh perimeter net (Plate 2, page 151) of up to 250 m by 500 m to prevent predators such as seals attacking the salmon. Divers in the industry are required to maintain these nets and pens and to remove diseased or dead fish from the pens. They are also required to inspect and maintain mooring lines. In some leases the perimeter nets and mooring lines extend to depths of 40 m. Divers contribute significantly to the quality of the salmon when it is finally ready for marketing.

This paper examines the diving practices of the industry, based on information gained from divers treated at RHH, in the hope of reducing the number of diving accidents in the future. One of the authors (DS) was privileged to visit one of the larger fish farm leases at Tassal, Dover, and witnessed its impressive operation at first hand.

Information gained

Nine aquaculture divers were treated at RHH. The majority (5) were aged between 21 and 30. Two were between 31 and 40. There was one in the 10-20 group and one aged between 41 and 50. Eight were male and one female.

Of concern is that only 2 divers had had appropriate training, i.e. specifically for the industry. Even more disturbing is the fact that 4 of the divers had had no formal training while the other three had only had formal training for using scuba as a recreational diver. Of these one had

more than 5 years' diving experience while 2 had less, one of them less than a year's experience.

Presenting syndromes

4 divers presented with decompression sickness. Two had neurological symptoms and two had musculoskeletal symptoms. Three presented with inhalational pneumonitis (discussed below) and two with barotrauma (also discussed later).

TABLE 2

BACKGROUND OF DIVERS REQUIRING TREATMENT

Age	Number of Divers	
10-20	1	
21-30	5	
31-40	2	
41-50	1	
Sex	8 Male	1 Female
Equipment used: Hookah surface supply (all divers).		
Diving Experience		
No formal training	4	
Sport diving training:		
Less than 1 year	1	
1 - 5 years	1	
More than 5 years	1	
Other training specific to the industry	2	
Presenting Condition		
Decompression sickness		
Neurological	2	
Musculoskeletal	2	
Hydrocarbon inhalation		
Pneumonitis	3	
Sinus and Aural Barotrauma	2	

Discussion

Divers working in the aquaculture industry are employed in a dual capacity to perform diving tasks as well as non-diving "farm hand" tasks. Their daily underwater tasks are to inspect and maintain salmon pens and nets and pick up dead fish from within the pens. They also dive and maintain anchor ropes, moorings and lines and inspect and repair the perimeter nets on a regular basis. Their other tasks are to change all salmon pen nets once a week, also clean and repair them. They have to repair and maintain their diving equip-

TABLE 3

RISKY DIVE PRACTICES IDENTIFIED FROM INTERVIEWS WITH PATIENTS

Diver training

Training is inadequate or inappropriate to the industry.

Equipment maintenance/use

- No set schedules exist for maintenance of gear.
- Untrained personnel allowed to tamper with gear.
- Use of non-recommended materials for air filtering.
 - Salt water in regulators.
- Use of non-recommended oil in hookah compressor.
 - Use of hookah apparatus to excessive depths.
 - Inappropriate thermal protection.
 - Limited use of specialised underwater tools.
 - No method of diver to surface communication.

ment, feed the salmon, fill the feeders and exchange batteries for the feeders and finally catch salmon for processing.

This system should allow some flexibility. Divers who are unable to dive, for reasons such as an upper respiratory tract infection, or who are on a "rest day" can still contribute to the running of the enterprise and so reduce their risk of diving related illness.

In theory this "multi-skilling" should work well, allowing for rest days from diving and rotating divers in and out of the water according to diver health and decompression table limits.

In practice, based on information supplied by patients treated at RHH, divers are at greater risk of accidents for many reasons (Table 3). The risk to health covers the whole spectrum of diving related illness.

Diver training

This is inadequate or inappropriate to the industry. At present there is no requirement by law in Tasmania that professional divers must receive formal diving training from any authority, although Australian standards for underwater air breathing (AS 2299)² recommends training specific to the type of equipment or diving apparatus being employed. Insufficient training is highlighted in this study. Only five out of nine divers had received formal diving instruction and only two out of nine had training specific to the industry, which is available through the Maritime College in Launceston. Sport diving basic qualification is better than no training at all, however this is not appropriate to the type of diving performed in the industry. Basic scuba training does not encompass the use of hookah apparatus, deep diving or equipment maintenance and varies considerably between



PLATE 1. Diver entering salmon pen wearing hookah apparatus.



PLATE 2. Salmon pens, work boats and the surrounding perimeter net.

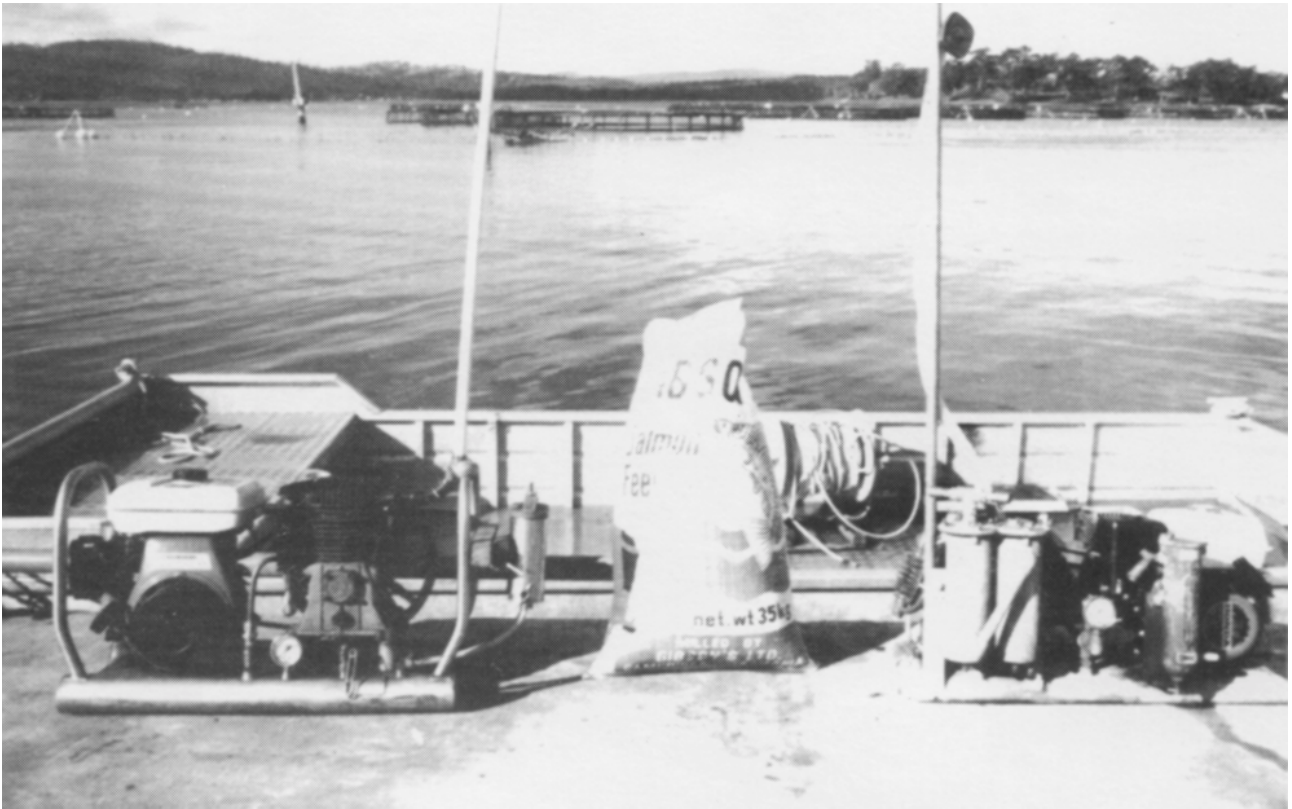


PLATE 3. Hookah apparatus and salmon feed bag with salmon pens in the background.



PLATE 4. Diver exiting from pen.

diving training bodies in the standard of life saving training. Basic scuba training does not cover the use of specialised underwater tools which enable more efficient work practices and reduced bottom times. Adequate training for such a strenuous, high risk occupation is essential.

Without knowledge of tables, basic physics, safety and equipment divers place themselves in potentially life-threatening situations. In addition, lack of training prevents a diver from recognising practices which place he or she at greater risk of accidents. It is known, for example, that barotrauma of the ear has a high incidence in novice divers³ who are involved in training courses due to inexperience with ear clearing techniques.

Diving equipment and its maintenance

Surface supply (hookah) is almost universally used for air supply to divers in the industry. A petrol motor drives a low pressure compressor feeding a reservoir. The reservoirs of the two available models in Tasmania are 12 litre and 17 litres (Plate 3 page 152). Air is supplied to the diver via a long hose and an upstream second stage. The second stage has a non-return valve to prevent diver injury in the event of a sudden cessation of the air supply. Depth limitations exist because of the inability of the equipment to deliver a sufficient volume of air to the diver at pressures greater than 3 to

4 atmospheres absolute (ATA). Thirty metres (4 ATA) is the maximum safe depth for hookah apparatus.

Hookah is a very safe system provided the equipment is well maintained and the air intake is positioned upwind and well above the exhaust. It is essential to use the correct filters and appropriate oil in the compressor. There must be no kinks in the air supply hose and the demand valve (or regulator) must be functioning properly. The depth restrictions of the equipment need to be strictly observed. Three divers reported using the hookah apparatus to depths greater than 30 metres for inspection and maintenance of perimeter nets surrounding the fish farm. This practice places them at significantly increased risk of inadequate air supply due to depth, the hazards of nitrogen narcosis as well as the higher risk of DCS of deeper diving. Surface attendants are essential with hookah diving. The compressor must never stop while the diver is underwater as the pressure reservoir can usually only supply a few breaths. For safety a backup diver should be available.

Divers in the study population reported that no diver-to-surface communications were used on the farms. Communication systems provide for additional diver safety, especially during deeper dives. However the cost of the equipment necessary (commercial diver full facemasks or helmets and microphones) is much higher than the usually cheap regulators normally used with hookah systems.

Written standards and protocols for equipment handling and maintenance need to be present and used in the industry so that high standards of safety can be practiced on all farms where diving occurs. Lack of protocols for equipment maintenance was demonstrated when three divers presented to Royal Hobart Hospital after breathing contaminated air. They presented after they had been using tea towels as output filters instead of the recommended charcoal filter.

These divers had also changed the oil in the compressor when the air tasted "oily". They demonstrated their lack of knowledge of the equipment they were using by replacing the original oil in the compressor with vegetable oil purchased from a supermarket. After breathing air contaminated with this oil their symptoms ranged from dyspnoea, chest tightness and cough to haemoptysis and headache. None had significant carboxyhaemoglobin levels and chest x-rays were all normal. All had reduced arterial oxygen saturations and mild impairment of ventilatory function which returned to normal after 24 hours breathing humidified oxygen.

These divers were diagnosed as suffering a mild form of hydrocarbon pneumonitis from which they were fortunate to make a full recovery. Inhalation of sufficient amounts of hydrocarbons can cause fatalities.^{4,5} The effects of inhaled hydrocarbons are compounded at depth (under pressure). Using vegetable oil has the added risk of carbon monoxide

poisoning from partial combustion of oil in the compressor.

If protocols for service and maintenance of equipment (including adequate filters) had been formulated and followed and the divers properly educated about use of their equipment, this dangerous situation could have been prevented.

Hookah equipment is used on fish farms for up to 8 hours every day and is subject to corrosion from salt water. Several divers reported inhaling salt water from the equipment they were using. This is a sign of a (poorly maintained) leaking expiratory valve. Salt water damage to equipment should be minimised by specific maintenance and handling schedules.

Two divers reported using 5 mm thickness wetsuits all year round. For short dives, very hardy divers may tolerate the Tasmanian winter (water temperature 12°C) in a 5 mm wetsuit but for professional diving this degree of thermal protection is inadequate and has accompanying risks of cold stress. There is theoretically an increased risk of decompression sickness. Hypothermia poses many risks to the diver and should be prevented with appropriate thermal protection and shorter diver profiles.^{3,6}

Another area which needs to be considered in the industry is maximizing diver efficiency while underwater. Tools designed specifically for underwater use are available and should be evaluated for use in the aquaculture industry. Every minute saved in bottom time reduces the risk to the diver.

Diving practices and profiles

We found a number of areas where divers are at risk of serious accident. In general there was no logging of dive duration nor of entry or exit times. No depth gauges were used. There was no correlation of dive times with accepted dive tables. As a result many divers exceeded the table time limits. There was no schedule for safe ascent rates. Some divers were performing deeper dives after shallow dives. Many divers did multiple bounce dives to depths of up to 15 metres. No divers used "safety" decompression stops. Many divers were performing unnecessary strenuous exits (vaulting the fence) during diving. As well many were performing strenuous exercise after diving. In one case further bounce diving after an emergency ascent produced decompression sickness.

The nature of the diving is unique and places the diver at greater risk of barotrauma and decompression sickness even if accepted protocols are followed. In their daily inspection of the fish pens, divers may be called upon to perform bounce dives to a depth of 10 m 20-40 times over a four to five hour period. Multiple ascents during dives (bounce diving) places a diver at higher risk of decompression

sion sickness (DCS) and barotrauma. Prolonged shallow saturation dives have been shown to produce a high incidence of DCS.⁷

Multiple ascents and rapid ascents are now accepted as major risk factors for DCS, independent of exceeding table limits.^{3,8,9} During these dives the pens are inspected for damage and repaired and dead fish removed from the floor of the pen. A risky practice identified by one author's (DS) personal inspection of the pens was throwing rocks into the pen if the floor of the pen did not remain submerged. As a result, the shape of the pen was converted from a cylinder to an inverted cone. The depth of the apex of the cone was measured at 15 m by one of the authors, using a hand held depth gauge. This deepening of some pens to 15 m further increases the risk of DCS.

Divers on the aquaculture farms have a very heavy physical workload. A long swim is required to inspect the pen nets for damage and there may be up to 40 dead fish, weighing 3-4 kg each, to collect from the pen. In addition, extracting oneself from the pen is no easy task requiring strong shoulders and legs and a rapid ascent from 1-2 m to reach the fence and heave oneself across it. Use of a ladder would simplify this task and eliminate extra unnecessary physical work.

After completing their diving the farm hands often are required to perform heavy physical work, such as changing and washing nets. These nets are extremely heavy and may weigh over one tonne when clogged with weed. The lightest work is feeding the salmon (each feed bag weighs 35 kg) and assisting in bringing the catch to shore. Performing heavy physical work after diving adds to the risk of DCS and should be avoided on diving days.

When the divers presented to RHH with medical problems it was impossible to precisely define the profiles they had been diving in the recent past because no records had been kept of dive times nor of entry and exit times, and no diver carried a depth gauge. Logging of dive profiles and following accepted decompression tables is essential for diver safety. Failure to adhere to safe diving practices and tables is the major cause of DCS in recent Australian series.^{7,10} No safety decompression stops were made by divers requiring treatment at RHH. One diver attempted to relieve his symptoms of DCS by performing another dive to 10 m for half an hour. A very dangerous practice, which compounded the existing problem, especially as one of his symptoms was vertigo.

In three of four cases of DCS the divers had performed a deeper dive (up to 30 m) after their previous shallower dives. All reported that it was not uncommon for divers to inspect mooring lines or the perimeter net (depths up to 40 m) after shallower pen dives were completed. Following shallow dives with deeper dives is a known risk factor for DCS.^{3,7,11} The practice of inspecting and repairing

moorings and perimeter nets on deeper dives after already completing a morning of shallow pen diving is extremely dangerous. A safer practice would be to perform all the deep dives before the shallower pen diving. It is the authors' opinion that no further diving should be performed on the same day a diver has been working at depths greater than 20 m.

Two of the four divers with DCS had to make emergency ascents due to air supply problems at depths greater than 20 m. One of these divers had complete cessation of air supply due to hookah motor failure and the other experienced inadequate air supply due to mechanical problems which was compounded by panic in a very low visibility environment. The small reservoir available was insufficient to supply adequate air to either diver to enable a controlled ascent. Their symptoms began after these emergency ascents. In neither case was a backup diver immediately available to assist the diver in trouble. Both divers were using hookah at the time and neither carried a backup scuba cylinder. Diving to greater depths to inspect the perimeter nets and moorings associated is with greater hazard because the risk of DCS increases in a non-linear fashion with depth. Without an independent air supply this risk is further compounded when air supply failure necessitates dangerous free ascents.¹²

Some divers dived with viral respiratory tract illnesses. Two divers presented with barotrauma (one with ear, and one with sphenoidal sinus barotrauma). Each had symptoms severe enough to result in referral to RHH by their general practitioner. Fortunately neither diver had other injuries. Respiratory tract illness prevents equalisation in the ears and sinuses and increases risk of barotrauma.¹³ Divers in the industry need to be free of any ENT or respiratory illness because the nature of the diving requires frequent equalisation.

Safety and first aid

A number of unsafe practices were identified. Diving took place without a backup diver immediately available on the surface. In most cases there was no first aid or oxygen equipment immediately available. The divers had insufficient knowledge of what dive practices were risky and of the treatment of diving accidents. Some divers dived with upper respiratory tract infections.

Lack of a backup diver immediately available places the diver at risk, especially when performing deep dives around nets with the risks of entanglements, loss of air supply and narcosis. Although a boat hand is always with the diver, divers reported that no first aid or oxygen equipment was in the boats ready for use if accidents occurred. Carrying an independent scuba source on deeper dives could have prevented two divers making free ascents when they experienced air supply difficulties with the hookah apparatus.

All divers reported that there was no protocol documented for initial management of diving accidents. First aid management of diving accidents should be included in a training programme specific to the industry.

Conclusions

At the time of writing this paper there are numerous risks to the diver employed in the aquaculture industry. Many of these risks could be reduced by ensuring adequate diver training specific to the industry, by adhering to accepted diving protocols and decompression tables, by having written schedules for the maintenance and care of equipment and by adequate safety and first aid procedures.

Standards do exist for Underwater Air Breathing Operations (AS2299-1979)², and the general principles governing these standards can be applied to the aquaculture industry. To date many of these principles do not appear to have been implemented. There are however a number of deficiencies when applying this set of standards to the unique needs of the industry. These deficiencies need to be addressed when formulating future guidelines for diving practices. Three very important areas which need attention, in the authors' opinion, are training specific to the industry, standards for the use of hookah apparatus and its maintenance, and dive schedules for multiple shallow bounce diving. Consultation with similar industries in other countries (for example Scotland) would assist with this process. It may require a change to the job classifications of farm hands to allow AS2299 to apply to their diving activities. The expense of on-site recompression chambers is probably not justified provided acceptable safety and first aid protocols exist for the management of emergencies.

Now moves, supported by employers and unions, are under way in Tasmania, to review the diving aspects of the aquaculture industry. It is pleasing to note that recent changes implemented at Tassal, Dover, have included the use of contract commercial divers to perform all dives in excess of 18 metres. This lead taken by Tassal constitutes a major advance in diver safety for the industry. It is hoped that in the near future a uniform set of standards will apply to maximize diver safety on all aquaculture leases in Tasmania.

Addendum

During the 6 weeks before submission of this paper (April 1990) a further three divers from the aquaculture industry had been assessed and treated for significant decompression sickness at Royal Hobart Hospital. Only one of these divers had any formal diving experience. One diver had dived every day for three weeks before presenting with DCS.

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References

- 1 Australian Bureau of Statistics. *Population Statistics for Tasmanian Census 1986*, Hobart, Australian Government Printing Service, 1986
- 2 Australian Standard *Underwater Air Breathing Operations*. Standards Association of Australia, AS2299, 1979
- 3 Edmonds C, Lowry C and Pennefather J. *Diving and subaquatic medicine*. Sydney Diving Medical Centre 1984
- 4 Perrone H and Passero M A. Hydrocarbon aerosol pneumonitis in an adult. *Arch Intern Med* 1983; 143: 1607-1608
- 5 Eade N R, Taussig L and Marks M I. Hydrocarbon pneumonitis. *Paediatrics* 1974; 54: 351-357
- 6 Taylor R F and Yesair D W. Diver hypothermia. *SPUMS J* 1988; 18(2): 66-69
- 7 Eckenhoff R G, Osbourne S F, Parker J W and Bondi K R. Direct ascent from shallow air saturation exposures. *Undersea Biomed Res* 1986; 13(3): 305-16
- 8 Gorman D F, Pearce A and Webb R K. Dysbaric illness treated at the Royal Adelaide Hospital 1987. A factorial analysis. *SPUMS J* 1988; 18(3): 95-102
- 9 Davis J C. Decompression sickness in sport scuba diving. *Physician and Sports Med* 1988; 16: 108-121
- 10 Orton J. Medical problems of recreational diving. *Aust Fam Phys* 1989; 18(6): 674-685
- 11 Lippmann J. Lessening the risk of decompression sickness. *SPUMS J* 1988; 18(2): 70-76
- 12 Walker D. Limbo diving. The dangers of free ascent. *SPUMS J* 1988; 18(4): 133-136
- 13 Farmer J C. Otolaryngology and diving, Parts I and II in: *Hyperbaric and Undersea Medicine*, Davis J C (Ed) 1981, Medical Seminars Inc., San Antonio, Texas

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REHABILITATION OF A PARALYSED DIVER

Jimmy How

Introduction

Since the 19th century, recompression has been utilised to treat decompression sickness (DCS). Since then tremendous work and research has been done to improve the efficacy of treatment. Decompression sickness has classically been divided into Type I and II, depending on site of involvement and presenting symptoms and signs. This clinical classification was presented as an attempt to differentiate serious from not so serious cases, and thus to facilitate therapy decisions and prognosis prediction.

The Diving and Hyperbaric Medical Centre of the Republic of Singapore Navy was established in 1971. From 1 April 1989, it has been reorganised and renamed the Naval Medicine and Research Centre (NMRC). In addition to providing therapeutic care to divers of the Singapore Armed Forces, its Diving Medicine Branch also caters to civilian divers of all categories.

In Singapore, sports diving has fast been gaining popularity and there is a growing population of sports divers. Another group of divers seen at NMRC are the commercial divers involved in offshore oil exploration around this region. However, it is the divers of the fishing vessels in the Indo-Pacific region that form the majority of decompression sickness cases seen at NMRC (Table 1).

NMRC has used a variety of recompression tables for the treatment of DCS. However, we have abandoned short oxygen tables and now only use long oxygen tables for the treatment of both type I and type II cases, as studies have shown that divers with so-called type I DCS can develop an overt neurological deficit over time.

Profile of a fisherman diver

In Singapore fisherman divers band together and contract themselves to owners of small fishing boats. The crew of each boat numbers from 6 to 12. Of these, 3 or 4 are fisherman divers. Fishing is usually done at the South China Sea or the Indian Ocean, depending on the monsoon season. These deep sea fishermen operate hundreds of miles from Singapore.

However, fisherman divers in Singapore are a dying breed. 10 years ago, there were about 3,000 of them. Now, only 300 are plying this trade still with the same primitive equipment. They usually come from a poor education

TABLE 1

FISHERMAN DIVERS TREATED AT DHMC 1972 TO 1988

Year	DS Type		Total
	I	II	
1972	0	3	3
1973	0	5	5
1974	0	4	4
1975	0	2	2
1976	0	3	3
1977	0	6	6
1978	0	5	5
1979	1	7	8
1980	0	6	6
1981	0	5	5
1982	0	3	3
1983	4	4	8
1984	1	6	7
1985	3	5	8
1986	0	0	0
1987	0	2	2
1988	2	1	3
Total	11	67	78

background and receive no formal instruction in diving physiology nor in safety precautions.

Today the situation is improving. Since the 70s, our Diving Medical Centre has been actively educating these divers in safety measures and avoidance of DCS. Now they know the various diving tables, ascend with decompression stops and the number of local DCS cases seen is dwindling. We are now seeing more fisherman divers with DCS from neighbouring countries such as Indonesia and Malaysia. During the period 1984 - 1988, one third of fishermen DCS cases seen were from Malaysia or Indonesia, compared with the period 1972 - 1983, when only 15% were from these 2 countries. (Table 2).

Diving characteristics

The job of a fisherman diver is typically as follows. He will first do a breathhold dive to survey the area for fishes. It is said that the "Taikong" (the chief of divers), has the sixth sense of "hearing" where the fish will be before the crew make their dives. Once a suitable site is found, he will descent with the aid of weight belts and surface supplied

TABLE 2

**NATIONALITY OF THE FISHERMAN
DCS CASES 1972 TO 1988**

	Number	Percentage
Singaporean	62	79.5
Malaysian	9	11.5
Indonesian	7	9.0
Total	78	100.0

TABLE 3

**THE MAJOR PRESENTING SYMPTOMS OF
FISHERMAN DIVERS WITH DCS 1972 TO 1988**

Presentation	Number	Percentage
Joint Pain	35	44.9
Weakness	52	66.7
Sensory Deficit	40	51.3
Bladder Involvement	21	26.9
Bowel Movement	19	24.4

breathing apparatus (SSBA) to plant the explosives. The divers may dive from 3 - 6 times a day, and each dive may take up to several hours. The depth of diving is down to 45 m (150 feet), but averages 24-30 m (80-100 feet). Diving is done using crude surface supply apparatus. Air is usually supplied by a compressor pump directly to the diver.

Not infrequently, the dives are interrupted due to compressor failure. As a result, the divers are required to ascent rapidly. Another occasion that they need to do this is when they are attacked by sharks or pirates attack the boat. Even in routine dives, no decompression stops are made as these divers are ignorant of diving physiology and safety precautions.

Characteristic presentation of a fisherman DCS case

Because of the high nitrogen load (due to repetitive dives) and rapid ascent rate (due to ignorance of decompression stops), the onset of symptoms usually occurs within 15 minutes of surfacing. They usually suffer from severe type II bends, frequently associated with loss of consciousness and paraplegia with loss of bladder and bowel functions (Table 3). Of the 78 fisherman divers with DCS seen at our centre, 18 (23%) had loss of consciousness at the site of the incident.

As most fishing boats operate hundreds of miles from Singapore, it will take them several days to reach our treatment centre. During this period, the fisherman divers usually attempt wet recompression during their journey back. However, no proper schedule is followed and frequently after alleviation of the initial symptoms, the patient will be pulled up so rapidly that there is a recurrence of symptoms and the diver becomes even worse off than his initial condition.

Associated with the paraplegia or paraparesis, there are various degree of sensory loss, bladder and bowel dysfunction, dehydration and bed sores due to poor nursing care. Because of the neurologic bladder with urinary retention, catheterisation is sometimes attempted by the fisher-

man, using non sterile techniques, including the use of wires or even the outer coating of a section of electrical wire. As a result, there is often urinary tract infection. Recently these fisherman divers have started to use sterile disposable catheters.

Effects of treatment

Despite the delay, which may be more than 10 days, recompression using oxygen tables is still the mainstay of treatment, together with correction of dehydration and treating any complications such as urinary tract infection, bed sores, etc. and then rehabilitation.

Unlike acute cases, where the injury is due mainly to the blockage of blood flow by nitrogen bubbles, in these delayed cases, the hypoxic state of the spinal cord is due mainly to complications caused by the bubbles. These include blockage of vessels by fibrin and platelet clots with damage to the walls of capillaries and small vessels leading to abnormal permeability. This allows leakage of plasma into the interstitial space and causes oedema, resulting in haemoconcentration and dehydration. All these will contribute to the hypoxic state. Thus in addition to recompression oxygen tables, rehydration therapy using crystalline fluids and plasma expanders, steroids and aspirin are commonly used to treat our delayed cases.

Table 4 shows the time lapse before recompression therapy was started. There were 4 cases which presented more than 15 days after the insult. These case histories appear below.

Case 1

Mr OKC, 47 year old Malaysian, Chinese, Male.

Date of incident: 16 October 1978

Place of incident: South China Sea

Dive profile:

Did 3 dives to 120 feet on the same day. Each dive lasted 60 minutes. No decompression stop.

Symptoms at site of incident:

Weakness left lower limb, loss of sensation of left

TABLE 4

THE DELAY (NO. OF DAYS) IN THE DECOMPRESSION TREATMENT 1972 TO 1988

Days of Delay	Treatments	Percentage
1 - 2	5	6.4
2 - 5	39	50.0
>5 - 10	21	26.9
>10 - 15	9	11.6
15 or more	4	5.1
Total	78	100.0

thigh, no loss of bladder or bowel function.

Date of first recompression treatment: 31 October 1978

Physical examination before treatment:

Power: 3-4/5 left lower limb

Sensation: Sensory level L3 left lower limb

Treatment:

IV Rheomacrodex in Saline

Dexamethasone Injection for 3 days

Recompression using Table 6B.

Result:

30 minutes after the start of recompression therapy, the patient reported improvement in sensation and power. By the end of one treatment, he had full power in both lower limbs, and normal sensation.

Follow-up:

He was found to have dysbaric osteonecrosis affecting both the humeral heads when long bone X-rays were done on 3 November 1978. He was advised against further diving, and was given a recommendation to help him obtain a hawker's licence. On 7 April 1979, he was awarded compensation for a 39% disability (\$14,000). He was last seen on 6 December 1979, when he was still diving despite our advice.

Case 2

Mr TCK, 40 year old Singaporean, Chinese, Male.

Date of incident: 25 May 1979

Place of incident: South China Sea

Dive profile:

Did one dive to 120 feet. Lasted 120 mins. Slow ascent with no decompression stop.

Symptoms at site of incident:

Onset 15 minutes after ascent. Had loss of consciousness, and wet recompression was attempted.

Date of first recompression therapy: 11 June 1979

Signs and Symptoms before treatment:

Only complained of joint pain involving the left shoulder and left elbow. Power 5/5, sensation intact, good bowel and bladder control.

Treatment:

Recompression using RN Table 61.

Results:

Complete resolution of pain after one treatment with no residual symptoms and signs.

Follow-up:

Last seen 15 September 1980. Found to be well. However, dysbaric osteonecrosis was noted in the shaft of both femur and tibia, and also in the right femoral neck. He was advised against further diving, and a recommendation letter was given to assist him to obtain a licence to be a fishmonger in a market.

Case 3

Mr YCH, 39 year old Singaporean, Chinese, Male

Date of incident: 22 March 1983

Place of incident: South China Sea

Dive profile:

Did 2 dives to 84 feet on the same day. First dive lasted 30 minutes. Second dive lasted 75 minutes. Ascent with no decompression stop.

Symptoms at site of incident:

Had joint pain, loss of sensation and weakness of both lower limbs. Also had loss of bladder and bowel function. Attempted wet recompression and self catheterised with Foley's Catheter.

Date of first recompression treatment: 6 April 1983

Signs and symptoms before treatment:

Power 5/5. Sensory level L3-4, Constipation +, anal reflex +, catheterised, cremasteric reflex +.

Treatment:

Recompression table 62 (modified)

IV Rheomacrodex 10%.

IM dexamethasone

Results:

Power 5/5. Hypoaesthesia from both knees down. Able to pass urine. Anal reflex +.

Follow-up:

Last seen on 8 August 1983. Power 5/5. Sensation: Slight numbness along the posterior aspect of both legs. Otherwise well. He was advised to limit his diving to non-decompression dives.

Case 4

Mr LMK, 26 year old Malaysian, Chinese, Male

Date of incident: 25 May 1979

Place of incident: South China Sea

Dive profile:

Dived to 150 feet for 90 mins. Slow ascent with no decompression stop.

Symptoms at site of incident:

Immediately after ascent weakness both lower limbs, loss of sensation from the waist down. Able to pass urine and use bowels.

Date of first recompression treatment: 20 June 1979

Signs and symptoms before treatment:

Power 3-4/5 both lower limbs, sensory intact.

Treatment:

Recompression Table 62.

Results:

Power 4/5 both lower limbs, sensation intact.

Second Treatment: 21 June 1979 Table 62.

Results:

Power 4+/5 - 5/5, sensation intact.

Follow-up:

Last seen on 3 June 1983. He was well with no permanent disability. No evidence of osteonecrosis noted in X-rays.

The above 4 cases clearly indicate that even after prolonged delay, recompression therapy with appropriate adjuvants will improve DCS. It is thus our policy to treat all decompression sickness and to repeat therapy if necessary until there is no further improvement.

Rehabilitation

As seen in Table 5, only 21.8% of those treated recovered completely from their DCS, although a further 28.2% had near complete recovery. So 50% of those treated have a significant disability, requiring some form of rehabilitation. The residual disabilities include loss of motor power, sensory loss, bowel and bladder dysfunction. With an effective programme, a good percentage of these disabilities can be minimised and overcome, and the patient can regain useful psychomotor and social skills.

In Singapore all cases of DCS who have residual disability after recompression therapy are further managed at the Department of Rehabilitation Medicine (DRM) at Tan Tock Seng Hospital. The majority of cases seen at the DRM are DCS Type II with spinal cord involvement. In a study conducted by Tan and Balachandran¹, there was a total of 15 cases of DCS with spinal involvement treated at DRM from 1973 to 1978 and referred to DRM. All these had bladder and bowel function involvement. Five also had bone complications. All were male Chinese, with ages ranging from 25 to 64. Out of the 15, two had weakness of all 4 limbs whilst the rest had weakness of both the lower limbs. The results of rehabilitation are shown in Tables 6, 7 and 8.

Team approach

For total management of a DCS patient with residual disability, a team of professionals is required. The NMRC and DRM have teamed up to provide the rehabilitation programme for divers. Each specialist may be called upon to assist from their respective units. This team consists of:

- 1 Specialist in diving medicine.
- 2 Specialist in physical rehabilitation medicine.
- 3 Physiotherapist.

TABLE 5

RESULTS OF TREATMENT GIVEN TO FISHERMAN DIVERS WITH DCS 1972 TO 1988

Results of Treatment	Number	Percentage of Total
Complete Recovery	17	21.8
Almost Complete Recovery	22	28.2
Definite Improvement	29	37.2
No Definite Improvement	10	12.8
Clinically Deteriorated	0	0
Died	0	0
Total	78	100.0

- 4 Social worker.
- 5 Psychologist.
- 6 Urologist.
- 7 Orthopaedic surgeon.
- 8 Radiologist.
- 9 ENT surgeon.

Aims of Rehabilitation

The aims of rehabilitation after hyperbaric therapy are:

- 1 To achieve the highest level of useful motor power possible.
- 2 To help the patient to adjust psychologically to his limitations.
- 3 To achieve a catheter free state where the bladder is able to empty satisfactorily with voluntary control.
- 4 To enable normal bowel function.
- 5 To achieve functional independence and ultimately to return to the patient's former occupation.

Bladder Training

Unlike motor weakness and sensory deficit which improve considerably after recompression therapy, a significant percentage of cases of neurologic bladder do not improve even after repeated recompression. These people require a different mode of rehabilitation. Some require surgical intervention (transurethral resection of the sphincter), medication (Baclofen to improve micturition), and finally bladder training. The aim is to prevent overdistension, minimise local damage and infection, and obtain an adequately emptying bladder, with a low and sterile residual urine.

TABLE 6
SENSORY INVOLVEMENT (PAIN)

Case	Before Recompression Therapy	After Recompression Therapy	After Rehabilitation
KNS	Loss below T12	Hypoaesthesia below T12	Normal
WSK	Loss below T7	Hypoaesthesia below T12	Hypoaesthesia
ESL	Loss below T7	Hypoaesthesia below L5	Brown Sequard below C7
OTB	Loss below C7	Hypoaesthesia below T5	Hypoaesthesia below T5
KTK	Hypoaesthesia below T3	Hypoaesthesia below T3	Hypoaesthesia below L3
LPC	Hypoaesthesia below T5	Hypoaesthesia below T8	Normal
TKL	Hypoaesthesia below T10	Hypoaesthesia below T 10	Hypoaesthesia below T10 improved
SKS	Hypoaesthesia below T7	Hypoaesthesia below L2	Hypoaesthesia below T2 improved
QTH	Loss below T8	Loss below L3	Hypoaesthesia below L3
THS	Loss below T9	Hypoaesthesia below T10	Hypoaesthesia below L1
LKL	Loss below T5	Loss below T8	Normal
TLK	Loss below T8	Loss below T10	Loss below L5
NKS	Loss below T7	Loss below L2	Hypoaesthesia below L3
CYP	Loss below L1	Loss below L3	Hypoaesthesia below L3
QKH	Loss below T5	Loss below T10	Hypoaesthesia below L1

Bladder training is done at the DRM. On admission the patient is first subjected to a series of tests to get a bladder profile. The URODYN 5000, the latest urodynamic equipment acquired by the DRM, is used. This machine is able to provide information about the urinary flow rate, intravesical pressure, abdominal pressure, detrusor pressure, urethral pressure, water and carbon dioxide cystometry and electromyography.

With this information, we can then determine whether the patient has an upper motor neurone (UMN), lower motor neurone (LMN) or mixed type of bladder dysfunction. The capacity and efficiency of the bladder and the condition of the sphincters can also be shown. With these findings, the appropriate bladder training method can then be planned for the particular patient.

With a LMN lesion, the bladder tone tends to be flaccid. Emptying of the bladder can be assisted by cholinergics like carbachol, ubretid and pyridostigmine. In UMN lesions, the bladder tends to be unstable with a small capacity. This state can be improved by medications like oxybutynin to relax the tone. Dibenyline is also used to relax the internal sphincter. The capacity of the bladder can be gradually increased by hydrodilatation, a process using water to distend the bladder. If the patient still finds difficulty in micturition after a bladder training programme with medication, he is again assessed by the urologist for possible

surgical correction. A sphincterotomy can be done for those with high sphincter tone, and transurethral resection of the prostate (TURP) done for patients with enlarged prostates.

Prior to 1987, an indwelling catheter was used. Now 4 hourly intermittent catheterisation is used. Every 4 hours a doctor will apply abdominal tapping and compression just before inserting the disposable catheter. The volume drained by the catheter is recorded. This method is continued until the residual volume is below 100 ml. Once this is achieved, the patient is taught to do tapping and compression every 4 hours.

During bladder training the patient is given restricted fluid (normally 2 litres between 6 am and 6 pm). Serum creatinine, serum urea and electrolytes are done on admission. Urine microscopy is done twice weekly and urine culture and sensitivity thrice weekly during the entire period. If possible, an annual intravenous pyelogram is also done to detect early outlet obstruction, including urinary stricture formation.

Results of Bladder Training

Although recompression therapy fails to restore fully the neurologic bladder in some of the cases, a proper bladder training programme can lead to a satisfactory functional

TABLE 7
BONE AND MUSCLE POWER INVOLVEMENT

Case	Before Decompression Therapy	After Decompression Therapy	After Rehabilitation	Dysbaric Osteonecrosis
KNS	Paraplegic: Right Grade 2 Left Grade 3	Grade 3 Grade 4	Normal	Right Femoral Head affected (2 years after injury)
WSK	Paraplegic: Grade 0 bilateral	Grade 2 bilateral	Grade 4 + bilateral	Nil
ESL	Tetraplegic: Grade 0 all limbs	Paraplegic Grade 4 Bilateral	Right Grade 4 + Left Grade 5	Nil
OTB	Tetraplegic: Grade 0 all limbs	Upper limbs Grade 3 + Lower limbs Grade 1 +	Upper limbs Grade 4 + Lower limbs - Grade 2-3	Nil
KTK	Paraplegic: Grade 0 bilateral	Grade 2 + bilateral	Right Grade 4 Left Grade 3	Nil
LPC	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 5 Grade 4	Nil
TKL	Paraplegic: Grade 0 bilateral	Right Grade 3 Left Grade 4	Grade 4 Grade 5	Nil
SKS	Paraplegic: Grade 0 bilateral	Right Grade 2 Left Grade 3	Grade 3 Grade 4	Both femora and tibiae and right humeral head and shaft (6 months after injury)
QTH	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 4 + Grade 4	Left Femoral Head affected (2 months after injury)
THS	Paraplegic: Right Left Grade 2-3	Grade 2 Left Grade 5	Right Grade 4	Normal Nil
LKL	Paraplegic: Grade 2 bilateral	Right Grade 3 Left Grade 4	Grade 4 + Grade 5	Right Femoral Head affected (1 month after injury)
TLK	Paraplegic: Grade 0 bilateral	Right Grade 3 + Left Grade 0	Grade 4 + Grade 0	Right Femoral Head (2 months after injury)
NKS	Paraplegic: Grade 0 bilateral	Right Grade 2 + Left Grade 3 +	Grade 3 + Grade 4	Nil
CYP	Paraplegic: Grade 3 + bilateral	Grade 4 bilateral	Grade 4 + bilateral	Nil
QKH	Paraplegic: Grade 0 bilateral	Right Grade 4 Left Grade 3	Grade 5 Grade 4	Nil

TABLE 8

BLADDER INVOLVEMENT

Case	Before Recompression Therapy	After Recompression Therapy	After Bladder Training	Drugs Used and/or Surgical Intervention
KNS	Neurogenic Bladder with acute retention of urine	Straining required to initiate micturation	Normal function	Nil
WSK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
ESL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
OTB	Neurogenic Bladder with acute retention	No improvement	Spontaneous Micturation	Transurethral resection of sphincter
KTK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
LPC	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
TKL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
SKS	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
QTH	Neurogenic Bladder with acute retention	No improvement	Micturation needs tapping and compression	Nil
THS	Neurogenic Bladder with acute retention	Straining required to initiate micturation	Straining required to initiate micturation	Nil
LKL	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Nil
TLK	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
NKS	Neurogenic Bladder with acute retention	No improvement	Micturation needs compression	Lioresal 5 mg tab tds
CYP	Neurogenic Bladder with acute retention	No improvement	Straining required to initiate micturation	Transurethral resection of sphincter
QKH	Neurogenic Bladder with acute retention	No improvement	Normal function	Nil

state. In the study done by Tan and Balachandran¹, the majority of cases with impaired bladder function after recompression therapy achieved near normal function after rehabilitation training.

Bowel Training

Those DCS cases with bowel dysfunction are first put on a high roughage diet and laxatives such as Senokot and Agarol to help defaecation. In addition, a Dulcolax suppository is given every third day. With this regime, most patients will be able to empty their bowels regularly. Other measures that may be taken are enema soap and water or digital evacuation of the rectum. With this regime, the bowel will be reconditioned to empty every third day. For resistant cases, the regime may be continued indefinitely, or until such time spontaneous defaecation is possible.

Physical Training

Fishermen treated for DCS who are left with significant residual motor weakness (usually spinal bends with paraparesis) are referred to DRM, whose doctors will assess them for suitability and potential.

The programme for physical rehabilitation starts with a baseline assessment of the motor power of various muscle group. There is a graduated exercise programme starting with non-weight bearing exercises, followed by partial weight bearing and finally full weight bearing.

Training is assisted with the use of various aids and appliances. For strengthening the upper limbs, free weights are used extensively. To strengthen finger grip, a ball of plasticine like material is used to provide resistance for finger exercises. In addition, spring exercises are done. The limbs are exercised against the recoil force of springs of various strengths.

For the lower limbs free weights and spring exercises are used for initial strengthening. When the lower limbs are strong enough to proceed to partial weight bearing exercises parallel bars, walking frames and walking sticks are used. Most of these require good upper limb strength. For paraplegics the main aim is to achieve good upper limb function by doing weight lifting. The patient is then taught wheel chair transfer by the occupational therapist. This is to enable them to move themselves from the wheelchair to the commode, bed, etc.

In addition, there is a pool for hydrotherapy at the DRM. This is ideal for patients with severe lower limb weakness as buoyancy will eliminate the effect of gravity, thus allowing the limbs, which are normally too weak to move against gravity, to move freely.

Social Adjustment

As noted before, about a quarter of the fishermen DCS cases suffer from dysbaric osteonecrosis. If the dysbaric osteonecrosis is severe or affects the articular surfaces, we advise them against further diving and assist them in seeking compensation and alternative suitable employment. The same is done for severe type II DCS with residual functional impairment, such as spinal bends with paraparesis or hypoaesthesia. They are assisted in their application for a hawker's licence, a job which is highly lucrative and sought after even by healthy persons.

However, there are some "hardcore" divers who continued to dive despite their disability. This is most commonly due to the fact that their livelihood depends on being a fisherman diver, and they cannot find an alternative job.

For those with permanent disability, we assess the degree of disability and assist them in obtaining their workman's compensation entitlement. In the rehabilitation centre, they are taught and trained in various self-care activities such as going to the toilet, eating, dressing, etc. This is done for all patients with severe physical disabilities and the aim is to achieve complete independence either with or without aids or appliances.

Somatosensory evoked potentials (SSEP)

SSEP involves the stimulation of a peripheral nerve either in the upper or lower limb and the recording of the evoked potential. It has been proven useful in the investigation of disorders affecting the nervous system. Demyelinating and degenerative disorders display abnormalities in SSEP and it can be used to provide information on the site of the lesion as well as a means to monitor the progress of the disease and the efficacy of therapeutic measures.

At our centre, we are now conducting a study to compare how well the SSEP recordings correlate with the clinical findings. So far we have managed to do a SSEP recording for 9 of our DCS cases after their recompression therapy. So far no pre-recompression recording has been done for any of the cases. The results obtained and the clinical findings are given in Table 9.

SSEP Results

NORMAL VALUES

Median Nerve

N1 : 16.5 - 20.9 ms

P1 : 20.8 - 28.4 ms

Posterior Tibia Nerve

N1 : 43.2 - 51.8 ms

P1 : 34.0 - 39.5 ms

TABLE 9
SSEP RESULTS

Case	Nerve tested	Result	Clinical Findings
KSL	Right Median Nerve	N1 19 ms P1 44 ms*	Hypoaesthesia right hand
	Right Posterior Tibia Nerve	N1 48 ms P1 35 ms	Normal
AFY	Left Median Nerve	N1 21 ms P1 28 ms	Normal
	Left Posterior Tibia Nerve	N1 51 ms P1 44 *	Normal
HCL	Left Median Nerve	N1 17 ms P1 49 ms*	Normal
	Left Posterior Tibia Nerve	N1 52 ms P1 36 ms	Hypoaesthesia T8 downwards
LE	Left Median Nerve	N1 18 ms P1 21 ms	Hypoaesthesia C5, 6, T1
	Left Posterior Tibia Nerve	N1 47 ms P1 37 ms	Normal
ZAK	Left Median Nerve	N1 16 ms P1 20 mns	Normal
	Right Median Nerve	N1 16 ms P1 20 ms	Normal
	Right Posterior Tibia Nerve	N1 49 ms P1 64 ms*	Hypoaesthesia L2 downwards
TCT	Left Median Nerve	N1 18 ms P1 26 ms	Normal
	Left Posterior Tibia Nerve	N1 41 ms P1 36 ms	Normal
AL	Right Median Nerve	N1 16 ms P1 20 ms	Normal
	Left Posterior Tibia Nerve	N1 45 ms P1 36 ms	Normal
AHG	Median Nerve	N1 19 ms P1 35 ms*	Numbness fingers
	Posterior Tibia Nerve	N1 70 ms* P1 60 ms*	Patchy numbness lower limbs
SGN	Left Median Nerve	N1 20 ms P1 30 ms*	Normal
	Right Median Nerve	N1 20 ms P1 31 ms*	Hypoaesthesia T1-2
	Left Posterior Tibial Nerve	N1 8 ms P1 39 ms	Normal
	Right Posterior Tibia Nerve	N1 6 ms P1 40 ms	Normal

* denotes prolonged

NORMAL SSEP RESPONSES

Group One	Normal Clinical Findings
AL	
TTC	
Group Two	Abnormal Clinical Findings
KSL	Loss of proprioception right lower limb
LE	Hypoaesthesia C5 6 T1
HCL	Loss of pin-prick T8 downwards

ABNORMAL SSEP RESPONSES

Group Three	Normal Clinical Findings
AFY	Prolonged P1 right PT nerve
HCL	Prolonged P1 left median nerve
SGN	Prolonged P1 left median nerve
Group Four	Abnormal Clinical Findings
KSL	Prolonged P1 right median nerve with hypoaesthesia R hand
ZAK	Prolonged P1 right PT nerve with hypoaesthesia L2 onwards
ASG	Prolonged P1 median nerve with numbness fingers Prolonged P1 PT nerve with numbness lower limbs
SGN	Prolonged P1 right median nerve with hypoaesthesia T1-2

Discussion

In the first group, normal SSEP responses correspond to normal clinical findings. As SSEP measures the electrophysiological integrity of the sensory pathways, it gives an indication that the recovery from decompression sickness is complete.

In the second group of patients where normal SSEP responses were obtained in spite of clinical neurological deficits, it lends support to the suggestion put forth by Giblin² and Halliday and Wakefield.³ They proposed that the evoked responses depended on the integrity of the posterior column pathways but were unaffected by lesions of the spinothalamic tracts. The only anomaly in the group, LKS, may be explained by a study which suggested that some of the afferent impulses responsible for the cortical potential travelled by the spinothalamic tracts.

Group three results suggest that in spite of apparent full clinical recovery, residual damage had occurred secondary to decompression sickness. SSEP then, provides a sensitive means to detect subclinical damage. This result is supported by Palmer et al⁴ where extensive morphological changes existed in the spinal cord of divers who had a history of decompression sickness but recovered fully. The possibility of recruitment for neighbouring neurones in such cases

to provide compensation for the damaged one may explain the normal clinical findings in such divers.

In group four, prolonged SSEP responses were obtained in patients with sensory loss mostly to pin-prick. This differs from the conclusion drawn by Halliday and Wakefield.³ Whether this is due to the different aetiologies of the two study groups, (decompression sickness vs non-diving conditions, e.g. prolapsed intervertebral disc, cervical spondylosis, cerebrovascular haemorrhage, etc.) is uncertain. There is the opportunity for a more extensive prospective study involving a much larger sample size.

The most important conclusion obtained thus far in our present study is that abnormal SSEP were obtained in patients who are apparently normal subjects. This has important implications for our young naval divers and brings to mind the question whether routine SSEP study should be conducted for them. The difficult problem of medico-legal compensation also needs to be clarified in cases where prolonged SSEP were to be obtained in apparently normal divers. The other question that needs to be addressed is whether one should allow divers who have apparent full clinical recovery after decompression sickness but abnormal SSEP recordings to continue diving. Whether one previous episode of decompression sickness predisposes the spinal cord to further insults remains to be solved.

Dysbaric osteonecrosis

Another complication of diving is dysbaric osteonecrosis. Although the majority of patients present with decompression sickness, 36 out of the 78 DCS patients were found to have dysbaric osteonecrosis by post-treatment long bone X-ray. The disability suffered by those affected varies from mild asymptomatic type B to severe type A requiring arthrodesis and arthroplasty to achieve a pain free or functional state.

Conclusion

Since the beginning of our work in 1972⁵, fisherman divers have made up the majority of all DCS. Because of their typical late presentation they have contributed enormously to our experience in treating delayed cases. From the results we have obtained we are convinced of the potential benefit in treating cases even after a delay of more than 15 days. We have yet to establish the cut off time after which decompression therapy will have no beneficial effect.

On the other hand, it is also clear that there will be some cases where recompression therapy will not help. However, even so, a well planned and carried out rehabilitation programme can help to improve the patient's physical, social and mental state. It is because of this that we have adopted the team approach with the Department of Rehabili-

tation Medicine and various specialist departments of the Tan Tock Seng Hospital. We aim to give all the DCS patients the complete management that they deserve.

References

- 1 Tan ES and Balachandran N. Rehabilitation of Caisson's disease with spinal cord involvement. *Ann Acad Med Singapore* 1979; 8(1 Jan):
- 2 Giblin DR. Somatosensory evoked potentials in healthy subjects and in patients with lesions of the nervous system. New York: *Ann NY Acad Sci* 1964; 112: 93-142
- 3 Halliday AM and Wakefield GS. Cerebral evoked potentials in patients with dissociated sensory loss. *J Neurol Neurosurg Psychiat* 1963; 26: 211-219
- 4 Palmar AC, Calder IM and Hughes, J.T. Spinal cord degeneration in divers. *Lancet* 1987; ii: 1365-1366
- 5 How Jimmy. Rehabilitation experiences of paralysed diver in Singapore. In: *UMS Workshop*: Publication No 66. Bethesda, Maryland: Undersea Medical Society, 1985; 83-93

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BREATH-HOLD DIVING

Michael Davis

Introduction

There is a beautiful photograph by Flip Nicklin in the December 1984 issue of the National Geographic magazine of a sperm whale sounding. This epitomises for me the wonder and majesty of the diving mammals, that diverse group of animals of widely varying structure, function, habitat and behaviour who grace our oceans (Table 1). This paper briefly reviews the physiology of these animals and aspects of human breathhold diving, whilst the bibliography provides a selection from the literature rather than an exhaustive list.

Asphyxia is a progressive process which begins at the moment that external gas exchange ceases. The cessation of respiration leads to hypoxia, hypercapnia and acidosis, the

TABLE 1

SOME ADAPTATIONS TO APNOEIC DIVING IN AQUATIC MAMMALS

Respiratory

Tolerance of thoracic squeeze

lung collapse
elastic chest wall
mechanically tough tracheo-bronchial tree

Protection from decompression sickness

lung collapse (loss of gas exchange)
Decreased sensitivity to hypoxia and hypercapnia
Increased ventilatory/gas exchange efficiency

Cardiovascular

Blood shift into thoracic cavity
Venous spincters and sinuses
Large spleen
Diving Reflex CVS components

Biochemical

Increased blood (haemoglobin) and tissue (myoglobin) oxygen stores
Diving Reflex Switch to anaerobic metabolism

Hypothermia/Insulation

Efficient locomotion

triad of asphyxia. The successful mammalian diver can postpone the inevitable functional collapse that follows the cessation of breathing by virtue of three main mechanisms:

- 1 Enhancement of the oxygen stores in the body
- 2 Acid buffering of the products of metabolism
- 3 Circulatory reduction and redistribution, leading to metabolic conservation.

Regulation of these responses is essentially identical to that governing the protective reaction to asphyxia in terrestrial animals, the observed differences being quantitative rather than qualitative.

Scientists have approached the study of these remarkable animals' adaptation to asphyxia both in the laboratory and in their natural environment. Although some laboratory work has been criticised, field and laboratory research are, in fact, complimentary, and not conflicting. "Exploring what an animal is capable of is not the same as asking how it normally behaves."

Adaptations in diving mammals

Animal behaviour is determined by ecological constraints and physiological limits. In diving mammals these limits are defined by the amount of oxygen carried, the rate of oxygen consumption and the maximum pressure the animal is capable of withstanding. For instance, harbour seals have been recorded whilst diving under the Antarctic ice for more than one hour and to depths greater than 600 m. The physiological and anatomical adaptations involved vary considerably from species to species, each adopting different strategies. Therefore, not all the features described below are shared by all.

ANATOMY

Quite apart from the sleek, well-insulated body habitus that allows high swimming efficiency and protection from hypothermia, numerous anatomical features are present in the diving mammals to facilitate tolerance to the challenges of the underwater environment. Many have a floating, compliant rib cage, with a very obliquely inserted diaphragm. The respiratory tract tends to be short, wide and mechanically tough. In some whales, but not in pinnipeds, the respiratory tract can be occluded by special anatomical structures in the blow-hole, spiracle chamber and glottis. In some species these and other features may improve tolerance to chest compression ("thoracic squeeze") during deep dives.

Dolphins have terminal respiratory bronchioles that take part in gas exchange and the structure of the pulmonary microvasculature allows a reduced diffusion barrier. Also present are rete mirabile and arterio-venous shunts in certain organs, including the lining of the airways in the sperm whale. In the deep diving species, a very large inferior vena cava with a precordial sphincter, and a large spleen are present, whilst harbour seals also possess an aortic bulb. All these features may be important either for gas exchange or in the oxygen delivery to vital organs, but in many cases their contributions remain ill-understood.

LUNG VOLUMES

Long-duration diving whales have small lungs which collapse during dives and are not used as an oxygen store, e.g. the bottlenose whale has 25 ml/kg body weight. Short duration divers tend to have lung volumes similar to terrestrial mammals and dive following inspiration. Thus they appear to use the lungs as an oxygen store, e.g. man and porpoise have 70 ml/kg body weight. Seals dive following partial expiration which also results in lung collapse during descent. Although lung size may vary, diving mammals tend to have large tidal volumes, low end-expiratory volumes and a breathhold ("skip") breathing pattern. Minute ventilation is high during surfaced periods between hunting dives. For instance it has been measured at greater than 60 l/minute in the harp seal.

OXYGEN DELIVERY

Oxygen may be stored in the lungs, blood and muscle. These stores are considerably greater in diving mammals than terrestrial animals:

Weddell seal	60 ml/kg body weight
California sealion	40 ml/kg body weight
Man	20 ml/kg body weight

Haemoglobin

Adaptation of haemoglobin-oxygen affinity parallels the modifications in lung volumes in that, where the lung does not act as an oxygen store, oxygen affinity is low. This leads to maximal unloading of oxygen at the tissues (i.e. a shift of the haemoglobin-oxygen dissociation curve to the right). For instance, seals and some whales extract 7-10 vol% of oxygen from the blood, compared to man at around 5 vol%. In those animals in which the lungs act as a store, affinity tends to be high. This ensures maximal oxygen uptake in the lungs (i.e. the dissociation curve is shifted to the left).

Blood stores of oxygen are one to three times that of terrestrial mammals. Both blood volume and haemoglobin concentration may be high. In Weddell seals, the red cell mass and haemoglobin concentration actually increase during a dive by about 8 g/l/min during the first 10 minutes of the dive. This extra red cell mass probably comes largely from venous pooling and sequestration of oxygen-rich blood in the spleen which has the highest weight as a percentage of body weight of any reported mammal. It is probable that the precordial sphincter in the inferior vena cava periodically relaxes to give a pulsed delivery of this oxygen-rich blood.

Interestingly, in seals resting on the surface, arterial oxygen tension is actually lower (70-80 mm Hg) than most terrestrial mammals, but can be rapidly raised to over 110 mm Hg by hyperventilation just before the dive. During the dive, oxygen tension rises to a maximum of about 230 mm Hg and falls to 25-35 mm Hg by the end. The lowest in-dive oxygen tension recorded in Weddell seals in Antarctica was 18 mm Hg.

Myoglobin

Oxygen stores in myoglobin vary from one to nearly ten times that of terrestrial mammals. Like haemoglobin, this parallels the dive duration capabilities of the species concerned.

CARDIOVASCULAR MODIFICATIONS

As the animal dives deeper and the gas volume in the lungs is compressed, the rib cage gradually collapses, as has been dramatically illustrated in photographs taken at depth. In addition, there is a major shift in blood volume into the lungs to take up some of the additional space, thus preventing lung rupture. As well as this increase in pulmonary blood

volume, the coronary vascular blood volume tends to be high during bradycardia. However, coronary blood flow demonstrates large fluctuations from virtually no flow to periods of high perfusion during a dive. This may allow the myocardium to derive maximum benefit from both its oxidative and anaerobic metabolic reserves by permitting periodic wash-out of metabolic products, and reperfusion with oxygen-rich blood.

AEROBIC DIVING

The calculated aerobic breathhold times based on estimated body oxygen stores and oxygen uptake fit very well with metabolic evidence from free-diving seals. For instance, in the Weddell seal the aerobic breathhold limit is about 20-30 minutes which is the same as for this animal's usable oxygen stores under normal aerobic metabolic conditions. Observations on Weddell seals and several other species suggest that more than 90% of dives are within the aerobic limits so that metabolic requirements are satisfied by the body's oxygen stores with minimal increase in lactate.

Short aerobic dives have a brief recovery time since they require restoration of oxygen stores only. Anaerobic dives, however, require metabolism of lactate and other metabolic products that takes much longer to complete. A series of short duration dives increases hunting efficiency compared with a few long dives by allowing a much greater proportion of time underwater. For instance, in harp seals about 88% of the time is spent diving when feeding, with an average dive time of about five minutes. During such "bout" diving there is virtually no disturbance to metabolic homeostasis except the rise in haematocrit seen during dives.

THE DIVING REFLEX, ANAEROBIC DIVING

The diving reflex is triggered by face immersion and breath-holding. It is mediated by both the parasympathetic and sympathetic nervous systems to produce some intense physiological and biochemical changes (Table 2). The components of the diving reflex have been seen best in captive laboratory dives. However, field work with seals and dolphins has shown that this response is not an all-or-none phenomenon, but extremely variable. There is now strong evidence that diving mammals can anticipate the dive duration and control both the degree of bradycardia and the extent of blood flow redistribution needed for a given dive. Animals also anticipate surfacing and their heart rate is seen to increase shortly before the end of the dive.

Once the anaerobic threshold is reached, some major metabolic adaptations come into play. Muscle glycolysis is an impressive anaerobic machine even in non-divers, but the diving mammals have refined it in a number of ways:

- a) Steady state concentrations of a few glycolytic enzymes are high, so increasing the capacity to maintain NAD/NADH (Redox Potential) ratios under anoxic stress.

TABLE 2

COMPONENTS OF THE DIVING REFLEX

Bradycardia

Intense peripheral vasoconstriction

Reduced cardiac output

Redistribution of cardiac output to vital organs

Metabolic shift to anaerobic glycolysis

Decrease in body temperature due to decrease in metabolic rate

b) Concentrations of fructose-diphosphatase which is a key regulatory step in the glycolytic pathway are amongst the highest reported in animals. This appears to amplify the cyclic-AMP signal for glycolytic activation.

c) Pyruvate kinase activity is enhanced. This enzyme is regulatory to the glycolytic pathway in that it is highly sensitive to both feed-forward activation by fructose-1,6 diphosphate and feedback decrease by ATP, alanine and citrate. This is thought to help particularly in switching to and from the aerobic to the anaerobic state.

d) Very high titres of aspartate and alanine transferases are present. These enzymes are very important in the control of the Krebs cycle.

Diving mammals can tolerate high lactate loads in their tissues. Plasma lactate concentration does not rise much until immediately after the dive, when metabolic products are washed out of the non-perfused tissues once the diving reflex ceases. The amount of lactate produced increases exponentially as dive time goes beyond the aerobic limit. During long dives, seals are able to sustain work loads more than twice their maximum aerobic capacity with little or no increase in oxygen uptake. To support this, both skeletal and heart muscle are rich in myoglobin (see above) which has important buffering functions under anaerobic conditions.

The brain and the heart of diving mammals have considerable anaerobic capacities and can produce large quantities of lactate toward the end of a long dive. In addition, the brain of diving mammals has a much greater tolerance to decreased oxygen compared with terrestrial mammals. Cerebral integrity is retained down to oxygen tensions as low as 8-10 mm Hg, whereas in humans this is about 25-30 mm Hg.

Organs such as the kidney, have also been shown to tolerate asphyxia far better than those of terrestrial mammals. For instance, the rise in intracellular sodium and fall in potassium in anoxic harbour seal kidney slices is consistently less than in rat kidney, especially at low pH. Also, the seal kidney functions differently in that water conservation occurs with immersion unlike the diuresis seen in man.

Hochachka has written extensively on the molecular mechanisms of defense against hypoxia. He believes that three main mechanisms are important:

- 1 Arrest of oxidative metabolism and electron-transfer system functions (i.e. reduced metabolic rate)
- 2 Arrest of glycolytic activation
- 3 Arrest of ion-specific channel functions

He hypothesises that hypoxia-tolerance depends on an exceptionally tight regulation of energy demand and supply. Such close regulation of ATP turnover as the tissue becomes hypometabolic is only possible if at least one regulator signal switches down ATP utilisation and synthesis rates simultaneously. It is not known what this regulator might be nor even if it exists. This concept has been called the "membrane channel arrest" hypothesis.

DECOMPRESSION SICKNESS AND THE DIVING MAMMALS

There are several potential mechanisms for limiting nitrogen loading in the tissues during long deep breathhold dives by reducing the nitrogen stores in the lungs:

- a) Dive at low lung volumes
- b) Produce a profound fall in pulmonary blood flow early in the dive
- c) Allow lung parenchyma to become gas-free with compression, the residual volume shifting into the airways.

It is the latter mechanism for which there is most evidence. Recent work has been done on Weddell seals during dives averaging about 23 minutes duration and 230 m average depth. This has shown that plasma nitrogen tension rises rapidly during the early part of the dive, peaking at 2,000-2,400 mm Hg at about 40 m depth. At this depth (shallower than previously believed) the lungs collapse. Nitrogen tension then slowly falls to about 1500 mm Hg near to the time for surfacing. Thus the seal is protected from decompression sickness and nitrogen narcosis by limiting nitrogen uptake and redistributing nitrogen during dives. Part of this redistribution may be into the red cell mass entering the circulation from the spleen during the dive. This would also protect against oxygen toxicity.

Other factors could also play a part in protecting the animal from decompression sickness. For instance, repeti-

tive breathhold dives might force gas micronuclei into solution, thereby decreasing any tendency to bubble seeding. Finally, the possibility has been raised that the acoustic echo-location system of whales and dolphins, which is fat-rich, may provide an early-warning bubble detection system.

Human breath-hold diving

Free-diving is an ancient and widespread human activity. In communities such as the Ama of Korea and Japan it became part of the economic basis of society. In the twentieth century it has become a popular sport, whilst free diving for a living amongst indigenous peoples has generally either dwindled markedly or they have moved on to scuba techniques.

TABLE 3

DIVING DATA FOR MAN AND AND DIVING MAMMALS

	Breath-hold Time (min)	Maximum Depth (m)
Man	2-4	? 100
Dolphin	6	300
Orca	12	?
Weddell Seal	75	600
Sperm Whale	? 75	? 1000 +

When I first learnt to dive in the early 1960s, I was taught two concepts about breathhold diving. The first was that the maximum depth for human breathhold diving was about 33 m (100 ft). This was because, as the lung gas volume diminished with increasing pressure, a point was reached where lung tissue would rupture and pulmonary haemorrhage occur. This was called "thoracic squeeze".

We now know the above concept is not valid. Thoracic squeeze probably does not occur in breathhold diving in man under most circumstances because there is a major shift of blood volume into the pulmonary vascular bed with compression. The second concept of much more practical importance to man than thoracic squeeze was that dive duration was limited to only one or two minutes by "shallow water blackout".

The longest human breathhold times recorded on air are about 4 to 5 minutes. The deepest dives recorded are those of Robert Croft to 240 ft in the 1960s, and Jacques Mayol to well over 300 ft in the 1970s. The pattern of diving activities has been extensively studied for groups such as the Ama, but less so for intensive sport divers such as underwa-

ter hockey players or competitive spearfishermen. The Ama dive 30 to 45 times an hour for 30-45 seconds with 45-90 seconds rest between dives for many hours, spending 1/3-2/5ths of their total time underwater. In contrast, the dive duration for underwater hockey players over a 30-40 minute playing period, averages only 8-10 seconds per dive, but recovery periods are only 3-12 seconds. Thus they spend 2/5-2/3 of the total time underwater (personal observations).

Such efforts pale into insignificance compared to the diving mammals! However, many physiologists have been interested to observe whether human breathhold divers demonstrate any adaptations in the ways seen in the diving mammals. The classic studies on the Ama make absorbing reading and the reader is recommended the proceedings edited by Rahn.

FACTORS INFLUENCING BREATHHOLD TIME IN MAN

TABLE 4
CONDITIONS FAVOURING
LONG BREATHHOLD TIMES

Large O ₂ stores
Diving Reflex
Pre-breathhold hyperventilation
Increased diving depth
Thermoneutral immersion

LUNG VOLUME

Record divers all have big lungs. Geoff Skinner, Australian spearfishing champion for many years, demonstrated an air breathhold of 3 minutes 8 seconds during the talk at Port Vila. He has a vital capacity of well over seven litres. Ama were shown to have an increased vital capacity compared with non-diving women in their community, but as long as 20 years ago, Hong questioned whether this was due to diving per se or rather to physical fitness. Recent studies suggest the lungs of breathhold divers are indeed no bigger than other athletes. However, studies of breathhold times have been conflicting. Dry-land athletes and divers performed the same in one study, but the divers had longer times in another. Breathhold times in children correlate with age in proportion to their lung size.

Thus, our primary oxygen stores for diving are in the lungs, as for the short duration diving mammals.

HYPERVENTILATION

By decreasing the CO₂ tension, respiratory drive is decreased. The increase in alveolar oxygen concentration increases the oxygen stores in the lung slightly but only minimally increases oxygen content in the blood.

OXYGEN BREATHING

Oxygen breathing increases the oxygen stores in the lungs by up to several litres. Following oxygen breathing for three minutes, Geoff Skinner's breathhold time at the Port Vila meeting increased to just under ten minutes.

METABOLIC RATE (VO₂)

There is an inverse relationship between breathhold time and VO₂. However, oxygen consumption varies with the type of diving being undertaken. For instance, a dive to 5 m depth without swim-fins and lasting 30 seconds has a mean VO₂ of about 0.8 litres, whereas a 10 m dive for 30 seconds uses 1.2 litres O₂. Oxygen consumption is increased when the descent is active rather than passive. Swimfins reduce oxygen uptake slightly at comparable swimming speeds. In underwater hockey, a higher VO₂ is likely because of the high swimming speeds whilst breath-holding. Since the total oxygen stores of an average 70 kg man with normal lung size are only approx 1.5 L breathing air, unconsciousness is likely to occur in approx 80 seconds at a VO₂ of 1.0 l/min (about that of moderate fin swimming). There is no evidence in man of a switch to anaerobic metabolism with the induction of the diving reflex.

OXYGEN AND CARBON DIOXIDE CHANGES DURING BREATHHOLD DIVES

Changes in O₂ and CO₂ are very important in the control of breathing, in determining the "breaking point" for a breathhold, and in the aetiology of shallow water blackout.

a) Oxygen

During most of a dive the relationship between VO₂ and oxygen tension is linear. However, once the "alveolar stores" in the lungs are exhausted, the "blood stores" of oxygen are used and the relationship becomes non-linear with rapid desaturation of haemoglobin. Thus, the rate of fall of PaO₂ below 100 mm Hg is highly dependent on the haemoglobin concentration and on circulating blood volume.

The alveolar-arterial oxygen difference is similar throughout the dive at approximately 10 mm Hg. If an ascent is needed at the end, there is a rapid decrease in PAO₂ and in very long dives this could result in an actual reversal of oxygen transfer across the alveolar membrane resulting in even more rapid desaturation. Towards the end of long dives, in fact, mixed venous and

arterial oxygen tensions approach each other. In the many studies by Craig, Hong and Paulens, oxygen tension at the end of most dives tended to be around 60 mm Hg but was quite variable, and in some dives levels as low as 30 mm Hg were observed.

b) Carbon Dioxide

Changes in CO₂ during a dive are more complex than those for oxygen:

1 Approximately half the CO₂ enters the alveoli during a dive in the first few seconds as a consequence of the acute fall in the CO₂ tension (P_ACO₂). This is due to dilution into the large inspired volume immediately prior to the dive.

2 With the initial descent, the PACO₂ increases with lung compression to approximately 50-60 mm Hg. This leads to an increased P_aCO₂ and therefore a decreased gradient for tissue off-loading. Tissue off-loading could be further reduced by decreased muscle perfusion as part of the diving reflex as in diving mammals. However, there is no metabolic evidence for this in man (see below).

3 The alveolar volume cannot serve as a CO₂ store due to its rapid compression with descent. During a dive this rapid compression is analogous to a rebreathing test and equilibrium between alveolar and arterial PCO₂ should occur rapidly. Indeed, it does so after less than a minute, after which the alveolar-arterial CO₂ gradient may actually reverse and alveolar CO₂ fall slightly. During the remainder of a dive after the appear to be related to the length of the dive and is non-linear.

In Craig's studies, PACO₂ tended to be approximately 60 mm Hg suggesting tissue CO₂ tensions close to this. This could explain the subjective experience of approaching the breaking point immediately after a rapid descent. This sensation then passes, only to return towards the end of the dive. During ascent the urge to breathe is often relieved again as the P_ACO₂ falls, enhancing CO₂ off loading into the alveoli.

4 The PACO₂ at the end of a dive is much less than after an equivalent period of breathhold exercise on dry land. Values are typically 48-50 mm Hg, but in Paulev's study they were normal in many subjects. Hong calculated that during a 4-minute oxygen breathhold, the lung supplied 700 ml oxygen, but only gained 160 ml CO₂.

Why does the PACO₂ rise initially then plateau during a dive? Where is the CO₂ stored, since there is no evidence of a shift to anaerobic metabolism in man? Exercise studies at 30% of VO₂ max

suggest a storage capacity for CO₂ in the body of 1.83±0.55 ml/kg/hr, this is very large and is due to the high solubility of CO₂ in body tissues. Thus it would appear that the tissues act as the buffer for P_ACO₂ changes during a dive and as a store for CO₂.

c) Shallow Water Blackout

The non-linearity of the relationship between oxygen tension and blood oxygen content at the end of a breathhold dive is very important to understanding the mechanism of shallow water blackout. To illustrate this, let us consider the two dives in Figure 1. The first graph is derived from a dive in Shaeffer's earlier work. This illustrates a 9 m dive divided into three equal 20 second periods for descent, bottom time and ascent.

During descent the PaO₂ increases, then gradually falls during the working part of the dive. Then, on ascent, this fall becomes steeper. If, however, we look at oxygen saturation in the blood during this period, it remains nearly fully saturated for virtually the whole dive, only dropping toward the end of the ascent. In the first dive the O₂ saturation never reaches dangerous levels at which a diver might lose consciousness.

If we now extend this dive by a further 20 seconds working time (Figure 1), then at the end of one minute the P_AO₂ is still relatively high and the S_aO₂ only just starting to fall. However, because the lung oxygen stores are almost used up at this stage, the diver starts to draw on his blood stores which, as we said earlier, are of limited capacity. During this ascent, saturation falls far more precipitously and the point of loss of consciousness is reached very rapidly. On the graph this point is arbitrarily set at about 29 mm Hg or the P₅₀ for the normal haemoglobin-oxygen dissociation curve, since consciousness is lost in the range 25-30 mm Hg.

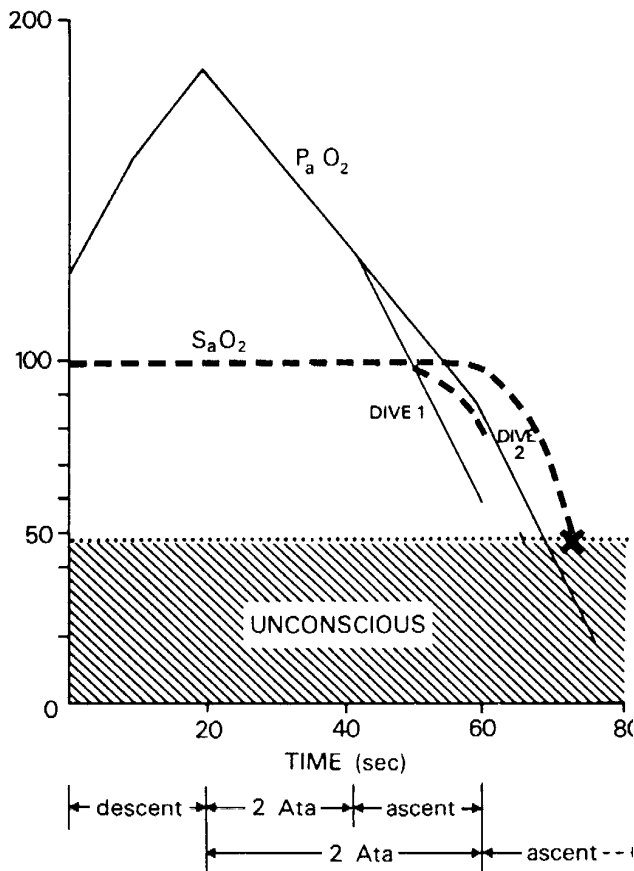
We are all aware that shallow water blackout does occur even without a descent/ascent dive pattern, particularly if respiratory drive is diminished by prior hyperventilation. What I wish to emphasise is the non-linear nature of the rate of change in oxygen saturation, and how narrow is the margin between a "safe" dive and shallow water blackout.

Bove has suggested that those with diminished carbon dioxide and hypoxic responses may be more prone to shallow water blackout. He believes this is partly because the initial decrease in heart rate with face immersion is later potentiated by hypoxia during long breath-holds.

OXYGEN AND CARBON DIOXIDE SENSITIVITY

There has been much work on the ventilatory responses to exercise, hypercapnia and hypoxia in athletes. These studies have demonstrated that the peripheral che-

FIGURE 1



The time course of changes in P_aO_2 and S_aO_2 during two breathhold dives to 9 m depth, one having a 20 seconds longer bottom time than the other. Note the steepness of the fall in S_aO_2 towards the end of the longer dive.

moreceptors in the carotid body are responsible for the respiratory compensations to the metabolic acidosis of exercise. For instance, if the carotid body is absent, both the rate of rise of ventilation and its peak level are diminished during graded exercise.

It is also known that there is marked individual variability in the responses to hypercapnia and hypoxia. High performance athletes tend to show decreased responses at rest, suggesting decreased chemoreceptor function. There appears to be a major genetic component to this in endurance athletes and swimmers, with no evidence that it is altered by physiological training.

The ventilatory response to exercise appears to parallel the hypoxic response at both low and high work levels and the hypercapnic response at heavy exercise. Also any

fall in ventilation with hyperoxia is proportional to the hypoxic response of the individual. It has been reported that breathhold time is inversely proportional to the CO_2 response ($r = -0.89$) in dry-land athletes, but such a relationship has not been the case in studies of breathhold divers.

DO DIVERS HAVE ALTERED SENSITIVITY TO CO_2 AND O_2 ?

It has been suggested for many years that divers may have reduced chemoreceptor sensitivity. What is the current evidence for this?

1 Of the two types of Ama divers, only the Funado have shown any evidence of decreased carbon dioxide responses, yet their breathhold times were the same as non-diving controls. There has been no convincing evidence of a decreased hypoxic response in the Ama.

2 Submarine-escape tower divers: One NMRI report suggested that the CO_2 response decreases during training in successful diving candidates. Schaeffer reported a low CO_2 response in escape tower safety divers, which increased after a five week lay off.

3 Melamed and Kerem reported that O_2 diving did not alter peripheral oxygen chemoreceptor response and there was no change in hypoxia response compared to controls. A sub-group of scuba divers in their study were reported to have a decreased CO_2 response.

4 Several studies have shown low CO_2 responses in underwater hockey players, but this has not consistently been less than for high-class dry-land athletes. Only a small effect of training was seen in one unpublished study (McKenna and Green, personal communication). This suggests that, overall, breathhold divers may have a diminished CO_2 response, but whether this is modifiable with training or whether they demonstrate other differences in ventilatory responses is not known. We have recently been studying in our laboratory the effects of breathhold training on the hypercapnic and hypoxic responses in underwater hockey players, but our data have not yet been analysed.

In summary, no clear relationship between peripheral chemoreceptor sensitivity and breathhold times has been demonstrated in man.

OTHER FACTORS

a) Water temperature: With decreasing water temperature there is a decrease in breathhold time proportional to the increased metabolic rate of cold immersion. This is in spite of an enhanced diving bradycardia in cold water. In other words, metabolic rate and the diving reflex are physiologically independent in man, rather than linked as in the diving mammals.

- b) The diving bradycardia (see below): There is no prolongation of breathhold time as diving bradycardia is elicited.
- c) Cortical stimuli: In man, motivation is probably the strongest factor in breathhold duration to the conventional "breaking point" (see below).

BREATHHOLD AND FACE IMMERSION - THE "DIVING REFLEX" IN MAN

Breathhold in man is characterised by bradycardia (Figure 2), a progressive rise in blood pressure, stroke volume, cardiac output and peripheral vascular resistance. There is no evidence for significant metabolic changes or major oxygen-conserving responses such as occur in diving mammals. Therefore, the evidence to date is that habituated divers such as the Ama and underwater hockey players do not show enhanced defences against asphyxia.

THE BREAKING POINT

The drive to break a breathhold is both chemical and mechanical.

Two end-points have been used in past studies:

- 1 The conventional breaking point is taken as the time to the first inhalation.
- 2 Physiological breaking point is the time to the onset of involuntary ventilatory muscle activity (usually measured using electromyography). This appears to be PCO₂ and VO₂ dependent, non-subjective and therefore a more precise end-point for laboratory studies.

Several components are important, including the chemoreceptor sensitivity to carbon dioxide and oxygen, signals from pulmonary and chest wall stretch receptors, and motivation. Conventional breathhold time is prolonged by rebreathing in the absence of alveolar O₂ and CO₂ changes. This shows the importance of signals from the chest wall and lung stretch receptors.

The "Diving Reflex" in man is:

- 1 Independent of posture
- 2 Relative to the level of body immersion
- 3 Proportional to lung volume at which breath is held
- 4 A function of intrapleural pressure
- 5 Potentiated by face immersion
- 6 Inversely proportional to water temperature
- 7 Enhanced by dynamic exercise, but not proportional to level of fitness (including Ama divers)
- 8 Enhanced in swimmers compared with non-swimmers
- 9 Diminished by wearing facemask and hood in warm but not cold water
- 10 The same in children in whom breathhold time is long enough to reach full diving bradycardia (approx 25 seconds) as it is in adults.

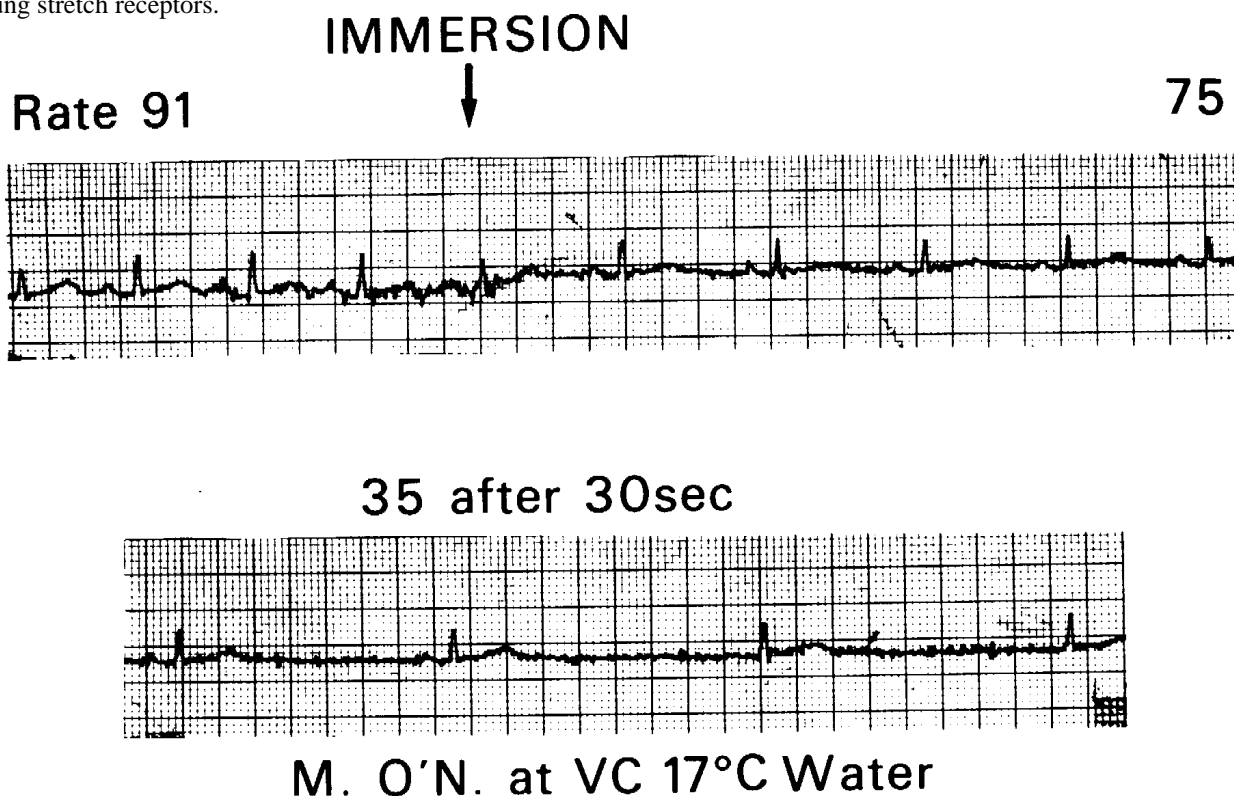


Figure 2 The human diving bradycardia with breathhold and face immersion. Note that the maximal fall in heart rate takes about 30 seconds to be achieved.

In summary, man is a poor diver, reliant on his lung oxygen stores during a breathhold. Whilst the diving bradycardia occurs in man, it is not accompanied by the important metabolic and other circulatory changes that characterise the full "diving reflex" in many diving mammals. In addition, the evidence for physiological adaptation to habitual breathhold diving is inconclusive. An understanding of the non-linear relationship between oxygen tension and content in the blood helps to explain the narrow line between a "safe" dive and one leading to shallow water blackout. Clearly, there is much research yet to be done in this fascinating field of environmental physiology.

UNDERWATER HOCKEY

Underwater hockey is a team water sport, with six players per side in the water, played on the bottom of a 1.8-2.5 m deep swimming pool with swimfins, mask and snorkel. A plastic covered lead or brass disc-shaped puck is pushed or flicked along the bottom of the pool with a short roughly triangular-shaped single-handed stick. A goal is scored when the puck is played into a shallow trough or goal at either end of the playing area. The game is played underwater during repeated breath-holds, and lasts 30-40 min.

At international level, it requires an extremely high level of fitness and breathhold diving skills. However, at all levels of competition it provides extremely good exercise for scuba divers, maintaining water skills and fin swimming fitness. It may thus be regarded as ideal exercise for maintaining scuba diving fitness in all age groups (there are now significant numbers of players over the age of 40 participating very successfully in the sport). Although it obviously will not appeal to all, I believe it should be recommended to divers as an excellent group activity for the maintenance of water skills.

Recommended Reading

- Craig AB and Harley AD. Alveolar gas exchanges during breathhold dives. *J Appl Physiol* 1968; 24: 182-189.
- Craig AB and Med WL. Oxygen consumption and carbon dioxide production during breathhold diving. *J Appl Physiol* 1968; 24: 190-202.
- Davis FM, Graves MP, Guy HJB, Prisk GK and Tanner TE. Carbon dioxide response and breathhold times in underwater hockey players. *Undersea Biomed Res* 1987; 14: 527-534.
- Elsner R. Perspectives in diving and asphyxia. *Undersea Biomed Res* 1989; 16: 339-344.
- Godfrey S and Campbell EJM. The control of breathholding. *Resp Physiol* 1968; 5: 385-400.
- Hill RD et al. Heart rate and body temperature during free diving of Weddell seals. *Am J Physiol* 1987; 253: R344-R351.
- Hochachka PW. Brain, lung and heart functions during

- diving and recovery. *Science* 1981; 212: 509-514.
- Hochachka PW. Molecular mechanisms of defense against oxygen lack. *Undersea Biomed Res* 1989; 16: 375-379.
- Hochachka PW and Storey KB. Metabolic consequences of diving in animals and man. *Science* 1975; 187: 613-621.
- Hong SK, et al. Alveolar gas exchanges and cardiovascular functions during breathholding with air. *J Appl Physiol* 1971; 30: 540-547.
- Hong SK, Moore TO, Seto B, Park HK, Hiatt WR and Bernauer EM. Lung volumes and apnoeic bradycardia in divers. *J Appl Physiol* 1970; 29: 172-176.
- Gooden RA, Lemna RG and Pym J. Role of the face in the cardiovascular response to total immersion. *Australasian J Exp Biol Med Sci* 1970; 48: 687-690.
- Kawakami Y, Natelson BH and Du Bois AB. Cardiovascular effects of face immersion and factors affecting diving reflex in man. *J Appl Physiol* 1967; 23: 964-970.
- Kooyman GL, Castellini MA and Davis RW. Physiology of diving in marine mammals. *Ann Rev Physiol* 1981; 43: 343-356.
- Kooyman GL, Wahrenbrock EA, Castellini MA, Davis RW and Sinnett EE. Aerobic and anaerobic metabolism during voluntary diving in Weddell seals: Evidence of preferred pathways from blood chemistry and behaviour. *J Comp Physiol* 1980; 138: 335-346.
- Leith DE. Adaptations to deep breathhold diving: respiratory and circulatory mechanics. *Undersea Biomed Res* 1989; 16: 345-354.
- Lin YC, Lally DA, Moore TO and Hong SK. Physiological and conventional breathhold breaking points. *J Appl Physiol* 1974; 37: 291-296.
- Lundgren CEG. Breathhold diving mammals. In *Hypoxia, exercise and altitude: Proceedings of the Third Banff International Hypoxia Symposium*, Alan R Liss Inc, New York, 1983; 365-381.
- Melamed Y and Kerem D. Ventilatory response to transient hypoxia in O₂ divers. *Undersea Biomed Res* 1988; 15: 193-201.
- Paulev P-E. Cardiac rhythm during breathholding and water immersion in man. *Acta Physiol Scand* 1968; 73: 139-150.
- Rahn H. Editor, *Physiology of breathhold diving and the Ama of Japan*. NAS-NRC Publication 1341, Washington DC, USA, 1965.
- Sherman D, Eilender E, Shefer A and Kerem D. Ventilatory and occlusion-pressure responses to hypercapnia in divers and non-divers. *Undersea Biomed Res* 1980; 7: 61-74.
- Snyder GK. Respiratory adaptations in diving mammals. *Resp Physiol* 1983; 54: 269-294.
- Song SH, Kan DH, Kang BS and Hong SK. Lung volumes and ventilatory responses to high CO₂ and low O₂ in the Ama. *J Appl Physiol* 1963; 18: 466-470.
- Sterba JA and Lundgren CEG. Breathhold duration in man and the diving response induced by face immer-

sion. *Undersea Biomed Res* 1988; 15: 361-375.
 Zapol WM. Diving adaptations of the Weddell seal.
Scientific American, 1987; 256 (June): 80-85.

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EDITED QUESTION TIME

Vanessa Haller

How do you feel about asthmatics playing underwater hockey as an alternative to scuba diving?

M Davis

If you want a let out in your response to these patients, swimming is an excellent sport for asthmatics. I cannot think of a reason why underwater hockey should not necessarily be good for them as well. Anybody who is in the water is at risk of aspiration precipitating an asthmatic attack. However, there is good evidence that swimming and water sports apart from scuba diving are often very good for asthmatics. I see no logical reason why asthmatics should not play underwater hockey. The water is not deep, up to 8-9 feet, it is warm and not likely to give cold precipitation of asthma. You have the other advantage of doing competitive exercise with high endogenous adrenalin levels, keeping the airways open! Certainly our top player in Christchurch is a moderately severe asthmatic. He actually controls his asthma by playing underwater hockey and swimming. If he lays off he is into the Ventolin inhaler 3-4 times daily. If he plays regularly he only occasionally needs his inhaler.

V Haller

It is good because it gives them an outlet for their aggression. Hating themselves for having asthma at that age.

V Brand

It is a pity to make a blanket condemnation of hyperventilation. Most spearfishermen and breathhold divers take 3-4 deep breaths before they dive. If they do not, they do not know what they are missing. The dangerous ones are when you seek kids who hyperventilate for a minute or two in order to compete with their peers.

Continued on page 176




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M A G A Z I N E

M Davis

Hyperventilation is probably physiologically advantageous because reducing PaCO₂ increases the PaO₂, slightly improving the overall oxygen stores. The problem is that there are grades of hyperventilation. A few breaths will lower the PaCO₂ to the low 30s whereas after 30 seconds it is down into the teens. The difference between the two in terms of oxygen stores is minimal but the difference in terms of drive for respiration is major. The longer hyperventilation period is much more likely to result in shallow water blackout. If you are determined enough, you do not need to hyperventilate to have shallow water blackout. It is always a potential risk in breathhold diving. There have been some blackouts in underwater hockey players. The RLSS in New Zealand has been concerned about this, but I have not actually heard of any cases during a match. My feeling is that the metabolic rate at which one is working is so high that you are almost certainly retaining CO₂ during a game.

P Chapman-Smith

Assuming constant depth, do you see any problems arising from skip breathing on scuba, apart from headaches?

M Davis

Several potential problems arise. If it is a deep dive skip breathing will contribute to narcosis. It may contribute to an enhanced risk of DCS. Yes, there are potential risks but it is very hard to get out of the habit.

G Olsen

You said that the oxygen desaturation was important. I have always thought the falling PO₂ with expansion of the lungs on ascent, combined with exhalation was the cause of shallow water blackout.

M Davis

The point I was making was that the relation between tension and saturation is non linear because of the shape of the haemoglobin dissociation curve. What becomes critical is where you are drawing the oxygen from at a given stage in the dive. If you are still drawing from the primary store in the lung, then you probably have a sufficient reserve for that not to be a problem. As soon as you are drawing from the remaining oxygen stores in the blood, which are very small, then the rate of fall of saturation becomes very rapid. So you are precipitated into a hypoxic episode very quickly. The critical thing is where you have used up your lung oxygen stores. There is still oxygen in the lung but on ascent there may be a reverse gradient from the blood and mixed venous and arterial PO₂ will be the same. Exhalation will hasten the fall of saturation. This is why the divers with big lungs are a lot safer than people with small lungs as they have much greater oxygen storage capacity.

G Blackburn

How do you overcome the initial dyspnoea that everybody gets as they descend?

G Skinner

People usually submit to this and say they cannot breathhold. There are many mechanisms like swallowing, exhaling a small volume and so on but you adapt and after a week you can extend your depth and time remarkably. None of us practise breathhold diving enough now to develop the skills. Underwater hockey is great training for breathhold diving.

A Santos

You mentioned that deep diving mammals may dive in exhalation. Is there any evidence that the lung may collapse to the point where it becomes a solid organ?

M Davis

I do not think anybody knows the answer to that fully. The bronchi in these animals are rigid unlike ours which tend to collapse, so it may well be the alveoli and respiratory bronchioles do collapse totally. There may be some interesting things about their surfactant that we do not know. I would think that they do collapse, and the residual gas volume lies in the small dead space volume of the rigid airways. The indirect evidence that this does occur is the measurement of blood nitrogen levels which show as the animal goes deeper the tensions fall which means that beyond 40 m they are not taking up any further nitrogen from the lung so there is no airspace for gas exchange to continue. This implies the alveoli have collapsed.

P Chapman-Smith

Can I make a comment that the early break point does not really relate to CO₂ levels. I think it relates to higher cerebral control and the change of breathing patterns in the elastic recoil from the lung volume receptors. The normal constant barrage of stimuli to the CNS and you notice a change in breathing pattern. This does not change with training or relaxation techniques. After about 30 seconds you feel all right.

M Davis

This is why in the laboratory you really need to measure the physiological break point, the re-emergence of spontaneous activity in the musculature, because it is not subject to voluntary control.

D Davies

I think the idea of adaptation is perfectly apt. When I was abalone diving most of our work was done on snorkel because we were not working in any more than 20 feet of water. What I found to be the most expedient way to work was to take 2-3 deep breaths, go down to work for 35-40 seconds and you could do this for hours on end. There were some ab-divers who would go down for a couple of minutes but they would peter out before the shorter time divers, who could work for hours. You are working on the aerobic mechanism rather than anaerobic, just like the diving mammals.

ARTICLES OF INTEREST REPRINTED FROM OTHER JOURNALS

LUNG FUNCTION REFERENCE VALUES FOR FEV₁, FEV₁/FVC RATIO AND FEF₇₅₋₈₅ DERIVED FROM THE RESULTS OF SCREENING 3788 ROYAL NAVY SUBMARINERS AND SUBMARINER CANDIDATES BY SPIROMETRY

G.J. Brooks, R.J. Pethybridge and R.R. Pearson

Introduction

In 1975 spirometry was instituted as a measure of respiratory fitness in the screening of Royal Navy submariners and divers. As the members of both groups are likely to be subjected to environmental pressure change, it has been considered important to detect abnormalities of lung function and structure that might precipitate pulmonary barotrauma and lead to potentially fatal cerebral arterial gas embolism (CAGE).¹⁻³ Identical spirometric testing was later introduced by the Health and Safety Executive for commercial divers subject to the Diving Operations at Work Regulations (1981).

Clinical opinion at the time of institution of the screening test was that dependent upon age, an FEV₁/FVC ratio of less than 70 to 80% suggested abnormal lung function and warranted further investigation. Accordingly, the main screening criterion adopted was that candidates for both submarine escape training and diving should achieve an FEV₁/FVC ratio of at least 75% initially and 70% on follow-up screening. Lower values may be accepted if the FVC is large and the FEV₁ normal. This rule has the advantage of simplicity, which is ideal for the purpose of mass screening. However, there has been little evaluation of the predictive value of the test as an indicator of risks for pulmonary barotrauma.

As a first step towards validation, a study was set up at HMS DOLPHIN and the Institute of Naval Medicine (INM) to determine whether the limiting criteria adopted are appropriate for the population to which the test is applied. This report details how results of screening have been used to construct a database from which reference values for the measured lung function variates have been calculated and their clinical value assessed. In order to maintain consistency within the study, it was decided that both a single spirometer and location should be used. Although only screening submariners, the Submarine Escape Training Tank (SETT) was chosen because of its large throughput of candidates, more than 1,000 RN submariners and trainees per year and the opportunities afforded for close medical supervision. A spirometer (Vitalograph), equipped with an interface to an Apple II computer, was installed at SETT and used to collect data, calculate and store results. Analysis was conducted at the Statistics Department of INM.

Subjects

The study population included all Caucasian submariners and submariner candidates presenting to SETT for screening between late 1983 and mid-1986. The trainees attending SETT are generally young males between the ages of 17 and 35. Before undertaking pressure work they must comply with the normal fitness requirements of the Royal Navy¹ and will have passed a submarine medical examination which includes spirometry. A three year limit was placed on the study as this is the requalification interval after which submariners must return to the SETT for further screening and training.

Methods

Software, including machine code sub-routines, were written for the computer at SETT in order that the following minimum data set could be collected from each candidate; age, height and weight (in underpants and shoes), smoking habit, history of recent upper respiratory tract infection (URTI), best FEV₁, best FVC and best FEF₇₅₋₈₅. The minimum data set was chosen to meet the purposes of screening and the management requirement that testing could be performed at least as rapidly as had been achieved before introduction of the computer.

Under the close supervision, and with the direct encouragement of one of two specifically trained medical assistants, each candidate, standing and wearing a nose clip, performed at least three good blows into the spirometer. "Good blows" were defined as closely grouped curves produced with apparent maximum effort. The computer recorded data points from each forced expiration for ten seconds after which it sounded a "beep". Once the test had been performed satisfactorily, the computer would calculate, store and display the best FEV₁ from any curve, the best FVC from any curve, the FEV₁/FVC ratio and the best FEF₇₅₋₈₅ using the points taken from one curve. Back-extrapolation was used to determine the start of the test and all lung function results were converted to BTPS.

Candidates attending for screening were excluded from the study if they admitted previous testing on the computerised spirometer or were suffering with an URTI. The accuracy of the software and interface were monitored regularly by random selection of spirometry tracings for manual calculation.

Throughout the study the spirometer received regular maintenance and calibration checks. During two weeks, mid-study results from the machine were compared with those obtained by an independent physiologist who re-tested

the screened candidates, within half an hour, using separate equipment.

STATISTICAL METHOD

The relationship between the measured lung function variates (LFV) and factors such as age and height of the form:

$$LFV = \beta_0 + \beta_1 \cdot \text{Height} + \beta_2 \cdot (\text{Age}) + \beta_3 \cdot (\text{Age}^2) \quad (1)$$

have been estimated for each smoking habit category by least squares analysis. Statistical tests have been conducted to assess if any parameters⁴ are unnecessary, and if the relationships for the smoking categories are identical, parallel or otherwise different. The standard deviations (σ) about the regression relationships and the proportion (R^2) of the total variation accounted for by the equations have been estimated.

Results

The results obtained from the computerised spirometer were found to be consistently accurate when compared with an independent spirometer, calibrated syringes (less than 50 ml difference) and manual result calculation from the tracings.

At the time of analysis, the database contained information from the screening of 3,788 Royal Navy submariners and submariner candidates. Of these, 1,488 declared that they had never smoked, 603 were ex-smokers, 592 admitted to light smoking habit (less than 10 cigarettes per day) and 1,105 to heavy smoking. The cumulative distributions by age for the smoking groups are shown in Figure 1. Light smokers have been omitted for clarity.

The estimated parameters in equation 1 for the measured lung function variates are given in Table 1. Information is given for each smoking habit category and is combined in the "all" group.

For both FEV₁ and FVC, the linear height and quadratic age relationships were found to be different for the smoking categories. The difference in predicted values with age and smoking habit are shown graphically for a man of average height (177 cm) in Figure 2. The variations with age between predicted values for non-smokers of heights 167 cm and 187 cm are illustrated in Figures 3 and 4. Attained FVC increases with height and also increases with age to the mid-twenties, then declines. FEV₁ also increases with height. There is an increase in FEV₁ with age to the early twenties before a decline that appears at an earlier age than for FVC.

The FEV₁/FVC ratio relationships do not account for much of the total variation ($R^2 = 0.09$ for "all" smoking groups). The relationships corresponding to the smoking

FIGURE 1

Cumulative age distributions for smoking groups

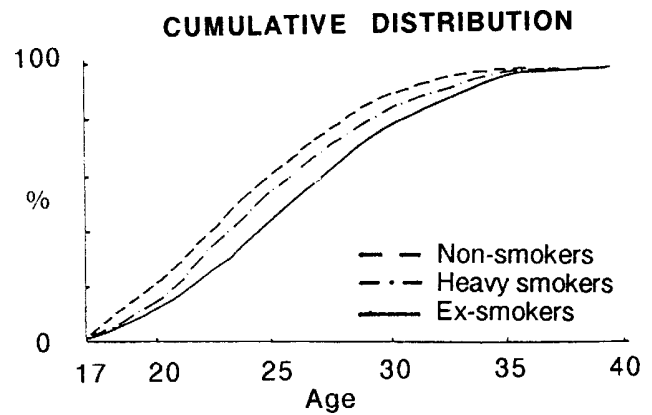


FIGURE 2

Reference values of FEV₁ and FVC for male non-smokers and heavy smokers of height 177 cm.

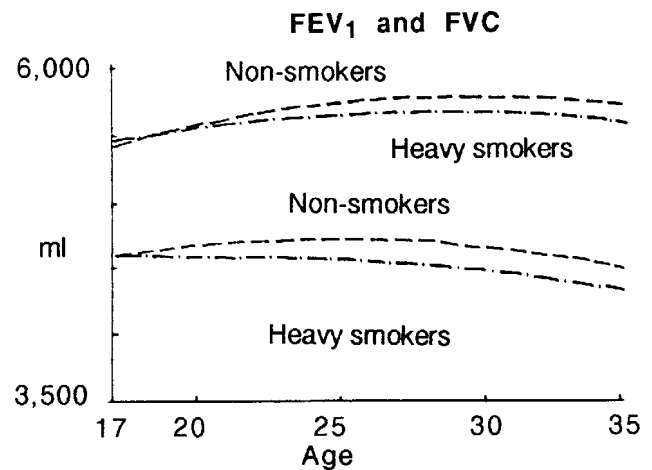


FIGURE 3

Reference values of FEV₁ for male non-smokers of heights 167 cm and 187 cm.

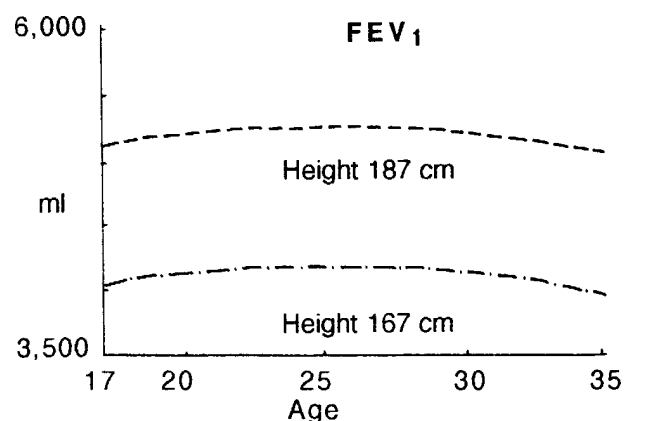


TABLE 1
ESTIMATED PARAMETERS OF LUNG FUNCTION, AGE AND HEIGHT RELATIONSHIPS

Lung Function Variate (LFV)	Subgroup	No. in Sub-group	LFV	Average Age (yrs)	Height (cms)	β_0	β_1 (height)	Parameter Estimates			$\hat{\sigma}$	R ²
								β_2 (age)	β_3 (age ²)	$\hat{\sigma}$		
FEV ₁ (ml)	Non-smoker	1488	4659	23.6	176.8	-6037	53.65	101.1	-2.029	482	0.36	
	Ex-smoker	603	4607	25.7	176.8	-3704	45.32	23.4	-0.570	478	0.30	
	Light smoker	592	4633	23.6	177.4	-5076	50.00	87.0	-2.072	498	0.36	
	Heavy smoker	1105	4522	24.6	176.6	-4293	47.65	45.7	-1.144	500	0.31	
	All	3788	4607	24.2	176.8	-4963	50.44	61.6	-1.370	493	0.33	
	Non-smoker	1488	5630	23.6	176.8	-8590	69.98	133.0	-2.223	544	0.44	
	Ex-smoker	603	5663	25.7	176.8	-6648	62.32	80.3	-1.120	527	0.38	
	Light smoker	592	5671	23.6	177.4	-7077	63.29	125.3	-2.454	565	0.39	
	Heavy smoker	1105	5571	24.6	176.6	-7021	64.18	92.5	-1.1611	540	0.38	
	All	3788	5625	24.2	176.8	-7519	66.37	100.8	-1.687	546	0.40	
% FEV ₁ FVC	Non-smoker	1488	83.0	23.6	176.8	104.51	-0.0776	-0.3322	.	6.21	0.07	
	Ex-smoker	603	81.5	25.7	176.8	105.25	-0.0776	-0.3908	.	6.00	0.12	
	Light smoker	592	81.8	23.6	177.4	93.51	-0.0180	-0.3614	.	6.04	0.09	
	Heavy smoker	1105	81.3	24.6	176.6	105.00	-0.0805	-0.3859	.	6.06	0.10	
	All	3788	82.1	24.2	176.8	103.25	-0.0690	-0.3713	.	6.10	0.09	
Loge FEF ₇₅₋₈₅	Non-smoker	1488	0.59	23.6	176.8	-1.053	0.0126	-0.0249	.	0.34	0.16	
	Ex-smoker	603	0.47	25.7	176.8	-0.565	0.0103	-0.0306	.	0.35	0.22	
	Light smoker	592	0.53	23.6	177.4	-1.424	0.0150	-0.0303	.	0.33	0.26	
	Heavy smoker	1105	0.45	24.6	176.6	-0.544	0.0103	-0.0335	.	0.34	0.24	
	All	3788	0.52	24.2	176.8	-0.879	0.0120	-0.0310	.	0.35	0.21	

habit categories can be summarised by parallel regression planes in the age and height space. The age-squared term was found to be negligible in terms of reducing the total variation. The summary equations are given in Table 1. Illustration of the variation of predicted values with age and smoking habit category are given for men of average height in Figure 5 and for non-smokers of heights 167 cm and 187 cm in Figure 6. The FEV_1/FVC ratio decreases as height and age increase.

The FEF_{75-85} data were not normally distributed and a logarithm transformation was used prior to the estimation of equation 1. The age-squared term reduced the variation about the regression by a negligible amount and has been omitted. The difference in predicted values with age and smoking habit are shown for a man of 177 cm in Figure 7 and non-smokers of heights 167 cm and 187 cm in Figure 8. Log FEF_{75-85} decreases with age and increases with height.

The effects of smoking on the lung function variates measured has been illustrated in the Figures 2, 5 and 7, by a comparison of the two largest groups, non-smokers and heavy smokers. Non-smokers were, on average, able to produce greater values of FEV_1 , FVC, FEV_1/FVC ratio and FEF_{75-85} than heavy smokers of the same height and age.

Discussion

A major advantage of this study over others has been the consistency of data collection.⁵ The methods used have followed the American Thoracic Society recommendations for spirometry.⁶ The spirometer was subjected to regular checks and was operated by two trained medical assistants using a standardised technique. Human error in graph interpretation was eliminated by electronic measurement of the volume changes (approximately 8,000 points were recorded over ten seconds from each forced expiration). The vast majority of Naval personnel attending the SETT were motivated to produce good lung function results in order to be allowed to continue with their training.

The database consisting of 3,788 results of screening represents possibly the largest most consistent source of lung function data for "healthy" Caucasian males, without the symptoms and signs of respiratory disease, between the ages of 17 and 35. The data has been analysed by smoking habit category. The cumulative age distribution, Figure 1, illustrates that there are few age range or distribution differences between the categories.

In interpreting the results, it is assumed that the submariners and candidates whose results have been included in the database are representative of the population that is to be screened. A possible source of bias arises from the requirement that submariner candidates be pre-screened by spirometry before attending SETT. If large numbers of candidates had been rejected on the results of spirometry at

FIGURE 4

References values of FVC for male non-smokers of heights 167 cm and 187 cm

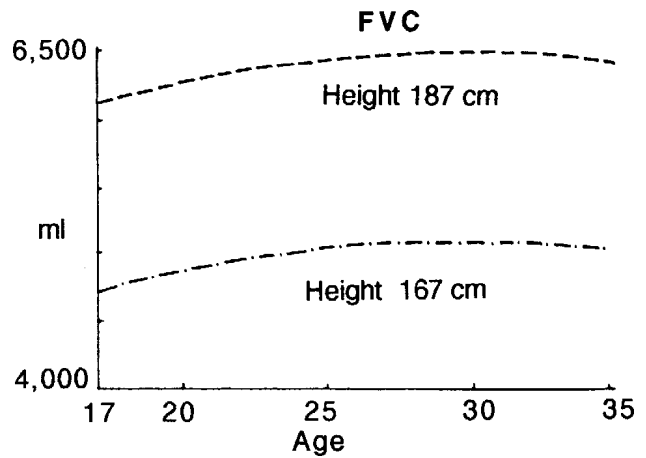


FIGURE 5

Reference values of FEV_1/FVC for male non-smokers and heavy smokers of height 177 cm.

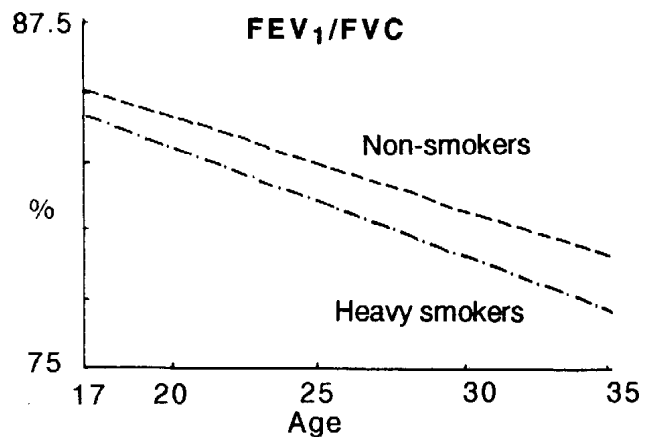


FIGURE 6

Reference values of FEV_1/FVC for male non-smoker of heights 167 cm and 187 cm.

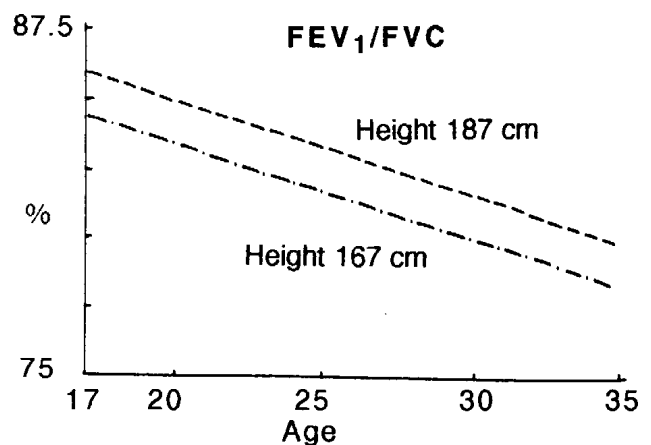


FIGURE 7

Reference values of FEF_{75-85} for male non-smokers and heavy smokers of height 177 cm

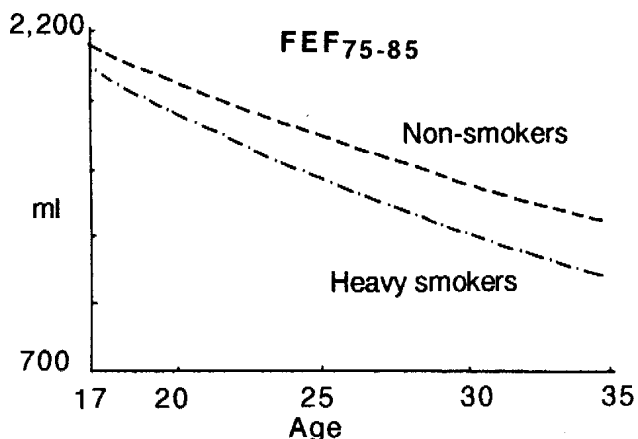


FIGURE 8

Reference values of FEF_{75-85} for male non-smokers of heights to 167 cm and 187 cm.

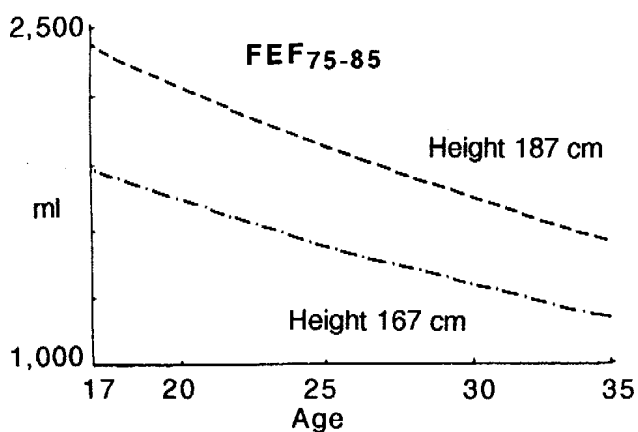


FIGURE 9

Comparison of reference values of FEV_1 for male non-smokers of height 177 cm with values of Knudsen⁷, Morris⁸, and Paoletti⁹.

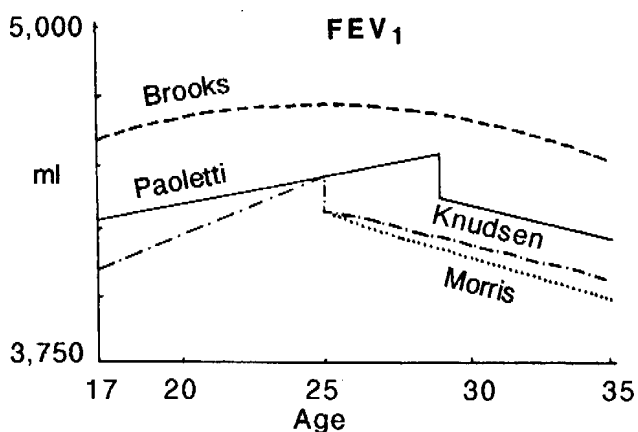


FIGURE 10

Comparison of reference values of FVC for male non-smokers of height 177 cm with values of Knudsen⁷, Morris⁸, and Paoletti⁹.

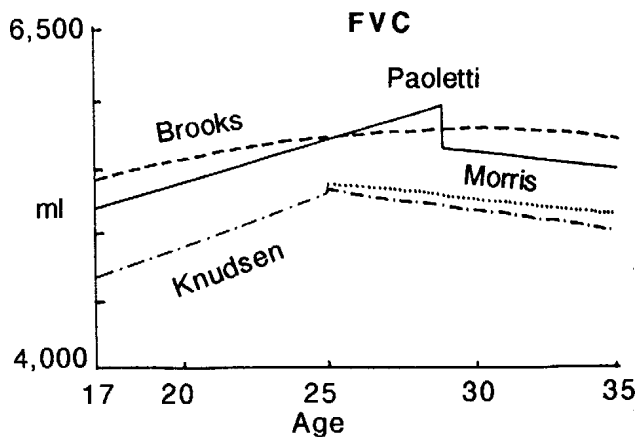


FIGURE 11

Comparison of reference values of FEV_1/FVC for male non-smokers of height 177 cm with values of Knudsen⁷, Morris⁸, and Paoletti⁹

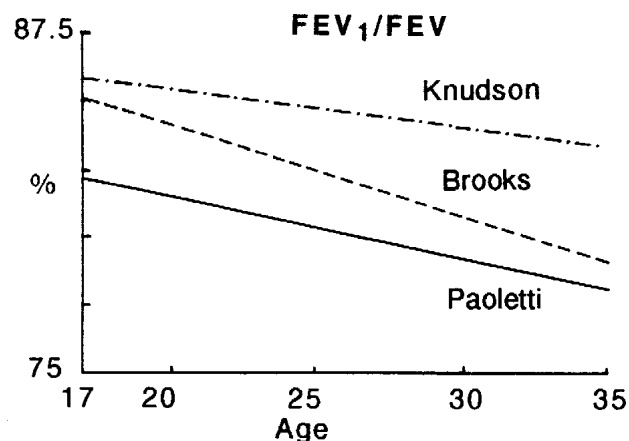


FIGURE 12

Comparison of reference values of FEF_{75-85} for male non-smokers of height 177 cm with values of Morris¹⁰ and Paoletti⁹.

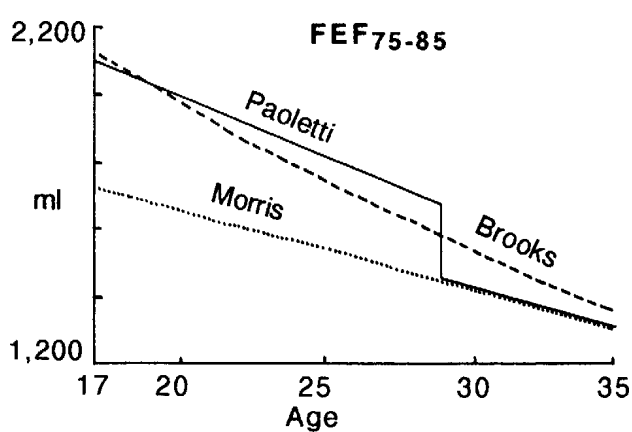
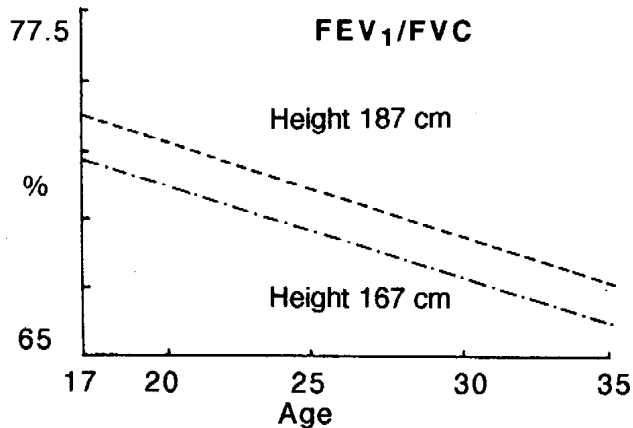


FIGURE 13

Predicted lower bound of 95% confidence interval for FEV₁/FVC in male non-smokers of heights 167 cm and 187 cm.



this stage, then the database would be biased towards high FEV₁/FVC ratios. It is policy that those candidates who are unable to obtain an FEV₁/FVC ratio of 75% on pre-screening but who are otherwise fit be referred to INM for further lung function investigation. During the data collection phase, twelve candidates referred for further lung function testing, for any reason, after pre-screening, were rejected and do not appear in the database. It is concluded from this, that minimal bias has been introduced by their exclusion. In fact, the great majority of candidates who were unable to meet the spirometric standard first failed on attending SETT. Review of previous records reveals that in many cases this was due to technical errors at pre-screening spirometry. It remains possible that bias has been introduced as an unknown number of personnel, who were suspected of having abnormal lung function at preliminary screening have been excluded from submarine training without having been referred to INM for physiological testing. If this selection process has disproportionately reduced the numbers of those with low FEV₁/FVC ratio in the database then it might be expected that the age and height specific predicted ratio values of this study would be higher than those of previously accepted standards. In fact, the predicted FEV₁/FVC ratio values from this study fall between those of Knudson⁷ and Morris⁸, suggesting that any exclusion bias is minimal. The predicted values from this study, for non-smokers, have been compared with others in Figures 9 to 12. The values of FEV₁ and FVC have been found to be greater, for each age, than predicted by Knudson⁷ or Morris.⁸ The values of FVC and FEV₇₅₋₈₅ calculated in the careful study by Paoletti⁹ are close to those predicted from this study. The contrast can be seen between fitting linear and quadratic solutions to the data. Unless split linear equations are constrained to a common solution, then there is obvious difficulty with interpretation at the junctional age.

The decline in FEV₁/FVC ratio with age and height is of particular interest because of the lower screening limits previously discussed. In Figure 13, the lower bounds of the predicted 95% confidence intervals for non-smokers of 167 cm and 187 cm have been plotted. This graph is only included as an illustration of principle as it inappropriately assumes a normal distribution of the data points around the regression line. However, it is sufficiently accurate to support some generalisations. Tall men are more likely to fail screening at any age than short men. Similarly, as the FEV₁/FVC ratio declines with age, older men are more likely to fail screening than younger men. In practice, this would mean that if an abnormal FEV₁/FVC ratio was found to be a predictor of barotrauma risk, that young short men with asymptomatic abnormal lung function would have the least chance of being detected at screening. It is perhaps of importance, that the younger inexperienced candidates are more likely to suffer pulmonary barotrauma on decompression² than their older trained counterparts although this may be, at least in part, the result of a selection process occurring as a result of accidents occurring in initial training. If the FEV₁/FVC ratio is to remain as the main predictor of success, then appropriate limits should be used to avoid height and age bias.

It has been considered elsewhere^{10,11} that measures of end-expiratory flow such as FEF₇₅₋₈₅ are more sensitive indicators of small airway function, and predictors of symptoms such as wheeze, than the FEV₁/FVC ratio. This is perhaps supported by the marked reduction of FEF₇₅₋₈₅ in heavy smokers (Figure 7). The effect of heavy smoking is less obvious in the other measured lung function variates (Figures 2 and 5). Previous attempts to offer predicted values for FEF(75-85) through linear regression have often proved unworkable due to an associated large coefficient of variation.^{10,12} In this study, a log transformation fulfilled more statistical assumptions associated with regression models.

This study allows the prediction, on a statistical basis, of ranges of normality for lung function in submariners. To have any value in screening, those who are found to be "statistically abnormal", when compared with the appropriate reference range, must be more likely to be the "clinical abnormal" requiring detection, than the rest of the examined population. In the case of submariners and divers, a set of abnormal could be defined as those candidates who have suffered pulmonary barotrauma. Records have been kept of the details of the accidents occurring during training at SETT. The majority of those recorded are thought to have suffered pulmonary barotrauma, although there is some variation between the totals reported by reviewers, depending upon the diagnostic criteria used.^{2,3} A comparison has been made, where data are available, between the recorded pre-ascent lung function and respective reference values for each accident case, where decompression sickness is not suspected.^{2,3} The 34 cases compared in this manner are listed

TABLE 2
SETT ACCIDENT CASES - COMPARISON OF ACTUAL AND PREDICTED LUNG FUNCTION

No.	Height	Age	Actual	FEV ₁		Standardised residuals	Actual	FVC		Standardised residuals	Actual	%FEV ₁ /FVC	
				Predicted	Standardised residuals			Predicted	Standardised residuals			Prediction	Standardised residuals
128	163.5	24	3125	3992	-1.80	3575	4763	-2.18	82.1	83.8	0.57		
120	182.5	20	3125	4860	-3.47	3950	5897	-3.61	79.1	82.6	-0.57		
131	174.5	18	4240	4474	-0.47	4950	5321	-0.69	85.7	84.0	0.27		
132	173.5	31	4200	4305	-0.21	5600	5430	0.30	75.0	79.2	-0.69		
133	172.5	20	4625	4383	0.48	5050	5256	-0.38	91.6	83.4	1.35		
134	164.5	24	3850	4046	-0.41	4500	4833	-0.61	85.6	83.8	0.29		
135	170.5	18	3600	4344	-1.49	4450	5174	-1.28	80.9	83.9	0.50		
136	169.5	21	4400	4285	0.24	4850	5084	-0.43	90.2	84.4	1.02		
137	179.5	20	4950	4717	0.47	6000	5705	0.55	82.5	82.8	-0.05		
138	165.5	16	4150	4061	0.18	4810	4774	0.06	86.3	84.7	0.25		
139	178.5	19	4500	4624	-0.26	5000	5598	-1.13	90.0	84.0	1.00		
140	177.5	31	4620	4714	-0.20	5280	5644	-0.67	87.5	83.8	0.60		
141	184.5	20	4575	5072	-1.03	6100	6092	0.01	75.0	83.5	-1.38		
142	184.5	19	4125	5050	-1.92	4950	6046	-2.01	83.3	83.9	-0.07		
143	176.5	21	4050	4662	-1.23	5350	5643	-0.52	75.7	82.7	-1.17		
144	182.5	21	5740	4982	1.57	7000	5994	1.85	82.0	83.4	-0.22		
145	173.5	19	4720	4398	0.67	5060	5286	-0.43	93.3	84.4	1.49		
146	178.5	16	4125	4651	-1.05	4575	5503	-1.72	90.2	84.5	0.94		
147	177.5	21	4310	4620	1.38	5840	5603	0.44	90.9	82.6	1.37		
148	177.5	22	4100	4579	-1.00	4400	5638	-2.35	93.2	82.9	1.72		
149	181.5	28	4300	4941	-1.33	4900	6093	-2.19	87.8	81.1	1.07		
150	184.5	29	5100	4862	0.48	6100	6148	-0.09	83.6	79.0	0.77		
151	177.5	22	4140	4728	-1.22	4400	5682	-2.36	94.1	83.4	1.72		
153	178.5	23	4950	4658	0.58	5450	5710	-0.48	90.8	81.8	1.50		
155	173.5	23	4750	4523	0.47	5280	5435	-0.28	90.0	83.4	1.06		
156	184.5	20	4575	5072	-1.03	4850	6092	-2.28	94.3	83.5	1.74		
157	184.5	30	5727	4840	1.77	7070	6145	1.71	81.0	78.6	0.40		
158	177.5	27	4900	4736	0.34	6900	5802	2.02	71.0	81.8	-1.78		
161	176.5	17	4843	4565	0.58	4868	5380	-0.94	99.5	85.2	2.31		
162	184.5	28	5080	4866	0.45	6640	6220	0.80	76.5	80.0	-0.58		
163	182.5	27	5158	5005	0.32	6317	6152	0.30	81.7	81.4	0.04		
164	187.5	18	4903	5093	-0.38	5022	6156	-2.10	97.6	83.0	2.42		
165	174.5	19	4317	4477	-0.32	5167	5354	-0.35	83.5	83.6	-0.01		
166	177.5	23	5112	4738	0.78	5653	5714	-0.11	90.4	83.1	1.18		

in Table 2. No details of the FEF_{75-85} values for these cases are available.

If a statistical limit of normality of two standard deviations from the mean is placed on each of the LFV's shown, then in a batch of 34 subjects, for any LFV, it is likely, at most, that one case would be detected as being outside the range. Amongst the "clinically abnormal" 34 accident cases, 8 have a recorded FVC below the lower statistical limit. In comparison, only one FEV_1/FVC ratio is below normal and in this case both FEV_1 and FVC are normal. This result is subject to the bias introduced by the rejection of candidates following spirometric screening at SETT. However, if a low FEV_1/FVC ratio is predictive of accident it would be expected that the proportion of accident cases with a borderline (close to 75%) ratio would be greater than for the rest of the population. No such association has been found.

A highly significant correlation ($P < 0.01$) has been found, however, between chance of accident and reduced FVC. The validity and practical value of this result depend upon the accuracy with which lung function was measured and the diagnosis assigned in each of the accident cases. It is known that errors in spirometry are most likely to have occurred amongst the earlier cases in the series of 34, before standardisation of technique was introduced. It is noteworthy that abnormally low FVC predominantly occurs in the later cases. This may indicate an even stronger correlation between reduced FVC and accident risk than has been estimated.

Conclusions and recommendations

Reference values for FEV_1 , FVC, FEV_1/FVC ratio and FEF_{75-85} for males between the ages of 17 and 35, have been produced from a study population of submariners and submariner candidates.

In a comparison between the lung function variates currently measured during screening with respect to predictive value for pulmonary barotrauma, a highly significant association has been found between the risk of accident and lowered FVC. No such association has been found for the FEV_1/FVC ratio although this result is subject to bias. It is concluded that up to $1/4$ of male submarine escape, and perhaps diving accident cases, involving pulmonary barotrauma could be prevented by making those who have an abnormally low FVC (greater than 2 standard deviations below mean) medically unfit. No further test is known that could be used sequentially or in combination to increase sensitivity and specificity of screening by FVC. The exclusion rate by FVC with a limit of 2 standard deviations would be far less, however, than that of the FEV_1/FVC ratio that is currently in use without clinical validation.

Acknowledgements

The authors acknowledge the assistance given by the medical staff of the Submarine Escape Tank, HMS DOLPHIN, without whose help this project would not have been possible.

References

- 1 Handbook of Naval Medical Standards BR 1750 A. London, Ministry of Defence 1975.
- 2 Pearson RR. The aetiology, pathology, presentation and therapy of pulmonary barotrauma and arterial gas embolism resulting from submarine escape training and diving. Thesis for Doctor of Medicine. University of Newcastle 1981.
- 3 Brooks GJ, Green RD and Leitch DR. Pulmonary barotrauma in submarine escape trainees and the treatment of cerebral arterial gas embolism. *Aviat Space Environ Med* 1986; 57: 1201-1207
- 4 Draper N and Smith H. Applied regression analysis. J. Wiley & Sons 1981.
- 5 Glindmeyer HW. Predictable confusion. *J Occup Med* 1981; 231: 845-849.
- 6 American Thoracic Society statement on standardisation of spirometry. *Am Rev Resp Dis* 1979; 119: 831-838.
- 7 Knudson RJ, Slatting RC, Lebowitz MO and Burrows B. The maximal expiratory flow volume curve. Normal standards, variability and effects of age. *Am Rev Resp Dis* 1983; 113: 587-600.
- 8 Morris JF, Koski A and Johnson IC. Spirometric standards for healthy non-smoking adults. *Am Rev Resp Dis* 1971; 103: 56-67.
- 9 Paoletti P, et. al. Reference values for vital capacity and flow-volume curves from a General Population Study. *Bull Eur Physiopath Resp* 1986; 22(5): 451-459.
- 10 Morris JF, Koski A and Breese. Forced and expiratory flow. *J Occup Med* 1974; 16: 750-762.
- 11 Douglas RB. Final Report: A six year follow up study of respiratory morbidity in London firemen: Report to the Home Office. London; 1985.
- 12 Clifford GM, Smith DJ and Searing CSM. An assessment of forced expiratory volumes and flow rates in the detection of early ventilatory impairment in RN submariners. *J Roy Nav Med Serv.* 1986; 71: 1670-7.

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GLEANINGS FROM MEDICAL JOURNALS

The story of two dive computers

At a recent seminar on the Edge and Skinny Dipper in Oakland, California, Orca Director of Engineering Paul Heinmiller displayed a slide of Skinny Dippers sitting on fire coral with a diver near the surface in the background.

Just as he was ready to flip to the next screen, a diver in the audience noted that the depth reading on one Dipper was 23 feet, while another, sitting right along side, read 20 feet. A few chuckles could be heard, while Neidenmiller, obviously surprised, could only say that they had better check the slides more closely before showing them.

But a few minutes later he recovered nicely. Flipping back to the 23/20 slide, he said that the Dipper has a depth accuracy of + or - 2 feet. Therefore, any two Dippers could display a four-foot difference between them. The difference in the two Dippers in the slide meant that the true depth would be either 21 or 22 feet.

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Fatal arterial gas embolism: detection by chest radiography and imaging before autopsy

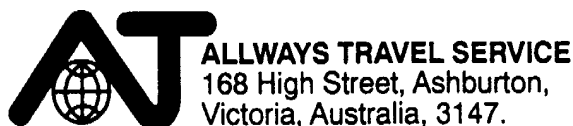
John A Williamson, Geoffrey K King, Vic I Callanan, Robert M Lanskey and Keith W Rich

Med J Aust 1990; 153: 97-100

Abstract

Two recent cases are reported from north Queensland of deaths from massive arterial gas embolism occurring in tourists scuba diving on the Great Barrier Reef. The diagnosis was established in each case by an external examination of the body, followed by a plain erect chest radiograph soon after death and before autopsy; in one of the cases it was further confirmed before autopsy by computed tomography (CT) of the head, neck and thorax. The diagnosis was also supported by analysis of a diving profile, inspection and investigation of diving equipment, and autopsy. In the light of previously published advice and reports, the experience gained from these two cases now dictates that investigation of an unexplained death occurring after exposure to, and change from, hyperbaric or hypobaric conditions, should begin with plain erect chest radiography on the body before autopsy. Combining this with a pre-autopsy supine chest film before standing the body erect, and CT scanning of the head, neck and chest, is also recommended.

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\$NZ 500.00

Further Information and enrolment contact

Cdr. Des Gorman, MB ChB, FACOM, PhD,
Director, DHMU,
RNZN Hospital,
Naval Base,
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SPUMS MEETING FEBRUARY 23RD AND 24TH 1991 COFFS HARBOUR, NEW SOUTH WALES

A weekend meeting will be held at Coffs Harbour, New South Wales, on February 23rd and 24th 1991. The organiser is Dr Darryl Wallner, 114 Vasey Crescent, Campbell, ACT 2601, phone (06) 248 5950, who would like to hear from those wishing to present a paper.

The lecture programme will be held on Saturday afternoon and will be followed by a dinner, approximate cost including wines \$ 30.00.

The meeting will be held at the Aanuka Resort which has first class facilities, pool, spa, gym, tennis courts and an excellent surf beach. The accommodation is on the basis of twin share in individual units set in beautiful tropical gardens. Early registration will enable numbers to be finalised with the Resort at very favourable room rates, approximately \$ 100.00 per person per night, which includes breakfast, lunch, Saturday afternoon tea and the use of all facilities.

Diving is unique at the Solitary Islands as it has a mixture of tropical and temperate zone marine life with true corals, sponges and tropical fish life mixed with colder water species.

There is a registration fee of \$ 10.00 which should be sent to Dr Wallner as soon as possible with the following information; name, address and phone number, accommodation required, arrival date, time and length of stay, method of transport and whether diving is required on Saturday or Sunday. A registration form will be found below.

SPUMS MEETING AT COFFS HARBOUR ON FEBRUARY 23RD AND 24TH 1991

REGISTRATION FORM

Name

Phone number ()

Address

Type of Accommodation

Share twin

Single (extra charge)

Family

Travel Car / Plane

Arrival Date

Time

Departure date

Number diving Saturday morning

Sunday morning

Do you wish to present a paper ?

Yes / No

Topic

Please send, with your cheque for \$ 10.00 made out to **SPUMS Coffs Harbour Meeting**, to

Dr Darryl Wallner,

114 Vasey Crescent, ACT 2601.

Phone (06) 248 5950