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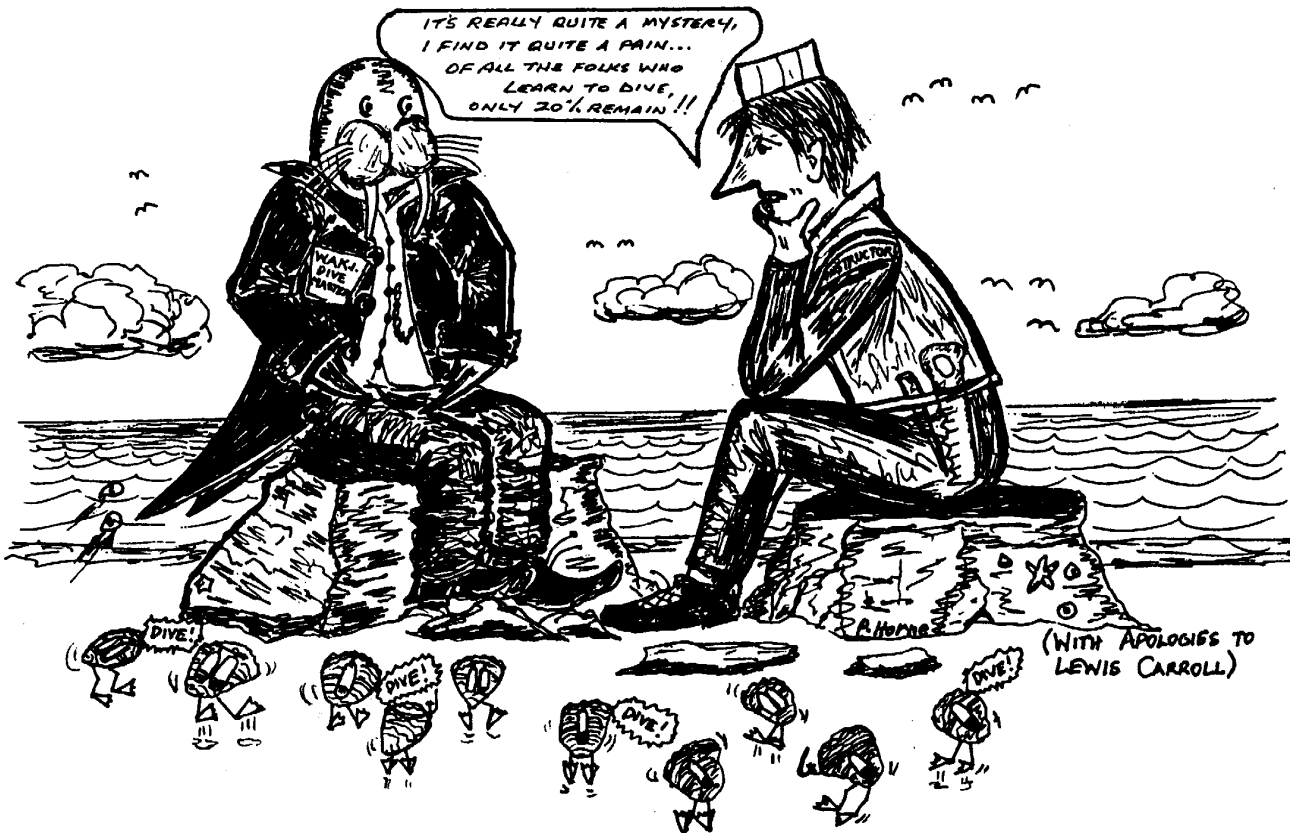

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OBJECTS OF THE SOCIETY

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

New Zealand Chapter

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.

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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 The author's name and address should accompany any contribution even if it is not for publication.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.

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This meeting is scheduled to finish so that members can have adequate time to travel to the

UNDERSEA AND HYPERBARIC MEDICAL SOCIETY MEETING

in HAWAII from June 7th to 11th 1989
and enjoy and contribute to this meeting

For further information about the
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UNIVERSITY OF NEW ENGLAND
Department of Continuing Education
will be holding a
MEDICAL UPDATE CONFERENCE
on
SPORT DIVING
on October 14th, 15th and 16th 1988
in ARMIDALE NSW

The conference objectives are to;

Inform medical practitioners and other persons involved in the care and tratement of sports divers, of the techniques for performing medical examinations on prospective scuba divers.

Inform medical practitioners and first aid personnel about emergency treatment in sports diver accidents.

Improve sports divers' awareness of physiological and medical aspects of diving.

Provide update material for practising diving instructors.

Speakers include Dr Chris Acott,Dr Carl Edmonds, Dr Rod Green (RAN School of Underwater Medicine), Professor Brian Hills and Mr John Mathieson. Ms Carol Wright will be the introductory speaker.

Registration fees
Residential $190.00, Non-residential $148.00. Registrations close on 29.9.88.

For further details contact

Mr Carl Petersen,
Conference Co-ordinator,
Department of Continuing Education,
University of New England,
PO Box 591,
TAMWORTH, NSW 2340.
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EDITORIAL

We wish to draw our readers attention to the minute recording the honouring of the first president of the Society, Dr Carl Edmonds, by electing him to the first Life Membership awarded by the Society at the recent Annual General Meeting. The minutes of that meeting do not record the part Carl played in the foundation of the society and in its early years to give it a firm footing on which others have built. We congratulate Dr Edmonds and we wish Dr Edmonds many years of enjoyment of his Life Membership.

Decompression sickness (DCS) is undoubtedly the most important problem impinging on the image and practice of recreational scuba diving and the commensal groups of the diving instructor organisations and the sports diving stores. Whether the present noticable increase in the numbers attending recompression units represents a true or only an apparent increase in the incidence of DCS is debatable but it is an inescapable fact that the numbers being treated have an effect on the sociopolitical view of the diving community. It is also an unfortunate fact that those affected will probably be left with some degree of residual damage even where they are clinically cured. For such reasons the papers presented in this issue are particularly opportune.

This issue of the Journal contains lesson for all readers. The experience of the Townsville group leads them to believe that all cases of DCS have neurological signs. The great majority of cases treated in Adelaide had neurological signs as did those reported to the Divers Alert Network (DAN) in the U.S.A. A careful neurological examination is essential for every case of DCS. Dr Lloyd Jenkins shared his learning experiences with the Annual Scientific Meeting and his conclusions should be noted by all readers, especially the need for recompression therapy if the diver's symptoms remit with 100 % oxygen.

The DAN Report, reviewed here by John Knight, is a mass of data the meaning of which is possibly somewhat obscured by a complex computer program. A reader may be forgiven for remembering a quotation from W. Edwards Deming that "figures by themselves provide no information, no meaning, no interpretation, in the absence of theory. In short, facts are no substitute for knowledge". This is well illustrated by DAN table juxtapositioning Asthma with Decompression Sickness and Arterial Gas Embolism without any case histories to connect the facts as being any other than coincidental. There are many asthmatics who dive, few of these come to grief, and then the asthma is not by any means the only critical factor. The true significance of having an over-reactive bronchial tree will never be evaluated while there is a pretence that no asthmatics dive and that all asthmatic divers come to grief. Gathering relevant facts is surely a job for "Project Stickybeak" but a job that this scheme can only fulfill if supported by the diving and diving medical communities.

John Lippmann has provided a resume of the American National Underwater Accident Data Centre (NUDAC) report covering diving fatalities in 1985. Once again many avoidable causes of death are recorded. Considering the litigious nature of US citizens it is suprising that any one has the courage to teach diving in the USA when so many people die under instruction each year.

Dr John Williamson's paper on arterial gas embolism (AGE) emphasises how little is actually known about the lesions in the lung in AGE and points the way for further research.

The papers dealing with divers' knowledge of the decompression tables draw our attention to something that is normally ignored, the inability of many divers to use the tables accurately. Perhaps there is something to be said for using a decompression computer, it only has to be read and followed, no mathematics or knowledge of procedures are required to get the information needed. But is the information accurate ? A paper in the next issue will touch on this subject, which has already been discussed in past issues.

The present organisation of advisory and treatment services in Australia and New Zealand is such that all divers have reasonable access to advice and treatment if they require either. The remarkably developed system of diver support which has evolved in Australia in recent years is detailed in the PADI publication reviewed on page 114, where Peter Horne's book on the South Australian diving fatalities is also reviewed. Publications such as the PADI book may in time bring an awareness of the diving safety work being done in Australia and in New Zealand to the notice of the European based Confederation Mondiale des Activities Subaquatiques (World Underwater Federation), which is usually known by the abbreviation CMAS. CMAS is largely a non-English-speaking organisation, as any reading of their English literature will confirm. The Australian Underwater Federation (AUF) is a member of CMAS but for reasons of cost cannot make an appropriate physical appearance at the meetings on a regular basis. Now is possibly the appropriate time to bring to the attention of the CMAS Medical Committee what has been and is being achieved in the antipodes. Readers of this Journal who are able to attend the Diving Medicine Conference to be held next year in Martinique may like to propagate this information to the assembled CMAS members.

Once again we have to apologise for the late publication of this issue of the Journal. We hope to catch up by the time the first issue of 1989 is due. However this is dependant on the the provision of papers for publication.

One final word of warning, readers are advised that the opinions expressed in Editorials do not necessarily represent the views of anyone other than the Editor.

SPUMS ANNUAL SCIENTIFIC MEETING 1988

THE TOWNSVILLE DIVING MEDICAL AND AEROMEDIVAC SYSTEM EXPERIENCES, LESSONS, AND THE FUTURE

John Williamson, John Orton, Vic Callanan, William Ellery, John Hardman and Ray Palmer

Since 1977 the Townsville Recompression Chamber (RCC) (Figure 1), located at the Australian Institute of Marine Science (AIMS) about 70 kms south of the city, has been used to recompress 68 patients to April 1988, the vast majority of them (64) divers. The RCC is a twin lock Comex deck recompression chamber, constructed in Singapore, and placed at AIMS with the birth of the latter, in 1968. Before 1977, it was used for equipment tests and AIMS diving staff familiarisation only, under the care of and maintenance by two of us (WE and JH).

The chamber weighs 6 metric tons, and has working pressure of 8.5 bars (86.7 metres of sea water, 127 psi, 900 kPa (approx.)). Its overall volume is 10,830 dm^3, or 383 cu ft (main chamber 7,600 dm^3, or 268 cu ft), and its external dimensions are:-

Length	4,700 mm
Width	2,500 mm
Height	2,100 mm
Diameter	1,819 mm
Single end (small lock), circular entrance door (Figure 1), diameter	700 mm.

From the inception of the service a splendid attitude of co-operation existed between AIMS and the Townsville General Hospital, which profoundly facilitated its operation. This happy state of affairs was continued and complemented by the arrival of the National Safety Council of Australia (NSCA) in Townsville in 1985. The NSCA brought with them the Drager "Duo-Com" portable RCC, which enhanced the treatment of divers requiring recompression. Some months later, the AIMS RCC had an adaptor flange (generously donated by the Utah Foundation Australia at a fitted cost of about $25,000) attached, which permitted transfer under pressure (TUP) between the DuoCom and the fixed RCC (Figure 2).

The NSCA also brought a superb integrated team of pararescue personnel, pilots, and maintenance and administrative staff to Townsville. They have a modern, well established Airport Base in Townsville which co-ordinates an extensive 24-hour search-and-rescue (SAR), and retrieval aerial service for the whole of Northern Australia. Their equipment available for the diver retrieval service is truly state of the art (Figure 3).

ACTIVITIES 1977-APRIL 1988

The table summarises overall experience of the team in treating patients with decompression sickness (DCS) and cerebral arterial gas embolism (CAGE) to April 1988.

With the exception of 1 fully categorised profes-

Figure 1. The Townsville Recompression Chamber (RCC), (showing the transfer-under-pressure flange for DuoCom attachment, generously donated by the Utah Foundation of Australia.)

TABLE 1
TOWNSVILLE DIVER RETRIEVAL/RCC TEAM EXPERIENCE
10 YEARS, 1977 - APRIL 1988

YEAR	D.C.S.*					C.A.G.E.**
	Spinal	Cerebral	Skin	Pulmonary		Joint
1977	1	1				
1980						1
1982	5	1				
1983	12	1	1			
1984	6	1		1	1	1(1)
1985	5					1(1)
1986	8	3				3 (1***)
1987	10	5				1
1988(4 months)	2			1		1
TOTALS	49	12	1	2	1	8(3)

* Decompression Sickness
** Cerebral Arterial Gas Embolism (Fatalities in brackets)
*** In addition to this fatality, another during that year in a recreational scuba diver, never reached recompression facilities alive.

The totals add up to more than 64 because some patients had more than one form of DCS co-existing.

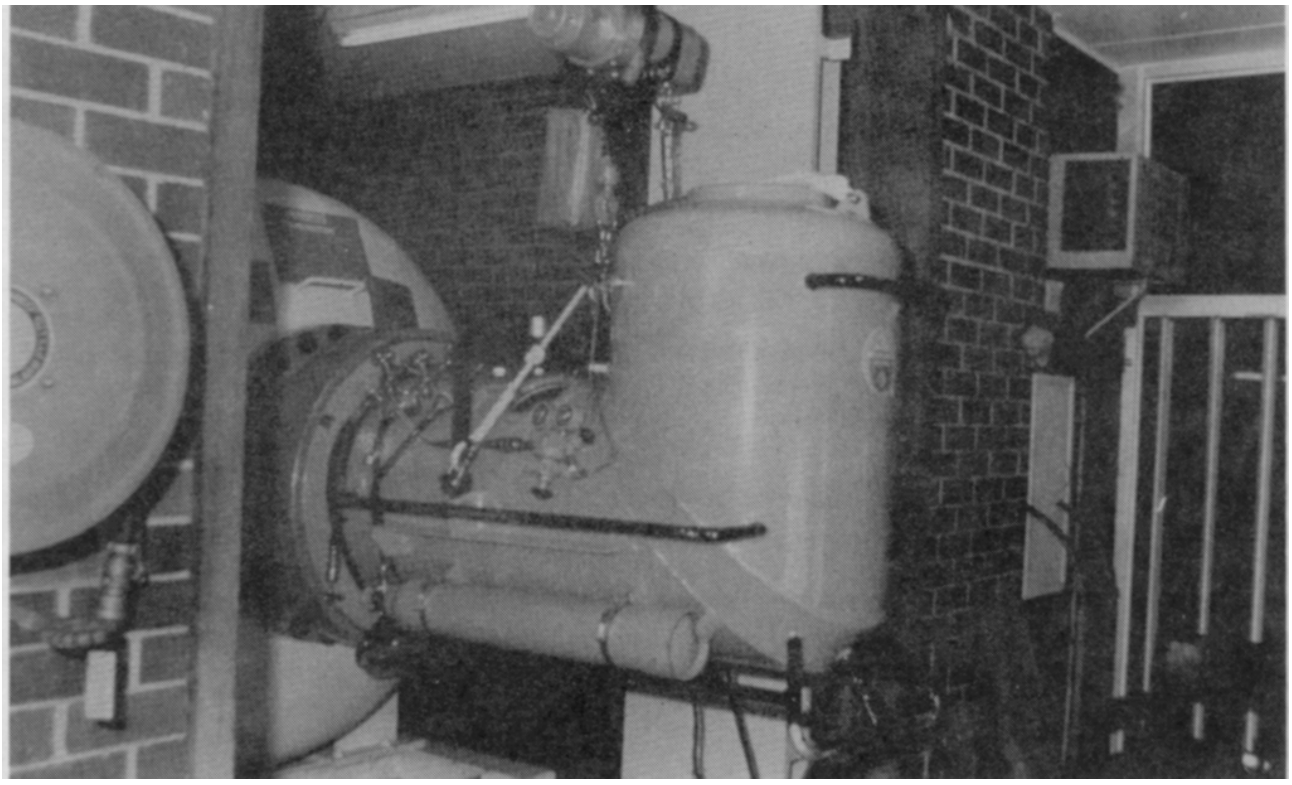

Figure 2. Shows the “lock on” position for a transfer-under-pressure from the DuoCom into the mainplace RCC.

sional diver (pulmonary and spinal bends), all cases were either recreational or occupational divers, i.e. trained to Australian "sport diver" levels, or their equivalent. At least 5 had no formal training whatever.

The overwhelming majority of divers in the area serviced by the Townsville facility (Great Barrier Reef and West Pacific area) indulge in relatively shallow (less than 30 m), repetitive recreational diving, using scuba to breathe compressed air. Only 7 were on "hookah" (surface supplied compressed air) at the time of the accident.

This diving pattern appears to produce predominantly spinal decompression sickness. Co-existing cerebral DCS is not unusual.

In our experience, a diver with DCS in whom central neurological signs cannot be elicited clinically, is rare. We currently doubt the existence of such a clinical entity!

We find no clear difference in the susceptibility to DCS between the sexes (24 female). Ages ranged from 18 to 63 years. Non-Australian nationals comprised about 17 of the 64 treated.

Both cases exhibiting pulmonary DCS had, or rapidly developed, spinal DCS.

True joint "bends" are rare, in our experience, with this pattern of diving.

The need for recompression in Northern Australian is steadily increasing, as is the amount of tourist diving.

It is clear that CAGE is not a rare event (see page). Episodes of loss of consciousness in association with recreational diving are more common than are indicated by the number of our CAGE cases. In view of the recent work by Gorman and his colleagues[1], some of these people who do not present for treatment would be expected to have central neurological sequelae.

CAGE may occur at all levels of experience, in "sport-diver" trained scuba divers breathing air, and in the complete absence of any detectable error or other misadventure. It is always associated with ascent, either planned or unplanned[2].

AEROMEDIVAC RETRIEVALS

Before 1985 and the regular availability of NSCA and the DuoCom only 3 long distance retrievals by air were made (2 from Thursday Island), out of a total of 24 patients treated. The patients mostly came to us. Since 1985, the management of 22 of the 40 patients has involved air retrieval in the King Air or the 412 (Figure 3), or rarely a Lear Jet. The three longest journeys were from Christmas Island in the Indian Ocean using a Lear Jet, and twice to Adelaide using the King Air. A combination of rotary-wing and fixed-wing aircraft was used on one occasion to deliver the doctor onto a boat at sea by winch, and subsequently to bring the patient back from Cooktown, once the boat had made land there. These all-weather aircraft with their skilled NSCA crews, and sophisticated navigation equipment, make such journeys relatively comfortable and "hassle-free" for the medical team. Using the NSCA facilities, no diver in need of medical attention and recompression in our area of responsibility, the West Pacific and from Papua New Guinea to Brisbane, has ever had his treatment delayed on our account. The location of the fixed RCC for such a huge area, in Townsville, with its large medical, intensive care, and NSCA bases, makes geographical and functional sense.

In 1981 the team published from AIMS a set of "Field and radio communication instructions for retrieval at sea", for the benefit of vessel masters, dive masters, and district medical officers. This proved most helpful in the smoother co-ordination of such events, which is always a challenge to participating persons. In 1985, the instructions were rewritten and updated, incorporating the facilities of NSCA, and published by them[3]. These are in current use, and continue to prove useful, especially for the burgeoning tourist charter boat operaters in North Queensland.

USE OF THE "DUOCOM" PORTABLE RCC

This device has made the rational and early application of recompression therapy possible to those divers in need. Twenty retrievals using the DuoCom enroute, have been performed since 1985, without incident. With one exception, all of these patients had their initial recompression (usually RN Table 62) completed in the DuoCom, on arrival at the NSCA's Townsville airport hanger. This has been dictated by factors such as the time remaining in the Table, the satisfactory condition of the patient and attendant, and the safety and cost of using helicopter transfer from the airport to AIMS. Only one TUP into the AIMS RCC has occurred to date. However practice runs are carried out on a regular basis. The relatively high usage (50% of patients treated since 1985) of portable recompression in our series reflects both the vast distances in our area of responsibility, and the high level of proficiency practised by the technical members of the team.

LIAISON WITH THE DIVER EMERGENCY SERVICE (DES), AND COMPLICATIONS

It is a pleasure to record the smooth and harmonious liaison our group have enjoyed with DES during the latter years covered by this report. The philosophy of the medical team has always been to consult readily and often, and it has paid off. No major complications in any patient, nor permanent morbidity due to the therapy, occurred in any of our patients, of which we are aware. One suspected case on spinal DCS was produced in a chamber attendant, early in the experience of the team (1983). Equalisation difficulties with first recompressions were relatively common, and

Figure 3(a) The National Safety Council of Australia's Townsville fixed wing aircraft, a Beechcraft "King Air". This is an all-weather aircraft, with a "pressurise-to-one-atmosphere" capability. It can fly, with an operating DuoCom and team on board, non-stop from Townsville to Hobart, or its equivalent, without refuelling.

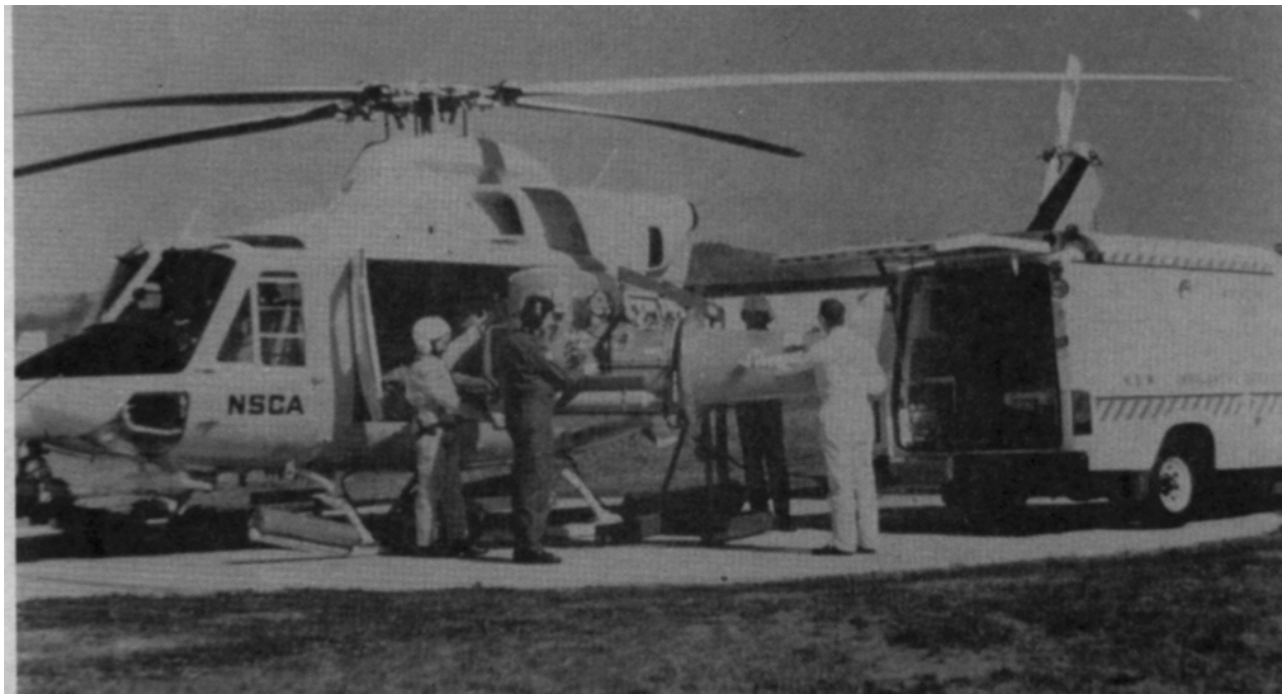

Figure 3 b) The NSCA's rotary wing aircraft, capable of carrying an operating DuoCom with team. This is Bell twin jet engined aircraft, equipped with state-of-the-art search and navigation equipment for night or day, and a vertical hoist. Its range fully laden is 400 kms. (Townsville to Rockhampton or its equivalent.)

middle ear squeeze has occurred on occasions. Surprisingly, we have had no florid otitis externas, despite our tropical location and non-climate controlled RCC! Sinus squeezes have been rare. Overt central neurological oxygen toxicity has not occurred to date, nor have RCC complications of pulmonary barotrauma. Early and minor signs of pulmonary oxygen toxicity were evident in some (retro-sternal discomfort, cough, small vital capacity decreases), which disappeared with completion of treatment. Initial patient and attendant dissatisfaction with our oxygen-bibs (built in breathing system) disappeared with the installation of silicone, autoclavable moulded face masks and Robertshaw demand-flow, overboard-dump circuits. It is remarkable to record that there was not one mechanical, maintenance, nor equipment failure during treatment in this series. This faithfully reflects the high standard of RCC maintenance that has occurred. The remote location of the AIMS RCC relative to the Townsville General Hospital has made for large logisitic strains upon all members of the team, and an enormous wastage in travelling time and costs, over the years, not to mention considerations of patient safety. We look forward to the re-location of the RCC to inside the Townsville General Hospital, which appears imminent.

RECOMPRESSION THERAPY TABLES USED

Of the formal recompression therapy tables, Royal Navy tables have been used almost exclusively. The commonest chosen have been RN tables 62 (frequently "extended") and 61. Nine metre "soaks" have also been relatively common, towards the end of treatment regimes. No deep air tables have been used in this series. Our 64 diver patients involved 207 recompressions, which were composed thus:-

RN table 62	RN table 61	9 m "soak"	Other
89 (20 extended)	58	38	22

In addition, there were 20 recompressions in the DuoCom (18 RN table 62, 2 RN table 61).

MEDICAL CONTROL DURING RECOMPRESSION

In accordance with currently favoured practice, there was always a qualified medical intensivist outside either the fixed RCC or the DuoCom during every recompression. These persons never entered the RCC during treatment except for a specific medical indication. His or her subsequent decompression was planned beforehand, and undertaken in conjunction with careful discussion with the RCC operator. In the earlier days of the teams experience, the medical person had to act as the RCC attendant. In all such cases, a second medical colleague was placed on site, outside the RCC. The provision of medical manpower has placed and still does place at times, considerable logistic strains on the staffing of the Anaesthetic and Intensive Care Department of the Townsville General Hospital. There seems to team members to be little medical administrative or political appreciation of that fact, although such persons are observed from time to time happily basking in the credit for the service!

RCC attendants, during the years before 1985, were drawn from a pool of interested volunteer sport diving instructors and dive masters with appropriate paramedical training. These wonderful people provided unstinted and troublefree service, frequently through the night, and all in their spare time, unsung over an 8 year period, and totally free of charge. This fact also remains largely unappreciated by the North Queensland community. On behalf of ourselves and all our patients, we gratefully acknowledge this humanitarian effort. More recently, with the advent of the NSCA in Townsville, combined with the establishment of a pool of intensive care nursing staff from the Townsville General Hospital, trained in hyperbaric nursing care, RCC attendants have become an integral part of the hyperbaric team.

All DuoCom attendants were, and are, NSCA Life Support Technicians. Portable RCC attendants require additional training and skills, over and above those necessary for fixed RCC duties.

LESSONS AND CONCLUSIONS TO DATE

The successful management of diving accidents necessitates the close integration of specially trained, and regularly practised teams of medical, paramedical, hyperbaric technical, and highly skilled aero-technical persons, as well as constant access to consultative hyperbaric medical expertise.

State-of-the-art aircraft and equipment should always be the goal, for considerations of safety. It also has the benefit of comfort for the treatment team. Nevertheless, equipment is only as good as the users.

Portable recompression facilities with attendant air transport capability, are now an essential component of diving medical care in Australia.

Decompression sickness is a disease of the central nervous system. Disease may begin before symptoms. The term "mild decompression sickness" is a misnomer, and should be abandoned.

Early adjuvant therapy in DCS, 100% oxygen and, intravenous fluid rehydration, is an important contributor to a favourable outcome. Many involved persons (including some medical ones) do not understand simple oxygen therapy apparatus, nor how to provide 100% inspired oxygen to a spontaneously breathing diver patient.

It is unlikely that even the earliest and most effective recompression and adjuvant therapy presently available for

DCS will reverse all the significant damage already sustained by the central nervous system. Long term follow-up studies are needed on a big scale. Most recreational divers have little understanding of the seriousness of DCS to themselves.

A level of gross irresponsibility still prevails amongst elements of the sport diving population. The pursuit of the dollar causes some to turn a blind eye to safe practices. In others irresponsible behaviour due to a combination of poor self-discipline and sheer ignorance has resulted in an expensive, and occasionally risky, retrieval and treatment; sometimes innocent dive buddies have needed treatment as well! Such financial costs are presently borne by State Departments of Health, or even carried by the NSCA! We believe the time has come for clearly evident irresponsibility by a diver or his supervisors to be rewarded with a bill for the costs of his or her retrieval and medical treatment. We would advocate consideration of carefully worded leglisation to that effect.

A therapeutic recompression chamber is a specialised intensive care patient locality. Its safe application is only possible as an integrated part of a fully functioning in-hospital intensive care unit, with staff trained in that speciality. At the same time its safe operation and maintenance requires full technical support. Large therapeutic RCC's are best located inside, or in immediate proximity to hospitals, and should function as part of an intensive care unit.

ACKNOWLEDGEMENTS

The inception of this community service occurred during the terms of office of Dr John Bunt Ph.D., former Director of AIMS, and Dr Alistair Cole FRACS, former Medical Superintendent of the Townsville General Hospital. We gratefully acknowledge the spirit of co-operation and forward thinking shown by these two men. It is also a pleasure to acknowledge the on-going assistance of Dr J.T. Baker Ph.D., Director of AIMS, Dr Kay James FACMA, Deputy Medical Superintendent of Townsville General Hospital, and the manager and staff of Townsville Radio VIT, of the Australian Coastal Surveillance network.

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Mr William Ellery was formerly Field Operations Manager, Mr John Hardman Field Operations Manager at: The Australian Institute of Marine Science, Private Mail Bag No. 3, Mail Centre, Townsville, Qlueensland 4810, Australia.

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DIVING CASE HISTORY

Lloyd Jenkins

This episode occurred over the Australia Day weekend (January 23rd to 25th 1988). The sequence of events involved a new instructor to the area, hereafter called A, his 22 year old girlfriend, who came up from Melbourne for the weekend, B, another experienced diver employed by the Dive Shop as coxswain and instructor's help, C, and three others, all from Melbourne.

The first dive was on Saturday, 23rd January, 1988, to 15 metres for 60 minutes in a class of 6 people and was uneventful. On Sunday, 24th January, 1988, the same class dived again to 15 metres for 90 minutes in the morning, presumably using 2 tanks, and in the afternoon to 18 metres for 40 minutes, both in open water. The surface interval was not specified but even allowing for 4 hours surface interval the second dive was 4 minutes over the limit for a no-decompression dive on US tables and 14 minutes over on Bassett tables. On Monday, 25th January, 1988, a group of 6 including A, B, C, dived in the morning to 12 metres for 40 minutes and around midday or perhaps 1300 hours, which would give a surface interval of possibly 4 hours, they dived

to 30 metres for times varying between 22 and 25 minutes depending on whose time-keeping is believed,. At best this is 4 minutes over no-decompression times on US tables and 8 minutes over on Bassett tables.

This day there was a slight southerly breeze and the sea was not rough. The boat was anchored over an area thought to contain a shark gutter, and all six people donned gear and dived overboard. On the trip out B complained of being cold and tired. She had no watch or time-keeping device. A (the instructor) had a non functioning depth gauge. He apparently stayed close to B and used her depth gauge, and she relied on his watch. After 22-25 minutes they had reached 30 metres and had not found the gutter and proceeded to ascend. C (the coxswain) had a free flow from his regulator at 15 m and made a rapid ascent, surfacing some 100 m from the boat. A ordered a decompression stop at 3 m for 3 minutes, but B would not stay with him because she was cold, and went on to surface where she found she was 70 m from the boat, to which she swam.

A stayed with the other 3 and they surfaced approximately 70 m from the boat as well. B needed to be helped aboard and immediately complained of exhaustion and sleepiness and lay down in the boat and appeared to sleep or doze all the way back to shore.

My first knowledge of the dive came from C (the coxswain) who presented at the surgery complaining of a pain in the left wrist. He said that he felt he had hurt his wrist helping the girl into the boat and then steering the boat through choppy water.

He was taken to the local hospital and put on 100% oxygen head down for two hours with 5 minute air breaks every half hour. He said that he was fine when I saw him again 2 hours later and was allowed to go home. That night at 23.45., I was called to the hospital to see three divers who had symptoms suggestive of decompression sickness. They were the instructor A, girl-friend B and another from Melbourne D, who admitted to no complaints at all but who had been persuaded to attend by A, with whom he was staying. B complained of skin tingling, headache, a rash on her left knee, pain in the left shoulder and right knee and loss of balance. She said that she had been exhausted immediately after surfacing but the other symptoms came on one and a half hours later. On examination she was quite unable to stand or walk without help, a rash was present, the blood pressure was 130/70 and the pulse 84.

A complained of headache, pains in his neck and right shoulder, lower abdomen and back This had started 15 minutes after surfacing. His symptoms were complicated by the fact that he had suffered neck and shoulder injuries in a motor vehicle accident some 2 years before and was still awaiting an insurance settlement or a court case. I had previously seen him with similar pains. He admitted to abdominal pain only after transfer to Sydney.

I put an I.V. line in to both and administered 100% oxygen while arranging transport to Sydney Prince Henry Hospital, and both were given intravenous dexmethasone 4 mg. There was no doubt about B, but at that stage I was not convinced that A was bent, however Dr. Ian Unsworth was in no doubt and recompressed him because of increased complaints. The Air Ambulance arrived at 5.15 a.m. and both A and B were transferred to Sydney, where they were recompressed on a modified Table 6 using 100% oxygen at 18 metres and 9 metres with 2 air breaks. Both responded completely to the single treatment.

The other chap with no complaint was very anxious to leave and go home to Melbourne and was allowed to do so.

C whom by now I had realised was part of the same dive presented again to Casualty at 10.30 a.m. on 26th January, 1988, with similar wrist pain which again subsided with oxygen therapy for 2 hours, and again he was allowed hom. On 27th January, 1988 he still complained of joint pain and now depression, and so he also was transferred by air ambulance to Sydney where he was treated successfully by recompression on modified Table 6.

There are lessons to be learned from this episode by both divers and medical therapists.

1. A sense of responsibility and psychological maturity is essential in diving instructors, and the instructor organisations for the most part are aware of this.

2. Equipment must be in first class condition and each diver should be fully equipped and use his or her own equipment.

3. Diving to the limit of no-decompression tables can be dangerous,

4. All conditions should be considered, such as the temperature of water and fitness of the divers, when planning deeper dives.

5. Nobody was left in attendance in the boat. This is a dangerous practice.

6. Some of the effort could have been taken out of the post dive period if a shot line had been used.

7. The girl was cold and tired before she dived and probably should not have dived at all.

8. The deep dive should not have followed the morning's shallow dive.

9. The symptoms were not recognised for 8 hours afterwards.

10. Oxygen therapy is only emergency treatment. If symptoms are suggestive of decompression sickness and they respond either partially or temporarily to 100% oxygen, then that should be sufficient to convince the attendant that recompression is essential.

Dr Lloyd Jenkins' address is Pambula Plaza, Pambula, New South Wales 2549, Australia.

ANOTHER WAY TO GET BENT

John D.McKee

This 32 year old patient had been abalone diving for sixteen years, mostly just south of Nowra and south of Eden. He had had seven abalone diving days during 1988, of which four were in March, and his most recent dive had occurred south of Eden on 21.3.88.

The history

His diving profile for the day had involved an initial dive to 18 m for 45 minutes, after which he brought his abalone bag back to the boat. He then immediately descended again. The second dive was to 21 m for 70 minutes, although in fact he did admit later that he spent approximately 10 minutes of the 70 minutes at 27 m. He then surfaced, gave his abalone to his sheller, and immediately descended to between 6 and 9 m where he spent the next ten minutes.

I saw this patient and his sheller on 23.3.88, and the sheller said that while he was aboard their boat at Cape Howe off the New South Wales-Victorian border, a boat, a shark cat, had approached at great speed, rammed their boat. The shark cat driver jumped aboard, pushed the sheller out of the way, and then drove their boat off at high speed, dragging the diver along below for a distance of about 100 metres. During this alleged episode, the sheller kept yelling out that there was a diver below.

Eventually, the "pirate" driver stopped the boat, allegedly indicated that he was a "Fisheries Inspector", and dragged the diver out of the water. He then drove the boat and its occupants to Gabo Island. Subsequently the patient and his sheller were interviewed for two hours, by Inspectors, and the boat was confiscated.

That night, 21.3.88, the patient complained of fairly severe pains in his hips, shoulders and right elbow, and he had a troublesome headache. He was seen by nursing staff at a local hospital, and apparently he was given oxygen to breathe for 20 minutes, but at no stage was he seen by a doctor.

The following day he was no better, and when referred to me the next day, 23.3.88, he seemed to be rather vague with a poor memory, and most of the information and history I obtained from his sheller, who was an amateur diver.

His mate had known him for some years, and he had observed a pronounced loss of alertness, lethargy, definite memory loss, a slowness in doing all things, especially manual activities, and he had observed the diver's hands shaking from time to time. The diver complained of a whistling sound in both ears, and he still had pain in both hips, both soulders, the right elbow and the upper abdomen.

On examination the patient was fully conscious, there was no gross neurological abnormality, he walked with a "wide base", but he was tender in both groins, and I suspected his abnormal gait was due to tearing of ligaments when he was towed through the water. His response to questions and his response to commands was slow, but all movements were normal, there was no obvious muscle weakness, but there was a suggestion of diminution of light touch perception in the lower limbs.

Treatment

I formed the opinion that this patient almost certainly had cerebral decompression sickness, and I arranged for his transfer by air ambulance to Sydney, by a fixed wing aircraft, pressurised to sea level, during which time he received 100% oxygen.

Following his arrival at the Prince Henry Hospital in Sydney, he was treated in the hyperbaric unit recompression chamber for 265 minutes at 2.8 atmospheres. At the commencement of treatment, he still seemed to have poor short term memory, he was complaining of a buzzing"in the left ear, fairly severe pain in his right shoulder, hip, and abdomen. He complained of some paraesthesia and numbness in both legs.

After 10 minutes at depth he indicated that he was feeling much better, his shoulder pain had almost completely disappeared, his hip pain was 50% better, and his abdominal pain had ceased.

Six hours after the commencement of treatment he had voided urine, he appeared to be much more alert, he had lost his headache, and he only had minimal residual pain in the right hip. As well, there was only minimal residual auditory hallucination.

Finale

This diver, who had had normal long bone x-rays in

September, 1987, was considered to have cerebral decompression sickness, and he was treated with Table No. 6, resulting in a complete resolution of his symptoms, although subsequently he was still found to be slow in speech and thought. These findings were thought to be normal for him.

I contacted him by telephone at the end of May, and he is certainly still very slow in speech and thought. He has not dived again perhaps he is following the recommendation of Dr Ian Unsworth, Director of the Hyperbaric Unit, however the main reason is that his boat remains impounded somewhere in Victoria! Currently he is unemployed. He plans to take the Inspectors to the High Court, with charges of attempted murder, claiming near drowning because of the constant kinking of the hookah hose while he was being towed, and in addition the cerebral decompression sickness complication, and the fact that his boat had been illegally impounded.

Dr John McKee's address is P.O.Box 256, Bega, New South Wales 2550, Australia.

ARTERIAL GAS EMBOLISM FROM PULMONARY BAROTRAUMA: WHAT HAPPENS IN THE LUNG?

John Williamson

ABSTRACT

The precise sequence of events in the lung parenchyma which precede and accompany arterial gas embolism remain unknown. Alveolar-vascular membrane disruption is still the favoured mechanism. The base of marginal alveoli which sit on pulmonary vascular sheaths appear to be one area of weakness in the face of alveolar overdistension; this does not necessarily explain the access of alveolar gas into the lumen of pulmonary blood vessels. Clinically arterial gas embolism in divers has no clear association with pre-existing pulmonary scarring, pneumothorax, or the depth of the dive. Some association exists, or is suspected, with reduced pulmonary compliance, rapid ascents, and air trapping. Autopsies, are not good at detecting the pulmonary consequences of such events, and diving medical examinations may be missing still unrecognised predisposing factors in some susceptible would be scuba divers.

While there is at present general agreement with the assumption that in arterial gas embolism (AGE) diving gases gain entry into the pulmonary venous system via the lungs, during pulmonary barotrauma of ascent, this event has never been directly demonstrated to date. The pathogenesis of these events remain unconfirmed and yet has direct relevance to both the prevention and treatment of the disorder.

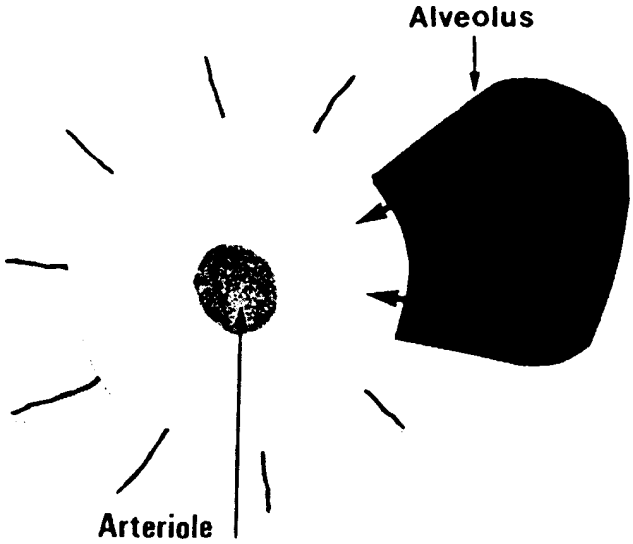

FIGURE 1 Where an overdistended marginal alveolus abuts on to a perivascular sheath in the lung, a pressure gradient may develop in favour of gas movement through the (disrupted?) base membrane (arrowheads) into the perivascular space (shown distended with gas). This will explain pulmonary interstitial emphysema, but not necessarily pulmonary gas embolism.

(Reproduced from Clinics in Anaesthesiology, with the premission of the publishers, and the author, Dr Ken Hillman.)

A search of the diving and medical literature will show that some well recognised papers[1,2] make only vague reference to how the gas actually gets into the vascular spaces. The author was able to find only one readily available text that made any attempt to grapple with this aspect of the problem[3]. Here it is suggested that once the first breath is taken (intrathoracic pressure is lowered) following pulmonary barotrauma, any extralveolar gas "can intravasate into torn vessels". (Further light has recently been thrown upon where and how the gas goes, once in the blood stream, by Gorman and his colleagues[4].)

With the steady appearance of published research (albeit mostly on experimental animals) combined with the rapidly accumulating clinical experience of diving medical physicians, who are now coping with the explosive increase world wide of recreational scuba diving, a reconsideration of certain pointers and clinical associations which are beginning to emerge may prove helpful.

Micro-anatomy of lung parenchyma

While the vulnerable site to overdistension in human lung tissue remains uncertain, animal work[1,2] suggests that at least one such site is located in the "marginal" alveoli that abut upon pulmonary vascular sheaths (Figure 1).

The delicate and attenuated alveolar-capillary membrane, particularly of the "partitional" alveoli, is a tempting anatomical site to postulate rupture and entry. However the evolving concepts of "thick" and "thin" components of this layer[5] and the existence of pore systems in this membrane[6] (pores of Kohn), together with certain theoretical pressure considerations[2], make this site as a portal for direct intravascular entry of gas an uncertain one at present.

Retrospective human studies suggest that fatal lung parenchymal barotrauma is not consistently related to pre-existing parenchymal scar tissue, nor to fixed parenchymal structures detectable by imaging and/or autopsy techniques[7].

Analogous non-diving clinical correlates

As every intensivist knows, the incidence of spontaneous gas embolism via the lungs (excluding direct penetration) in ventilated patients is a rarity. However the other pulmonary barotraumatic events, particularly, mediastinal air, pulmonary interstitial emphysema and pneumothorax, are not. These latter events are known to be associated with conditions of reduced pulmonary compliance and high peak inspiratory airway pressures[8]. Their association with positive end expiratory pressure is less clear.

Cerebral arterial gas embolism (CAGE) arising from direct lung injury (e.g. blast injury, penetrating chest wounds) does occur, although uncommon and is still frequently not thought of by attending clinicians. It usually occurs at the time of the injury, but may occur or recur during management (e.g. ventilation). The resulting central neurological disturbance may be attributed to co-incident injury and/or resultant hypoxia from other co-existing causes.

Diving related clinical correlates

As a glance through recent diving medical literature will show, it is now apparent that among recreational divers sudden impairment of consciousness associated with breathing compressed gases is by no means a rare event. It would seem likely that some of these are a result of CAGE from pulmonary barotrauma. Many of these persons recover consciousness and apparent well-being acutely and spontaneously, a fact now rendered comprehensible by Gorman's work[9]. Their subsequent medical course remains unknown, as they seldom present after that to a diving medical, nor perhaps to any, physician. However one would expect significant central neurological sequelae[4,9].

Table 1 outlines the Townsville Recompression Chamber teams' experience with recognised CAGE in scuba divers, since the chamber commenced operation in 1977. Eight (8) of these cases have been dealt with in the last 4 years. Several other episodes of sudden loss of consciousness in the water which were reported to team members during the 11 years could not be followed up.

TABLE 1

The Townsville Diving Medicine Team's Experience Cerebral Arterial Gas Embolism in Divers* 1977-March 1988

Total Number of Cases (all diagnoses)	68
Total Number of CAGE Cases	9
CAGE Fatalities	3
Identified Ascent Problems	5

*****This pattern is similar to world wide experience[11].

From our experience, and from the documented experience of others, we can say that:

1. In sport scuba-divers, the combination of clinically demonstrable CAGE with other forms of pulmonary barotrauma (e.g. pneumothorax, pneumomediastinum and subcutaneous air) is decidedly uncommon. In our experience it is less than 5%. It seems as if the development of any of the latter intrathoracic phenomena "protects" against the occurrence of CAGE.

2. CAGE in scuba-diving invariably occurs in association with an ascent, planned or accidental. CAGE may occur in the absence of any recognised problem during planned ascent, and in divers of all levels of experience with no known or previously detectable (at present) predisposition.

3. There appears to be no association between CAGE in diving and:

   (a) pre-existing scar tissue in lung or pleura[8]
   (b) measured lung size[10]
   (c) depth of the dive.

4. An association does appear to exist between CAGE in scuba-diving and:

   (a) decreased pulmonary compliance[11]
   (b) rapid ascents, irrespective of the depth of the dive. This may perhaps be related to the rate of intrapulmonary gas pressure rise.
   (c) premature airways closure[11] which may occur during forced exhalation during ascent.

It should be remembered that CAGE occurring during breath-hold diving (snorkeling) is well documented[12], although still poorly appreciated.

5. Present macroscopic and microscopic autopsy techniques and training, although improving[7,13,14], remain inadequate for the investigation of parenchymal lung damage in fatal cases of diving-related CAGE.

6. Because of the prevailing ignorance surrounding the pathology of gas embolism in pulmonary barotrauma, and allowing for those cases of CAGE resulting from errors in safe diving technique, diving medical examinations, no matter how carefully performed (and many still are not!), may not be excluding all would-be scuba divers who are medically predisposed to this potentially lethal complication.

Epidemiological, clinical and experimental research into this problem is warranted.

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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, 1988 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.)

18 May, 1988
NEWPORT BEACH,
N.S.W. 2106

THE SOUTH PACIFIC UNDERWATER MEDICINE SOCIETY
STATEMENT OF RECEIPTS AND PAYMENTS FOR YEAR ENDING 30 APRIL 1988

	1988		**1987**	
OPENING BALANCE				
Standard Chartered Finance Ltd.	1,000		1,000	
National Mutual Royal Bank	7,361		7,659	
National Australia Bank	403		80	
Cash on hand	26		60	
		8,790		8,790
INCOME				
Subscriptions	27,475		22,945	
Interest	1,787		1,680	
		29,262		24,625
Total		$ 38,052		$ 33,424
EXPENDITURE				
Secretarial	3,735		2,696	
Stationery	198		240	
Journal	10,487		6,481	
Postage	4,198		2,465	
Travel	10,335		4,415	
Equipment, see note	850		7,451	
Miscellaneous	358		576	
Bank charges	286		268	
Returned cheques	25		42	
Audit	200		-	
Legal expenses	392		-	
		31,064		24,634
CLOSING BALANCES				
Standard Chartered Finance Ltd.	1,000		1,000	
National Mutual Royal Bank	5,177		7,361	
National Australia Bank	789		403	
Cash on hand	22		26	
		$ 38,052		$ 33,424

NOTE: Equipment is written off as purchased.

THE MINUTES OF THE ANNUAL GENERAL MEETING OF SPUMS JUNE 1988, MANA ISLAND, FIJI

The Annual General Meeting was opened by the President, Dr. A. Slark at 16.45.

Present

All members attending the Scientific Conference.

Apologies

Drs D.Brownbill, P.Chapman-Smith, D.Davies, M.Davis, D.Gorman, C.Lourey, P.McCartney, J.Mannerheim, A.Veale, D.Walker.

Minutes of the last meeting

These had been previously published and were taken as read. Acceptance moved: Westlake. Seconded: Barry. Carried.

Business arising

1 Telephone Conference. The Secretary reported that he had investigated the costs of holding Executive Meetings by telephone conference hookups. The costs are those normally applying to STD and ISD calls to each person plus $1.80 surcharge for each person plus the particular person fee on all ISD calls. At current rates this would amount to about $10.00 per minute.

Dr. Sutherland pointed out that SPUMS is currently only funding 2 Executive Meetings per year.

2 Connection to Viatel. This facility is currently not a viable proposition for the Society. SPUMS has neither the capacity nor the potential market to justify an outlay of around $50,000 per year.

Alternatively, "Keylink", which is an electronic mailbox facility into which messages, copies of papers, etc., can be put by use of a modem. The recipient electronically opens the box regularly and extracts the information onto his own P.C. Costs would be much more compatible with the Society's standing. Dr.J Lloyd volunteered the use of his mailbox. The Secretary will liaise with Dr.Lloyd.

President's Report.

This was presented to the meeting. The full text was published in the SPUMS Journal, Vol 18, No 2 page 62.

Secretary's Report.

This was read to the Meeting in the Secretary's absence and was published in the SPUMS Journal, Vol 18, No 2 page62.

Treasurer's Report.

The financial statement was explained and the meeting agreed that the annual subscription be held at $35.00 for Members and $25.00 for Associates. The Statement of Receipts and Expenditures appears on page 93. Acceptance of the Treasurer's report was moved by Dr.Haller. Seconded: Dr.Chesterfield-Evans. Carried.

New Executive Committee

President:	Dr. A. Slark
Immediate Past President:	Dr. C. Acott
Secretary:	Dr. D. Davies
Treasurer:	Dr. G. Barry
Editor:	Dr. D. Walker
Committee:	Dr. C. Lourey
	Dr. P. McCartney
	Dr. D. Gorman
President, NZ Chapter:	Dr. P. Chapman-Smith

New Business

Name Change

The President brought to the attention of the meeting the recent change of name of UMS to include the word "Hyperbaric". He raised the question that perhaps SPUMS should follow suit. The consensus was that the name should remain unchanged. Dr.J Williamson pointed out that SPUMS was the only Society almost exclusively concerned with recreational and sports diving and the membership was actively involved.

Advertising in the Journal.

The need for the Journal to become self-supporting was discussed. Any profits would benefit the Society. The Editorial Committee will investigate. It was suggested that Diving organisations and travel agents be approached. Further discussion centred on Guest Editorials and Journal articles.

Life Member.

It was proposed by the President and Dr.Knight that, in the Bicentennial year, Dr.C. Edmonds, the Foundation President, be elected to Life Membership of the Society. This was carried by acclamation.

Venue for 1989 AGM.

This was discussed more fully at a separate, informal meeting. The Executive have suggested that Port Vila be the appropriate venue.

There being no other business the President thanked all members for their attendance and closed the meeting at 1900 hours.

ORIGINAL ARTICLES

DYSBARIC ILLNESS TREATED AT THE ROYAL ADELAIDE HOSPITAL 1987 A FACTORIAL ANALYSIS

D.F. Gorman, A. Pearce and R.K. Webb
Hyperbaric Medicine Unit, Department of Anaesthesia and Intensive Care,
Royal Adelaide Hospital.

Introduction

The accepted predispositions to both decompression sickness (DCS) and arterial gas embolism (AGE) in divers are listed in diving medicine texts[1], but supporting data are often anecdotal, and analyses of series of divers with these diseases have caused the significance of some of these predispositions to be questioned[2-4]. The grading of DCS into clinical types I and II does not appear to be predictive of outcome[5,6], perhaps as a result of the remarkable variability seen in the manifestations of this illness[7]. This variability, and the limited data available for the treatment of AGE[8], may explain why the management of these conditions remains controversial. Also, while it is conceded universally that both DCS and AGE can affect the nervous system[3,9-13], agreement on the frequency of neurological involvement[6, 10, 12-15], and on both the frequency and the nature of neurological sequelae[6, 16-21], cannot be reached.

It is clear that the understanding of these illnesses is limited.

During 1987, 64 divers were treated at the Royal Adelaide Hospital Hyperbaric Medicine Unit (RAH HMU) for either DCS or AGE. While this is not a large series, it is nevertheless of interest in that the mean time from the onset of the diver's symptoms and signs to compression in a recompression chamber (RCC) was relatively long, 26.4 hours, and the frequency of incomplete resolution of symptoms and signs at the time of the diver's discharge from the hospital was high, 54.5%. Consequently, a factorial analysis of these diving accidents was performed to determine what factors were associated with a poor outcome. The results are discussed in the context of the controversies listed above.

Clinical Review Period; 1987

Sixty-four divers suffering dysbaric illness, 58 with DCS, and 6 with AGE, were treated by recompression at the RAH HMU. None of these had used gas mixtures other than air. All of the divers were treated in a multiplace RCC, and with one exception they were intravenously rehydrated with crystalloid solutions. Only 2 of the divers with DCS were given intravenous steroids and none intravenous lignocaine. Amongst those with AGE, one diver received both intravenous steroids and lignocaine.

The RAH HMU protocol for treatment of either illness involves initial compression to 2.8 Bar absolute pressure (1 Bar = 1 atmosphere) and administration of 100% oxygen to the casualty. For those being treated within 24 hours of the onset of their disease, if this regimen fails to achieve significant relief, then the diver is changed to either an oxygen-nitrogen or an oxygen-helium mixture (depending on the gas breathed during the dive) and compressed further to either 4 or 6 Bar. Recurrent or persistent symptoms and signs are treated with daily hyperbaric oxygen (HBO) exposures, until either resolution has been achieved or the deficit is considered refractory to treatment.

Of those divers with DCS, 22 presented for treatment within 24 hours. All had their symptoms and signs completely relieved during the initial compression treatment. However, 16 relapsed and required repeat HBO treatments. Repeated treatments were successful in only 6 of these 16 divers, so that 10 left hospital with persistent symptoms and signs.

The remaining 36 divers with DCS (62%) presented for treatment 24 hours or longer after the onset of their illnesses. Despite repeated HBO treatment, 21 of these divers never experienced complete resolution of their symptoms and signs.

Overall only 27 divers with DCS (46.5%) had complete relief during their period of hospitalisation. None of the residual symptoms and signs in the remaining 31 divers with incomplete resolution were present prior to the current episode of DCS. These residual phenomena were predominantly arthralgia (25 divers) and neurological symptoms and/or detectable neurological deficit (8 divers). Two divers had both arthralgia and a neurological deficit.

Only 6 of the 64 divers had AGE. In 2 of these symptoms and signs did not fully resolve.

Data from all divers were recorded on a designated proforma and transferred to a computerised data base (dBase III plus). Data were analysed using Fisher's Exact Probability Test[22].

Results

The data for all divers with DCS are summarised in Table I (p 100) and for those with AGE in Table II (p101).

Those factors that were significantly ($P < 0.05$) associated with incomplete resolution of DCS symptoms and signs, with the calculated level of probability,are listed in Table III. Those factors that were not significantly ($P > 0.05$) associated with incomplete resolution of DCS symptoms and signs, with the calculated level of probability, are listed in Table IV.

TABLE III

FACTORS SIGNIFICANTLY ASSOCIATED WITH INCOMPLETE RESOLUTION OF DCS SYMPTOMS AND SIGNS IN 58 CASES OF DECOMPRESSION SICKNESS

	Factor	Fishers Exact Probability
1.	Previous DCS:	0.012
2.	Compliance with DCIEM tables:	0.050
3.	Abalone divers:	0.003
4.	Number of ascents:	
	1 v more than 1;	0.034
	Less than 3 v 3 or more;	0.045

TABLE IV

FACTORS NOT SIGNIFICANTLY ASSOCIATED WITH INCOMPLETE RESOLUTION OF DCS SYMPTOMS AND SIGNS IN 58 CASES OF DECOMPRESSION SICKNESS

	Factor	Fishers Exact Probability
1.	Age: Less than 40 .v 40 or older;	1.000
2.	Sex:	1.000
3.	Occupation:	
	Manual v sedentary;	0.590
4.	Maximum depth:	
	Less than 10 msw v 10 msw or deeper;	0.116
	Less than 20 msw v 20 msw or deeper;	0.162
	Less than 30 msw v 30 msw or deeper;	0.172
	Less than 40 msw v 40 msw or deeper;	0.694
	Less than 50 msw v 50 msw or deeper;	1.000
5.	Number of divers:	
	1 v more than than 1;	0.782
	Less than 2 v 2 or more;	0.292
	Less than 6 v 6 or more;	0.131
6.	Type of disease	0.773
7.	Time to onset of symptoms:	
	1 hour v more than 1 hour;	0.174
	Less than 6 hours v 6 hours or more;	0.425
8.	Delay prior to compression:	
	Less than 6 hours v 6 hours or more;	0.212
	Less than 12 hours v 12 hours or more;	0.193
	Less than 24 hours v 24 hours or more;	0.420
	Less than 48 hours v 48 hours or more;	0.271
9.	Initial hyperbaric treatment table:	

TABLE I

58 CASES OF DECOMPRESSION SICKNESS

1.	Mean age:		30 years	
2.	Sex:	Males;		46
		Females;		12
3.	Occupation:	Manual;		35
		Sedentary;		23
4.	Activity:	Recreational;		39
		Instructing Recreational		1
		Scientific;		3
		Fishing (eg. abalone);		12
		Commercial;		2
		Dry Chamber dive		1
5.	Previous decompression sickness:		13 (22%)	
6.	Mean experience (one diver had no formal training)		2.17 years	
7.	Mean depth:		25 msw	
8.	Gas mixture:	Air ;		58
		$O2N_2$;		0
		O_2H_e;		0
.	Compliance with DCIEM tables:		18 (31%)	
10.	Decompression tables:	None;		15
		USN;		16
		PADI;		19
		Others (eg. RNPL);		8
		Decompression meters;		0
11.	Repetitive dives:		40 (69%)	
12.	Mean number of dives:			2.9
13.	Mean number of days diving:			2.2
14.	Mean number of ascents:			3.15
15.	Alcohol intake on the same day before the dive:			0
16.	Symptoms precipitated by decompression to altitude:		5 (8.6%)	
17.	Equipment failure:		6 (10.3%)	
18.	Mean time of onset of symptoms and signs:		6.48 hours	
19.	Type of disease:	Type I;		17
		Type II;		41
20.	Diving Emergency Service contact:		41 (70.7%)	
21.	Retrieval to RAH HMU:		43 (74.1%)	
22.	Retrieval by transportable RCC:		30 (51.7%)	
23.	Mean delay from onset of DCS to compression in a RCC		26.4 hours	
24.	Multiple hyperbaric treatments:		44 (75.9%)	
25.	Mean number of hyperbaric treatments:			3.43
26.	Intravenous steroids:		2 (3.4%)	
27.	Intravenous lignocaine:			0
28.	Outcome:	Complete resolution:	27 (46.5%)	
		Incomplete resolution:	31 (54.5%)	

TABLE II

6 CASES OF ARTERIAL GAS EMBOLISM

1.	Mean age:		31.6 years	
2.	Sex:	Males;		
6		Females;		0
3.	Occupation:	Manual;		5
		Sedentary;		1
4.	Activity[1]:	Recreational;		3
		Instructing Recreational;		1
		Scientific;		1
		Fishing (eg. abalone);		0
		Commercial;		1
5.	Previous arterial gas embolism:			0
6.	Mean experience (3 experienced divers and 3 scuba trainees):		3 years	
7.	Mean depth:			25 msw
8.	Gas mixture:	Air;		6
		O_2N_2;		0
		O_2H_e;		0
9.	Compliance with DCIEM tables:		3 (50%)	
10.	Decompression tables:	None;		1
		USN;		2
		PADI;		
		Others (eg. RNPL)		1
		Decompression meters;		0
11.	Repetitive dives:		2 (33.3%)	
12.	Mean number of dives:			2.5
13.	Mean number of days diving:			2.
14.	Mean number of ascents:			1.0
15.	Alcohol intake on the same day before the dive:			0
16.	Symptoms precipitated by decompression to altitude:			0
17.	Equipment failure:		2 (33.3%)	
18.	Mean time of onset of symptoms and signs:		19.8 minutes	
19.	Type of disease:	Neurological;		6
		Cardiological/pulmonary;		3
20.	Diving Emergency Service contact:		3 (50%)	
21.	Retrieval to RAH HMU:		4 (66.7%)	
22.	Retrieval by transportable RCC:		3 (50%)	
23.	Mean delay from onset of AGE to compression in an RCC:		19.98 hours	
24.	Multiple hyperbaric treatments:		6 (100%)	
25.	Mean number of hyperbaric treatments:			2.9
26.	Intravenous steriods:		1 (16.7%)	
27.	Intravenous lignocaine:		1 (16.7%)	
28.	Outcome:	Complete resolution:	4 (66.7%)	
		Incomplete resolution:	2 (33.3%)}	

Discussion

Most reviews of divers with dysbaric illnesses have described early recompression treatment, and have reported high resolution rates[14]. However, the same authors have described persistent arthralgia in some divers despite conventional therapy, and furthermore, the frequency of neurological sequelae varies with the extent of investigations and the intensity of follow-up examinations[6, 16-21]. The natural history of these persistent symptoms and signs is for spontaneous resolution over weeks to months[6,14], and this resolution can occur despite persistent histologically-evident nervous system damage[23]. As such, much of the nerous system recovery may be due to the recruitment of previously uncommitted neurons.

Delay prior to treatment appears to be an important determinant of outcome[3, 9, 24-27]. In a series such as the one reported here, this finding will be obscurred by the earlier onset of fulminant neurological DCS in comparison to that of milder disease[28], and the more likely early presentation for treatment of divers with such severe disease. The important finding in this series is the frequency of incomplete resolution of symptoms and signs. Regardless of the outcome of these residual symptoms and signs, their frequent occurrence enables a factorial analysis to be performed. Such analyses are impossible in those series where the resolution rates are high[14].

Predispositions to DCS

The accepted predispositions to DCS include[1, 29], being female, increasing age, multiple dives, multiple ascents, multiple days diving, diving at altitude, decompression to altitude after diving, ingestion of alcohol, exercise, cold stress, obesity, dehydration, retention of carbon dioxide, physical injury, fatigue and the level of complement protein activity.

The data presented here do not permit detailed discussion of most of these phenomena, because the total diving exposure of the community from which our patients are derived is not known. For example, it is necessary to know the percentage of the total hours of diving attributable to females, before the significance of the 12 female divers (20.7% of the total) with DCS in this series can be determined.

However, some significance can probably be attributed to the following observations: 69% of all of these divers had dive exposures that exceeded the limits of the DCIEM decompression tables[30] These tables were chosen as a reference because they were developed after extensive laboratory and field testing and the associated probabilities of DCS are known[31]. 25.8% of the divers were not using a decompression table of any sort. 69% of the divers developed DCS after a series of repetitive dives. Only 10.3% had their episode of DCS precipitated by equipment failure. The majority of patients (70.7%) had neurological symptoms and signs. And despite 75.9% of these diver patients receiving multiple HBO treatments, 54.5% had incomplete resolution of their symptoms and signs. In addition, 5 divers developed symptoms of DCS only after a decompression to an altitude of more than 300 metres above sea level. In 3 of these episodes, this decompression occurred between 12 and 24 hours after the dive, and in one, more than 24 hours after the dive. DCS occurred in 2 of these 5 divers despite them having done dives that complied with the DCIEM tables[30].

It is reasonable to argue both that the use of any decompression table is probably better than using none at all, and from the data presented here, that recreational divers should use a conservative decompression table such as that produced by DCIEM[30]. The frequency of repetitive diving in this series of DCS is consistent with convention[1], but contrasts with the 1987 experience of the United States of America Divers' Alert Network (DAN), in which 75% of DCS episodes resulted from a single dive[2]. Nevertheless, the observation that repetitive diving is a risk factor for DCS is not surprising, given the demonstrated slow clearance of inert gases from biological systems in comparison to rates of uptake[32], and the observation that should gas phase separation occur during the decompression from a dive, that gas elimination will be even slower on subsequent (repetitive) dives[33].

The data presented here are also in conflict with the 1987 DAN experience in 2 other areas: the absence of alcohol as a risk factor (c.f. DAN; alcohol in 50% of cases of DCS); and the absence of decompression meters as the only controller of decompression (c.f. DAN; 38 cases of DCS in divers using meters in such a fashion)[2]. The former observation is particularly surprising, and the latter may change as the use of decompression meters becomes more widespread in Australia[34].

Although it is claimed by some authors that the majority of divers who develop DCS after a dive where nitrogen is the diluent gas involved will have isolated musculo-skeletal disease[14, 15], 70.7% of divers with DCS in this series had overt neurological symptoms and signs. These data are consistent with those reports that suggest that most DCS incidents will involve the nervous system[10, 12, 13].

Despite multiple hyperbaric treatments, more than half of these divers did not have their symptoms and signs fully resolved before they left hospital. Although this greatly exceeds the anticipated failure-rate for hyperbaric treatment of this disease[14], it is in agreement with the finding that almost half of a series of divers with DCS had abnormal electro-encephalograph (EEG) findings one week after they had completed treatment for DCS[6]. In that series the prevalence of abnormal EEGs fell significantly ($P < 0.0001$) during the subsequent month suggesting that the abnormalities were indeed related to the episode of DCS.

The time-frame of the altitude-precipitated episodes of DCS is a strong argument that divers should not fly within 24 hours of a dive, regardless of the nature of that dive.

Predispositions to AGE

Although not established, it is widely accepted that AGE complicates the decompression of divers because of an increase in airway pressure which can cause gas embolism of the pulmonary veins[9, 35, 36]. Consequently, it is argued that rapid decompression, breath-holding, and pulmonary pathology that can result in air trapping predispose to AGE[1]. However, reviews of patients who have suffered AGE have shown a very low concurrence of overt pulmonary damage (e.g. pneumothorax, mediastinal emphysema, surgical emphysema)[3]; and the pulmonary lesion that typically underlies the evolution of gas emboli in this situation has not been described. In this small series, firm conclusions are not possible, but 2 of these 6 divers performed a free ascent to the surface after equipment failure, and 3 of the 6 were recreational scuba trainees. All divers had neurological manifestations, and 3 had symptoms of chest pain and/or dyspnoea. None of these 3 divers had radiological evidence of pulmonary barotrauma, and their symptoms may have been cardiac in origin. Cardiac symptoms can occur when arterial gas emboli enter the brain stem circulation and so affecting neural control of heart function, by emboli entering the coronary circulation, and the heart chambers, or cardiac function can be indirectly affected by emboli enhancing the release of catecholamines into the systemic circulation[35, 37-40].

The mean time of onset of symptoms was almost 20 minutes after completion of the decompression. This finding conflicts with the convention that the onset of this disease is within 5 minutes of decompression[1], but is in agreement with other reports of delayed presentation[3, 9]. The probability of DCS calculated from their reported dive profiles was less than 1% ($p\ DCS < 0.01$) for each of these divers[31], so an incorrect diagnosis is unlikely. Two of the 6 divers had incomplete resolution of their symptoms and signs despite repeated HBO exposures, but given their delay prior to treatment (mean delay = 19.98 hours) this is not surprising[3, 9, 24, 25, 27]. These small numbers prevent further analysis.

Factors that influence the outcome of patients treated by recompression for DCS

The following were not significantly associated with outcome (complete or incomplete resolution): sex, occupation, increasing age, increasing maximum depth, an increasing number of dives, the type of DCS presentation (I or II)[5], increasing time from decompression to the onset of symptoms, and from the onset of symtoms to compression; the initial hyperbaric treatment used, and the number of hyperbaric treatments. While the insignificance of age is surprising, and it appears from other studies that the more severe the DCS the shorter is the latency before symptoms develop[28]; many of these results can be explained by the probable earlier presentation for treatment of those divers with the more severe forms of DCS, and the more frequent and aggressive treatment of such divers. The similar frequency of incomplete resolution of symptoms and signs for those divers with type I and those with type II DCS can also be explained by this phenomena, but may also be interpreted as supporting the argument that this typing of DCS is not predictive of outcome[6]. Indeed, it is likely that many divers with type I DCS have covert nervous system involvement[6].

In contrast, being a diver whose occupation is collecting abalone, a history of previous DCS, having had a diving exposure that did not comply with the DCIEM decompression tables[30], and an increasing number of ascents (decompressions) were all significantly associated with a poor outcome (incomplete resolution).

The local abalone diving community has been investigated previously[16], and shown to have a high incidence of DCS, and an increased prevalence of hearing loss and dysbaric ostenonecrosis. Twelve abalone divers were treated for DCS in this series, and 11 had incomplete relief. Nine of the 12 abalone divers had a past history of at least one episode of DCS.

The association between a previous history of DCS and poor outcome is highly suggestive that much of the recovery from DCS may be due to phenomena such as neuron recruitment, and requires that a return to diving after an episode of DCS should involve very conservative decompression practises to minimise the risk of further DCS.

The significance of both complying with conservative decompression tables such as those issued by DCIEM[30], and of minimising ascents so as to improve outcome is obvious from these data. Multiple ascents are already known as a risk-factor for DCS[1], but this study shows that not only may multiple ascents increase the probability of a diver developing DCS, but also that dives involving multiple ascents reduce the chances of a good outcome after treatment of any ensuing DCS.

Summary

The effects of treatment in a series of 58 divers with DCS and 6 with AGE, that occurred after dives involving compressed air, are presented. The findings in this series include a predominance of neurological DCS, long delays prior to treatment by recompression, and a poor overall resolution rate. Factorial analysis shows that the chances of a poor response to treatment increase if the diver has a previous history of DCS, if the diver is are an abalone diver; and if the dive profile did not comply with conservative decompression tables such as those of DCIEM[30], or if it involved multiple ascents.

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DIVING ACCIDENTS WHAT IS THE MAGNITUDE OF THE PROBLEM IN NEW ZEALAND ?

Allan F.N. Sutherland

Diving is one of the fastest growing sports with the New Zealand Underwater Association (PADI Franchise) now claiming that 5% of New Zealand's adult population has been either trained, or is undertaking scuba diving from training numbers and tank inspections. This figure is twice the percentage quoted from Australia of 2.5% and higher than that of other countries. In New Zealand, the numbers of both deaths and accidents requiring hyperbaric oxygen, recompression therapy, are on the increase. Last year there were 13 deaths and 35 recompression therapies (30 in Auckland) with no accurate count of the number of serious

ear problems or other major scuba diving injuries. But from experience, there must have been many times more with decompression therapies being but the tip of the iceberg. Consequently diving medicals, accident prevention and treatment are areas of medical attention by doctors interested in Sports Medicine. So far in 1988 (May)there have been 7 deaths (scuba) and 15 recompression.

At a recent course at the Philomel Naval Hospital conducted by Dr Des Gorman, a Hyperbaric Medicine Specialist from the Royal Adelaide Hospital in South Australia, the physics, physiology and mechanisms of air embolus and decompression sickness were clearly presented, as were other diving medical problems related to lung and ear.

Air embolus is an intravascular collection of air or other respired gas resulting from barotrauma to lungs usually after a rapid ascent from depth. The exact site of this intravascular gas entry in the lungs is only rarely accurately located. The resultant gas can pass to vital areas such as the cerebral circulation if the person is head up and the coronary circulation if prone. Cerebral arterial gas embolsim, CAGE, causes loss of consciousness and other neurological symptoms. The natural history of CAGE is that some cases spontaneously resolve, regaining consciousness if unconscious, as the gas embolus passes through the cerebral circulation.

Decompression sickness (DCS) is now thought to be a tissue disease rather than a vascular disease with nitrogen dissolving more slowly out of some tissues, especially fatty tissue, neural tissue and myelin sheathes at a rate slower than it can be cleared and thus bubbles are formed. It is these bubbles which cause local tissue effects of local compression, evoking chemical effects and rupturing into blood vessels. The single and multi tissue models of nitrogen off-gassing used by decompression tables and decompression meters have little relevance except as an empirical model when one considers the multiplicity of tissues which are off-gassing and at varying rates. Doppler studies show intravascular bubble formation in most divers who have dived below 30 feet. These venous bubbles usually clear in the circulation at the lung unless there is an arterial-venous connection and momentary back flow, e.g. Atrial septal defects are potentially patent in 20% of the population.

Intravascular bubbles, be they air embolus or decompression sickness in origin, not only can cause immediate intravascular effects, but can become lined by surfactant produced in the lungs making these bubbles stable. This may explain why delayed signs and symptoms, especially with DCS, present many days after exposure.

Any patient presenting with unusual signs or symptoms following a scuba dive should have a careful history and examination with the physician considering a scuba diving cause. The first aid management is as follows for acute dive accidents:

1. A, B, C, resuscitation.
2. Head down 30° left lateral.
3. Give fluids, preferably intravenous and carefully record fluid balance.
4. Give oxygen at maximal rate, carefully recorded.
5. Obtain diving medical advice re resuscitation, diagnosis and retrieval to an appropriate treatement site.

THE DIVER EMERGENCY SERVICE

This toll-free, New Zealand-wide, telephone number, paid for by the New Zealand Underwater Association, located at the Philomel Naval Hospital is (09) 458-454. South Island cases being referred to Christchurch (03) 792-900.

This is a summary of a paper presented to the International Sports Medicine Meeting held in New Zealand 12th to 15th May 1988

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HOW DO AMERICAN DIVERS DIE?
A Review of the Scuba Diving Fatalities in the USA in 1985

John Lippmann

This review is a summary of an extensive report titled "U.S. Underwater Diving Fatality Statistics, 1985" issued by the National Underwater Accident Data Centre (NUADC) at the University of Rhode Island.

The full report addresses two distinct types of underwater fatality. The first is the Non-occupational fatality, which includes all fatalities of a sport or recreational nature which occurred while using scuba (or in a few cases, some other type of underwater breathing system). The second type of underwater diving fatality is titled Occupational, and addresses fatalities associated with professional, commercial and military diving.

This review will only consider the Non-occupational fatalities since these are far more relevant to the sport diver.

NUADC defines an active diver as one who dives at least three times per year, and estimates that in 1985 there

were 2.6 to 2.9 million such divers in the U.S.A. This estimate allows for drop-outs, cross-certifications and speciality certifications, but did not count resort courses, which are thought to account for 300,000 to 400,000 dives per year.

In 1985 there were 76 Non-occupational fatalities, 14 Occupational fatalities and 8 skindiving deaths.

The total of 76 Non-occupational deaths gives a fatality rate of 2.6 to 2.9 deaths per 100,000 divers. When this rate is compared to the rates for previous years, it appears that sport diving may be becoming safer.

Location

The locations of the non-occupational fatalities were as follows:

54 occurred in ocean, bay or sea (this represents 71% of the total)
12 occurred in lakes
9 occurred in caves
1 occurred in a river.

Thirty fatalities occurred while the divers operated from the shore or a shore-line facility, whereas 22 deaths occurred from dive charter boats. An additional 17 divers died while diving from private vessels.

Most of the sport diving deaths happened in relatively shallow water, with 75% occurring in water shallower than 27 m. On this basis the NUADC suggests that sport diving is relatively safe to depths of approximately 30 to 39 m, which is in line with the recommended depth limit of most sport diver training agencies.

Conditions

Weather and/or other environmental conditions may have been factors in several of the deaths during 1985. Three cases mentioned strong currents, four fatalities occurred in heavy or dangerous surf, and an additional eight divers died in conditions where the wave height was greater than 0.7 m (although in these latter cases the wave height may not have been a contributory factor to the accident).

Age distribution

Thirteen of the 1985 fatalities were divers aged more than 50 years. This represents 17% of the total deaths, which seems to me to be disproportionately high for the number of divers in this age group. Although the NUADC were hesitant to make any statement about this high percentage, it appears that divers older than 50 years of age may have a greater risk of becoming a diving fatality than a younger diver does.

Diving experience

The latter three levels in Table 1 indicate an exposure level far higher than in the first two levels, and may indicate dozens or even hundreds of dives per victim. The more dives one does the more one exposes oneself to the risk of an accident.

Fatalities while under instruction

In 1985, 8 fatalities occurred during formal diver training and one occurred while the victim was being taught by a "friend" who was not qualified to teach diving. I have summarised specific aspects of these deaths as it may provide some interesting insights, especially for diving instructors.

1. A 60 year old female doing an advanced level diving course developed difficulty after a dive to 15 m for 30 minutes, ascended safely to the surface and complained of a tight wetsuit and difficulty breathing. She began foaming at the mouth and passed out. Despite extensive CPR she died. Her medical history indicated a diabetic condition and possible heart problems. The exact cause of death was not confirmed.

2. A 29 year old male who was 14-18 kg overweight was diving with group of 13 students led by one instructor and one assistant instructor. It was their first openwater dive. About 6 m from shore the victim tried to descend but surfaced immediately, thrashing wildly. He lost consciousness and was rushed to shore where CPR was begun. The cause of death was drowning.

3. A 47 year old male was doing his openwater certification dives. After diving to 15 m he lost consciousness while swimming towards the boat on the surface. His subsequent death was thought to be due to a combination of drowning and poor coronary circulation due to a pre-existing heart condition.

4. A 34 year old female became entangled in lines from a buoy and subsequently ran out of air just under the surface and drowned.

5. A 52 year old male suffered a stroke and died during an ocean dive with three other students.

6. A male dive student was noticed to be missing during a night dive to 18 m. He was found tangled in kelp with his regulator out, his weight belt in place and BC uninflated.

7. A 52 year old male surfaced several times during a 9 m reef dive. He complained of an ill-fitting mask. The last time he surfaced he lost his mask, became unconscious and died despite extensive CPR. The cause of death was arterial gas embolism.

8. A 24 year old diver became unconscious immediately after demonstrating a controlled emergency swimming ascent from 12 m. Despite extensive CPR he died of a massive arterial gas embolism.

9. An 18 year old female died during her first ocean dive. She was taking a lesson from a "friend" who was not a trained instructor. She panicked and refused to take her regulator. She had previously dived once before; in a pool.

It was noted that a disproportionate number of these training fatalities were in divers older than 35 years. It was also noted that some of the training facilities lacked immediate close-by supervision or assistance for a distressed trainee.

Cave diving fatalities

As previously mentioned, 9 divers died in caves. These included two double-fatalities.

The report notes that the pattern of cave diving fatalities has remained the same over the 15 years or more of reportage by the NUADC. The typical cave diving fatality involves young men who have completed openwater training and have had some openwater experience, but no experience whatsoever in cave diving. The NUADC has never reported a cave diving fatality in a properly certified cave diver.

Wreck diving fatalities

Four sport SCUBA divers died while diving on submerged wrecks. The depths of the dives range from 25 m to 62 m. Three of the four divers had become entangled and two were not able to free themselves. The third freed himself but lost his weight belt, had an uncontrolled ascent and died from a massive arterial gas embolism. The fourth was found to have a faulty BC and was thought to be overweighted and unable to ascend.

Thrill seeker ?

One diver was driving an underwater scooter at 22 m at night. He hit a submerged object, was knocked unconscious and died.

Autopsies

The results of the autopsies of 53 of the 76 Non-occupational fatalities were obtained and are shown in Table 2.

Probable starting causes of recreational facilities

After careful analysis of the accident and in some cases the diver's medical history, a list of the likely starting causes of the accidents was constructed. Table 3 is a summary of this list.

TABLE 1

EXPERIENCE OF NON-OCCUPATIONAL DIVING FATALITY VICITIMS, 1985

Experience	Percent of cases
First ever dive with SCUBA	2
Early openwater	19
Some experience	30
Considerable experience	37
Very experienced	12

TABLE 2

RESULTS OF AUTOPSIES, 1985

Cause of death	No. of cases
Asphyxiation or drowning	30
Barotrauma/embolism, etc.	16
Acute decompression sickness	1
Pulmonary embolism	1
Cardiovascular event (e.g. heart attack, stroke)	5
Total	53

I hope that this review provides some interesting insights to its readers. We can all learn from other people's misfortunes and mistakes, and can use this knowledge to increase the safety of our own diving.

Readers who are interested in obtaining a copy of the complete report (Report No. URI-SSR-87-19) can probably obtain a copy by writing to:

NUADC,
University of Rhode Island,
P.O. Box 68,
Kingston, R.I. 02881
U.S.A.

TABLE 3

PROBABLE STARTING CAUSES AND NUMBER OF RECREATIONAL FATALITIES

A.	**Medical and Injury Causes**	39	
1.	Possible exhaustion, embolism or panic		15
2.	Diagnosed embolism		16
3.	Cardiovascular event		5
4.	Aspiration of vomitus, etc.		3
B.	**Environmental Causes**	19	
1.	Lost/out of air in cave		9
2.	High waves/surf		3
3.	Strong current		2
4.	Entangled in kelp/weeds		1
5.	Entangled in external lines		1
6.	Suspected shark attack		1
7.	Lost in wreck		2
C.	**Equipment-Related Causes**	4	
1.	Overweighted at depth		2
2	Weight belt tangled in BC straps		1
3.	Faulty tank pressure gauge		1
D.	**Causes not defined**	14	

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THE DIVERS ALERT NETWORK REPORT 1988 COVERING DIVING ACCIDENTS IN 1987

John Knight

The Divers Alert Network (DAN) is the United States equivalent to the Australian Diving Emergency Service (DES). The DAN 1988 report makes interesting reading with masses of tables and figures. This is an attempt to convey those parts of the report that I found most interesting. The report is strong on tables but there is little text, which makes the interpretation of the tables difficult. All the tables in this paper have been constructed from, or are modified from, the tables in the report. Any errors of interpretation in this paper are mine.

The report states that DAN received 402 case reports from the US and Caribbean in 1987. 74 were not sports divers so were excluded, 63 histories were too incomplete to be used, so 265 cases were left for study. 149 (56.2%) of these decompression accidents came from the South East region, which includes Florida and the Caribbean, presumably reflecting the large numbers who dive in these tourist areas. Somewhere the mathematics are incorrect as most of the analyses were done on 264 cases with the odd one using 265. The mathematics get queerer when the case reports are broken down by region, as the cases tabulated by states in regions as having been reported in 1987 add up to 557, including 92 cases of arterial gas embolism (AGE) or decompression sickness (DCS) and AGE, instead of 402.

Symptoms and signs

DAN uses a Type I and Type II classification for DCS. Only pain, rash and itching are classified as Type I which provided 31 cases (6 female, 25 male) compared with 204 (51 female, 153 male) of Type II. For some of the analyses a disease severity code was used. Type I DCS was Code 1. Codes 2-5 were Type II and Code 6 AGE. Code 2 patients had "pain, numb/tingle, headache, skin sensation" symptoms. Code 3 "Ringing ears, dizziness, pain, fatigue, reflex". Code 4 "weakness, numb/tingle, breathing, nas/vomit, hearing loss, skin sensation, personality, walk/standing", while Code 5 had "visual-dis, speech-dis, weakness, paralysis, bladder, bowel", whether the whole constellation of symptoms and signs had to be present for each code is not spelt out. These cases who were semi-conscious or unconscious, who had convulsions or who had bilateral paralysis were classed as AGE. There were 29 (7 female, 22 male) cases classified as AGE.

Experience

The table headed "Years Diving Experience and Diagnosis Code. Analysis variable; Average number of dives a year", deals with the number of dives a year rather than years of diving. The minimum of 0 dives a year is unlikely to be achieved by a diver developing symptoms, while the maximum of 999 dives a year seems improbable, involving as it does 3 dives a day for 269 days a year and 2 a day for the remaining 96 days, for sports divers diving for fun. But American's on diving holidays in Australia have been known to do 7 dives a day. At this rate only 143 days diving would be needed for 999 dives.

Age

The ages and sex of the sample are shown in Table 1. Nearly half the victims (47%) were aged from 30 to 39. From the data presented one cannot guess why they figure so prominently. Based on the Australian diving community most of these people would have been diving for some years. I would hazard that some had got into trouble from over confidence of the years of trouble free diving; while others

TABLE 1

DIVERS AGE AND SEX

Age	Female	Male	Total	%
10-14	-	2	2	0.8
15-19	-	10	10	3.8
20-24	6	21	27	10.2
25-29	14	34	48	18.2
30-34	17	47	64	24.2
35-39	18	42	60	22.7
40-44	6	25	31	11.7
45-49	-	11	11	4.2
50-54	1	2	3	1.1
55-59	1	2	3	1.1
60-64	1	4	5	1.9
Total	64	200	264	100

TABLE 2

CERTIFICATION

	Female	Male	Total	%
Basic	14	28	42	15.8
Open Water	21	73	94	35.5
Advanced	17	42	59	22.3
Dive Master	5	10	15	5.7
Instructor	5	26	31	11.7
Other	-	1	1	0.4
None	-	11	11	4.1

could blame a return to diving after some years "retirement", and many would be at risk from not really understanding the decompression tables.

Certification Levels

The divers certification levels are shown in Table 2. These were a surprisingly high number of instructors in the sample when one considers the small proportion of divers who become instructors in Australia.

Depth and time of the precipitating dive

The report includes a graph of depth versus time for the precipitating dive. There is the USN no-decompression limit line running through the scatter of divers. Each diver is represented by a different symbol depending on whether the dive was the first, second, third, fourth, fifth or sixth for the day. One of the symbols on the chart, a solid square, does not appear in the key. It could be a printing error for a thick walled square (three dives that day) so I have counted it as such. By my count (there being no text to go with the graph) there were 72 divers who got into trouble after their first dive, 57 after the second dive, 50 after the third dive, 10 after the fourth dive, 4 after the fifth and 3 after their sixth dive of the day. This gives a total of 194 divers. 40 divers had dived dives outside the USN no-decompression limits. 16 of them were doing their second or later dive for the day. Unfortunately I could find no discussion of the repetitive nitrogen loads of the 122 divers on the graph who had dived more than once on the day of their accident. I suspect that many had not used the tables correctly.

Delay in seeking treatment

The delays in seeking advice were considerable. Only 124 (47%) contacted DAN within the first 12 hours. Of these 26 had AGE, the other 3 AGE cases rang before 24 hours had passed. Of the 235 cases of DCS only 98 (41.7%) made contact in the first 12 hours, 44 (18.7%) others contacted DAN in the next 12 hrs. Only 142 (60.4%) made contact in the first 24 hours. The next 24 hours brought to light another 43 cases (18.3%). The third day 22 people (9.4%) contacted DAN, on the fourth day another 6 rang, all later than 84 hours after the incident. 22 people waited till the 5th day or later to contact DAN. As a result the average times to contact are high, 2 days 15 hours for Type I, and 1 day 15 hours for Type II. By contrast the average time to contact for AGE was 3 hours.

Delays in achieving recompression

All the cases of AGE were recompressed within 24 hours 25 (86%) in the first 12 hours. Only 8 (26%) of Type I DCS cases were recompressed within 12 hours, 18 (58%) were under pressure within 24 hours, 4 were treated in the next 24 hours and 4 more the next day. 5 people took more than 4 days to present for treatment. The delays in Type II cases were equally depressing. 80 people (39%) were recompressed within 12 hours, 107 (52.5%) saw the inside of a chamber within 24 hours. In the next 48 hours another 46 (22.5%) were treated and 19 the next day. 13 presented for treatment on the fourth day. Surprisingly 19 (9.3%) presented on the fifth and later day.

The average times to treatment were once again distorted by the later comers, being 6 hours for AGE, 2 days and 18 hours for Type I DCS and 2 days and 17 hours for Type II.

Table 3 shows those who delayed for 10 days or more before presenting for treatment, 3 with Type I DCS and 5 with Type II, their treatments and results.

Spontaneous recovery

Of interest is the fact that 28, (2 AGE, 2 Type I, 24 Type II) (10.6%) of the 264 patients had complete relief of symptoms and 78 (12 AGE, 9 Type I, 58 Type II) (29.5%) had partial relief before compression.

Later in the report there is a table that showed that twenty-two people had symptoms which cleared spontaneously or only received first aid. It seems that this group was not recompressed. Table 4 gives the details.

Risk profiles

Once again the mathematics change. Only 214 cases of DCS were analysed, but the AGE cases had grown to 50 as opposed to 29 earlier in the report.

Risk factors for the 264 cases analysed are given in Table 5. The top four risk factors were square dives (71%), no-decompression dives (71%), dives deeper than 24 m (67.5%) and repetitive dives. 64% of the divers who developed DCS did so after a repetitive dive.

I find it difficult to reconcile 71% of the total number of DCS producing dives being no-decompression dives with 42% of the dives being outside the tables. Even if all the decompression dives (29% of the total) had been outside the tables this still leaves 13% of the total dives classified as no-decompression dives which must have been outside the tables, so not no-decompression. Perhaps there is an explanation but it is not in this report.

Asthma

10 Asthmatics appear in the statistics. Table 6 shows the activity of the asthma, diagnosis, time to onset of and first symptoms, whether the diver was within the tables, had buoyancy problems, a rapid ascent or air shortage problems and the water temperature. None of these people smoked. Case 3 had had pneumonia and bronchitis 3 months earlier and Case 7 had had epiglottitis two months before the dive. There appeared to be no relationship in three cases (8, 9 and

TABLE 3
DELAY BEFORE TREATMENT OF 10 DAYS OR MORE

Diagnosis	Time to onset of Symptom	Days before Treatment	Time Treated	Chamber Type	Residual Symptoms
Type I	1.00	11	18	Multiplace	None: Developed Aseptic Bone Necrosis
Type I	.01	10	1	Multiplace	None
Type I	6.00	35	1	Multiplace	Type I pain after 2 months
Type II	1.00	11	18	Multiplace	None
Type II	8.00	30	27	Multiplace	None
Type II	24.00	14	1	Monoplace	Pain only after 2 months
Type II	.05	42	4	Multiplace	Pain with weather changes
Type II	1.00	21	1	Multiplace	None

There is no explanation of the meaning of "Time Treated". It probably means"number of treatments"

TABLE 4
BREAKDOWN OF SPONTANEOUS RECOVERY OR FIRST AID TREATMENT ONLY CASES

Condition	No. of Cases	Time to onset of symptoms	Type of Therapy			Symptoms cleared in 2 days or less
			First Aid	Oxygen	Head down position	
DCS I	1	0:07	1 (Aspirin Only)	0	0	0
DCS II	14	0:00-26:00	5	4	2	8
AGE	7	0:01-0:15	3	2	2	5
TOTAL	22		8	6	3	13

DCS II: All of the cases treated with O_2 cleared on the day of treatment. One case used aspirin with less than 2 day recovery. Three cases cleared without treatment.

AGE: One of the cases treated with O_2 cleared the same day; the other O_2 case cleared 3-4 days after treatment. Three cases cleared in a two day period without any type of therapy.

10) between their DCS and their asthma. These figures bear out the conventional Australian view that asthmatics should not dive.

Flying after diving

70 people flew after the critical dive, 49 of them with 24 hours. 7 AGE cases were air evacuated for treatment. I suspect that there is a misprint in the report and that there were 5 Type I cases, two of which were air-evacuated for treatment. The other 3 developed symptoms either during or shortly after the flight. The remaining 37 cases all had Type II DCS. 7 flew with symptoms, 6 of them being evacuated. 4 of the 7 had dived within the USN tables. One flew after treatment. The other 29, 21 of who had dived within the USN tables, developed their symptoms during or after the flight. Flying within 24 hours of a dive is obviously risky!

Of the 21 cases who flew more than 24 hours after the last dive only 2 developed symptoms during the flight. All the others had symptoms before the flight. 3 unfortunates developed recurrences, after full relief by earlier treatment, during or after the flight. Two were retreated and left

TABLE 5
RISK FACTORS

Risk Factor	% of 204 cases of DCS	% of 50 cases of AGE
Square dive	71	90
No decompression dive	71	88
Deeper than 24 m	67.5	52
Repetitive dives	64	24
Second, or later, continuous day of diving	55	46
Current	43	30
Outside table	42	22
Fatigue	38	32
Exertion	36	22
Single dive	36	76
Single day's diving	33	50
Decompression diving	29	12
Multilevel diving	29	10
Less than 1 year's experience	25	32
Rapid ascent	23	48
Cold water	20	20
Alcohol	20	16
Equipment Problems	20	16
Smoker	18	12
Previous DCS	17.5	4
Buoyancy problem	14	28
Diving after a day's break from continuous diving	12	4
Lower air or out of air	10	22

symptom free while the one who was not retreated remained with residual symptoms. These three flew up to 8 days after the dive. Nitrogen bubbles take a long time to disappear.

Equipment failure

41 people had equipment failure as a contributing factor to their problems. Table 7 gives the details. 29 divers (70%) made a rapid ascent as a result of their equipment failure. Failure of a regulator, buoyancy vest problems and weight belt problems were usually (22/29, 75%) associated with a rapid ascent. To quote from the report: " There would seem to be a direct cause and effect between some equipment failures and arterial embolism. The relationship between decompression sickness and equipment problems is less clear and no firm conclusion can be drawn.

Not using necessary equipment

25 people (9.5%) were noted not to be using a depth gauge. 39 (14.4%) were noted not to be using a timing device. 20 people (7.5%) were noted not to be using a buoyancy compensator. These percentages seem high in the face of modern teaching, diving magazine editorials and persuasive advertising. There is no indication in the report as to how many non-users of equipment doubled up on their stupidity. But at least 39 (14.4%) of the DAN cases were diving stupidly by having at least one essential item of equipment missing and the percentage could be (if none overlapped) as high as 31.4%.

Unfortunately these figures cannot be fed into the risk table (Table 5) as, although the total number of cases is much the same, the breakdowns are different. These figures are based on 29 cases of AGE and 235 of DCS. The table is based on 50 cases of AGE and 204 cases of DCS.

Decompression computers

40 people developed problems when using a computer. For reasons that are not stated, the 5 who misused their computer, the 2 with AGE, the 36% nitrox user, and the one with the unspecified profile were excluded from analysis.

Of the remaining 31 cases 22 dives (71%) were outside the USN tables. 2 were bent using their computers for a single dive. Both were multilevel decompression dives outside the USN tables. 9 others were bent diving repetitive dives in a single days diving. The precipitating dives were 1 square dive with no decompression requirement, 4 square

TABLE 6
ASTHMATICS

Case No.	Diagnosis	Asthma: Previous Current	First Symptom	Time to onset.	Within Tables	Low on or Out of Air	Buoyancy Problem	Rapid Ascent	Water Temp°C
1	AGE	Previous	Dizzyness/ Disoriented	.04	Yes	Yes	-	Yes	22
2	AGE	Both	Unconscious	.00	Yes	-	No	Yes	21
3	AGE	Both	Hip & Chest Pain	.01	Yes	-	Yes	Yes	26.5
4	Type II DCS	Previous	Headache	.02	Yes	Yes	Yes	Yes	28
5	Type II DCS	Previous	Back Pain Numb/Tingle	.05	No	-	-	-	23.5
6	Type II DCS	Previous	Extreme Fatigue	.30	No	-	-	Yes	Cold
7	Type II DCS	Both	Numb/Tingle (3 hrs later complained of chest pain)	1.00	Yes	-	Yes	Yes	14
8	Type II DCS	Both	Nausea Vomiting and Fatigue	4.00	Yes	-	-	-	22
9	Type II DCS	Both	Numb/Tingle	6.00	Yes	-	-	-	65
10	Type II DCS	Both	Numb/Tingle	32.00	No	-	-	-	26.5

TABLE 7

EQUIPMENT FAILURES

	Total	DCS	AGE
Regulator	13	10	3
Buoyancy vest	5	4	1
Weight belt	8	7	1
Dry suit	3	3	0
Inflation hose	5	5	0
Unknown	1	1	0
Pressure Gauge	2	1	1
Watch	2	2	0
Back pack	1	0	1
Wet suit problem	1	0	1
TOTAL	41	33	8

TABLE 8

DIVE CHARACTERISTICS

COMPUTER USERS AND TABLE USERS

Dive	31 Computer Users	180 table users
	%	%
Square	32	79
No-stop	48	75
Multiday	74	65
Repetitive	77	60
Single day	26	40
Single dive	23	35
Decompression	52	25
Multilevel	68	21
Outside USN Tables	74	37

decompression dives, 3 multilevel dives with no decompression requirement and a multilevel decompression dive. 8 were outside the USN tables.

20 cases resulted from multiday diving. 5 came to grief on the first dive of the day after at least one day's diving. 1 did a square dive with no decompression requirement, 3 did multilevel dives with no decompression requirement and one did a multilevel dive with decompression. 2 of these dives were outside the USN tables. 15 came to grief during repetitive dives. 2 did square dives with no decompression requirement. 2 did square dives with decompression. 5 did multilevel dives with no decompression requirement and 6 did multilevel decompression dives. 10 of this group were outside the USN tables.

Over half the computer users (62.5%) came to grief after first dives to less than 30 m. However this table (disease severity code by depth of first dive) has a denominator of 40 cases, but no cases of AGE. If the report is dealing with all dive computer users there should be 2 cases of AGE. If the previous exclusions were in force there should be only 31 cases in all. Another mathematical puzzle.

Decompression Meters vs Tables

180 divers in this series developed their DCS after using the USN tables. There were 40 dive computer users, nine were excluded from analysis because "the computers were used improperly, or there were symptoms of air embolism". While it is reasonable to exclude the 2 cases of AGE in a comparison with safety of the USN tables excluding the others; detailed in the paragraph as decompression computers, weights the scales in favour of the decompression meters, as it is highly improbable to say the least (see page114 of this issue) that every diver using the tables could use them properly.

Of the 31 computer divers 68% had Type II DCS while 79% of the table users had Type II DCS. Not a significant difference.

The 31 computer divers estimated a collective total of 1,609 dives "per year", an average of 51 dives a year. The 180 table divers claimed a total of 8,100 dives "per year", an average of 45 dives a year. "Per year" appears to refer to the dives done in the 12 months before the incident. The risk of DCS for computer users was calculated as 1.9% and 2.2% for table users. Again not a significant difference.

The characteristics of the dives are detailed in Table 8. The figures confirm the unsafeness of square dives. The high rate of no-stop dives may partly be due to the exhortations to sports divers not to do decompression dives. Multiday diving is a high risk activity as is repetitive diving using a decompression meter. No comments about brands of meter were made because of the small sample. In later years, as the sample grows, it should be possible to construct a "best buy guide"! Multilevel diving with a computer seems to be more dangerous than with the tables, but this may merely represent the difficulty of calculating multilevel dives without a computer.

TABLE 9
DEPTH AND DCS

Depth m	31 Computer users % DCS	180 table users % DCS
above 18	2.5	15.5
18-21	2.5	11.5
21-24	2.5	8.5
24-27	19.0	17.0
27-30	13.0	15.0
30-33	19.0	8.0
33-36	6	7
36-39	6	8
39 and deeper	26	9

Reconstructed from slide 10 of the DAN report.

Differing dive depths of the deepest dive on the day seemed to influence the appearance of DCS differently for table users and computer users (see Table 9). Tables appear more dangerous at shallower depths, about the same after 26 m until 39 m when computers take over as much more dangerous. I suspect that these findings are more a consequence of the patterns of the dives done than of anything else.

Another quote from the report is applicable "For both computers and tables, repetitive and multiday diving are common risk factors. At the present, a more conservative approach to these types of diving would seem to be indicated in the use of both computers and the Navy tables.

From the DAN figures it would seem that properly used computers for diving no deeper than 30 m are no more and no less dangerous than the USN tables although they allow longer bottom times in multilevel diving. What the result would be if compared with a data base of table users who used the tables correctly is unknown. We do know that many table users use them incorrectly for the second dive. I hope that DAN's data base will, one day, be able to provide the answers.

I hope that the next DAN report will include more text describing the data laid out in the tables as no everyone is adept at extracting information from computer generated tables. Explanations of why the sample size changes from table to table would be much appreciated.

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BOOK REVIEWS

Diving Accident Management in Australia.
PADI Australia.

This publication is an excellent review of the present situation in Australia in relation to the investigation, management and statistics of serious diving-related accidents. It is a truely ecumenical review and in no manner a purely PADI orientated perspective. A few years ago it would have been inconceivable that an Instructor organisation would publish anything like this, containing as it does a discussion of fatalities in association with scuba diving. Naturally there is a claim that safety statistics have steadily improved, a matter which has received attention in the pages of the SPUMS Journal without any definitive decision being possible. Undoubtedly the information concerning the diver emergency service (DES) scheme and the National Safety Council will show many overseas readers a picture of the great strides in safeguarding divers which have occurred in Australia, which demonstrates that Australia is now in the vanguard in the management of diving problems and attempting to identify and remedy the problem areas.

Australian readers will be interested to read about the early days of diving and something of the various recompression chambers, which they will hopefully only visit socially. This reviewer is pleased to note the interest and support given to the reporting and investigation of "incidents", and a little surprised to learn some details of the background to some fatalities which had not been revealed in the relevant Inquests. The utilisation of the Provisional Reports data was a heartening demonstration of the philosophy of the DIVEDATA project, that others could use the data in a somewhat different manner than the compiler had and so illuminate problems from a fresh perspective.

The Editor is to be congratulated on the manner in which he has persuaded so many contributors to produce papers which fit together in a coherent whole. It is a publication well worth reading.

D.G. Walker

South Australian Diving Fatalaties 1950-1985
Peter Horne.
ISBN 0 9594383 3 5.
Price (including postage) In Australia $ 7.00
Overseas US $ 8.00
Available from Peter Horne
12 Addison Road,
Hove,
South Australia 5048

This completely revised edition contains comprehensive case history studies and detailed analyses of the fatal accidents which are known to have occurred in South Australia between 1950 and 1985.

This 80-page A-5 sized publication discusses.the thirty six fatalities. Ten divers died at Port Noarlunga. Thirteen divers died in the caves and sinkholes of the Mount Gambier region. The case histories and conclusions show what were the major factors responsible for causing so many divers to lose their lives.

Each case history has been extensively researched by the author, who has spent many hundreds of manhours conducting interviews with survivors and witnesses, delving into Coroner's reports and Inquest documents, and looking through dozens of newspaper microfilms in an effort to ensure that the known facts would be accurately reported in an easy-to-read format. He has succeeded in this objective The text is supported by graphs, photographs, diagrams and the most accurate underwater cave maps available so that readers who are unfamiliar with South Australia can gain an appreciation of the localities concerned and the dangers inherent in them.

Diving is often commercially promoted as a safe sport these days, with dive shops targeting specific populations to acquire trainees. This study deals with an unpleasant and rather 'negative' aspect of diving about which it would be criminally irresponsible to withhold information which could identify the dangers which existed in the not-so-distant past and which might still exist today. If Australian divers are being trained to be as equipment reliant as their American counterparts (see "How many divers: How safe the sport" on page of this issue) it is only a matter of time before someone becomes exhausted in the sea and then finds that the buoyancy compensator is defective. The efforts of the Cave Diving Association of Australia (CDAA) have kept the Mount Gambier caves open for diving. Their standards are high and the testing is rigorous. I recommend that every diver undertake category 1 and 2 training with its emphasis on buoyancy control. The ability to be completely happy about buoyancy control is well worth the effort. One does not need to be in a cave, avoiding stirring up the silt, to benefit from the ability to hover exactly where you want to. The only diver with CDAA training (but he had never sat the test and become qualified) who died in the Mount Gambier caves and sinkholes became entangled in his guideline when doing a deep dive, more than 45 m, without any work up. In other words he was not obeying the cave diving rules. In my opinion it is most unfortunate that the CDAA membership did not accept a recent proposal to limit dives in the Mount Gambier caves and sinkholes to a maximum depth of 42 m. I know depth appeals to many divers, but it has led to the death of many as well.

I strongly recommend this book to all divers interested in learning from other people's mistakes, and I hope that all our readers fall in this category.

John Knight

SO YOU THINK YOU CAN USE THE TABLES ? SO DID THESE SEASONED DIVERS

One criticism often levied at the use of decompression computers is that they make it too easy for a diver to rely on an electronic device and not his own knowledge. That argument assumes, of course, that divers know how to use the USN tables properly.

But, we've always ascertained that most divers don't know how to use the tables. Now we have some interesting supporting data from Homer Fletcher, who teaches the use of decompression tables to students in commercial diving classes. Fletcher administered a basic test to 95 certified scuba divers who were enrolled in the College of Oceaneering, a commercial diving school in Huntington Beach, California, where Fletcher teaches.

These 95 students represented a broad range of certifications levels (from basic to instructor), and experience (from less than a year to 16 years).

Before we tell you how well the students did, grab your own set of tables and take the exam. You m ay use any of the charts based on the US Navy tables, including the Nu-Way, PADI or NAUI tables.

By Ed. SPUMS J.

Those who use the Bassett tables should put their answers in the brackets. Question 4 should be ignored by Bassett table users.

The Test

1 I completed a dive on Saturday at 4.00 pm. The following day at 10.00 am I leave the surface to a depth of 63 feet. My timing device indicates a bottom time of 32 minutes. The no decompression limit is ..... (.....) minutes.

2 My repetitive dive group designation (group letter) is ..... (.....).

3 I plan to leave the surface at 12.42 pm. My repetitive dive group designation (group letter) is ..... (.....).

4 The planned depth is 55 feet. My residual nitrogen time is .....

5 The maximum time I can stay and avoid a decompression stop is ..... (.....).

6 My actual bottom time was 20 minutes. My total bottom time, or total nitrogen time is now..... (.....).

7 My repetitive dive group designation (group letter) now is ..... (.....).

8 If all of the following conditions exist, what is the proper procedure to follow (USN Dive Manual)?

a My actual bottom time for the repetitive dive in question 6 was 55 minutes.

b I surfaced without making a decompression stop.

c I have no pain, numbness or paralysis.

d There is no decompression chamber on site.

Write down your answer before going any further !

STOP

The answers are inside the back cover. But if you haven't taken the exam, don't kid yourself and say that you'll take it later. You won't. Let us urge you, before reading on, to take a few moments to get your set of dive tables to see how well you performed. Hopefully, it will be much better than Fletcher's students.

You see, only 18 of the 95 (19%) correctly answered all of the questions from 1 through 7. Newer divers were more likely to err

Years of Diving	Students	All Correct Answers	%
0-1	29	3	10
1-4	44	8	18
5-9	13	4	30
10-16	9	3	33

Diving certification level also reflects the ability to work the problems.

Level	Students	All Correct Answers	%
Basic	19	1	5
Open Water	41	6	14
Advanced	9	3	33
Advanced Open Water	7	3	43
Rescue	4	2	50
Divemaster	1	0	0
Instructor	3	1	33
No certification	11	2	18

No one answered number 8 correctly, which really can't be worked out without the USN Diving Manual. Fletcher doesn't penalize anyone for an answer, noting that it demonstrates just how complicated the tables can be. *Can you give an answer that shows an understanding of the problem ?*

The conclusion barely needs to be stated. Most divers are not proficient at using the tables. Where do you stand ?

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SPUMS MEMBERS AND THE TABLES

John Knight

This test was tried out at the SPUMS ASM in Mana Island. 50 copies of the questions, modified by the inclusion of spaces for users of the Bassett tables, were handed out on the first day after a short talk, describing the results with trainee commercial divers that had been published in *Undercurrent* , with a request that they be completed anonymously and handed in three days later. Inspite of reminders only 19 had been handed in by the deadline. While this small sample and poor response rate mean that the results are not statistically significant I think that there are lessons to be learnt.

I am pleased to record that this small sample of SPUMS members did better than the trainee commercial divers at the College of Oceaneering. Perhaps Australian sports diver training is better than that available in the U.S.A. This is unlikely seeing that PADI and NAUI courses originate in the U.S.A. However it is more likely that the SPUMS members who handed in their tests were confident that they could get the answers right ! Like Homer Fletcher I have ignored the answers to question 8, which was answered correctly only by two people whose diving tables had the information printed on the table.

Results

By certification level

Level	No	All correct	%	US %
Basic	10	4	40	5
Advanced	3	2	66	37
Openwater	4	2	50	14
Instructor	2	2	100	33

By years of diving

Years	No	All correct	%	US %
0-1	3	1	33	10
1-4	4	2	50	18
5-9	3	1	33	30
10-20	9	6	66	33

Errors

The errors were interesting. Three people got questions 1 to 6 right but got the wrong repetitive group for question 7. These three could be considered safe users of the table for a maximum of two dives a day ! Two made simple errors of addition and subtraction. Two made errors transcribing times from the tables. One person using the Bassett tables forgot to use the total time underwater for calculating the repetitive group, presumably because the diver had not read the instructions on the back of the table. One diver added the dive time to the time available instead of to the residual nitrogen time in question 6. Quite obviously this diver did not understand the USN tables.

Why no answers

It is of great interest to me why the majority of those given the questionnaire decided not to hand it in. The reasons that come to mind include idleness (the I'll do it tomorrow syndrome), fear (I am not sure that I know how to use the tables properly so I am not going to make a fool of myself),and disdain (I know how to use the table and this test is silly). From my observations on dive boats, of divers of all experience levels, it is a sad fact that a large proportion of divers wait for the divemaster to tell them their repetitive group for the second dive and their allowable bottom time. One can only conclude that a large proportion of Australian divers do not know how to use the tables. Regretfully I have concluded that fear of failure was the main discouragement to completing the questionnaire.

Recommendations

If only 40 % of well educated divers thought that they could use the tables properly the SPUMS Executive's call, some years ago, for a pass rate of 100 % correct in the test on the use of tables (part of the basic certification of divers) should definitely be implemented by the training agencies. But economics are against this simple safety measure being implemented.

Dr John Knight's address is 80 Wellington Parade, East Melbourne, Victoria 3002, Australia.

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.

AN EFFECTIVE SHARK REPELLENT EXISTS

JUST HOW NECESSARY IS IT?

Researchers have tried everything from electrical shocks, underwater guns and recorded sounds of purpoises to repel sharks, but nothing as been as effective as something we use everyday , detergent.

According to Samuel Gruber, a professor of marine science at the University of Miami, it is "100% effective" as a shark repellent. He has tested it by loading it into a squirt gun and shooting it into a shark's mouth. When the sharks get a taste of the soap, they turn tail, swim off and do not return. The active ingredients cause the shark's gills to dissolve and it paralyzes their jaws.

The discovery goes back more than ten years to the Red Sea, where marine biologist Eugenie Clark found a small flatfish, the Moses sole, which seemed impermeable to shark bites. Although a shark might have its jaws wide open, ready to clamp down on the little sole, the shark would soon become paralyzed, and swim off as quickly as it could. The poison works faster than nerve gas.

Israeli scientists were able to isolate the ingredient the Moses sole emanated, and found it chemically similar to detergent soap. The question then, became whether it could be converted into a usable repellent for divers and shipwreck survivors.

The U.S. Navy wanted to find out and provided a grant to Dr. Gruber for further research. The Navy has had a long and unsuccessful history developing shark repellent. Its biggest boner was the Shark Chaser, a packet that hung on all U.S. Navy life jackets for more than twenty-five years. Shark Chaser allegedly contained a depressant to stun the shark and a black dye to hide a person from the shark. By the time the Navy got around to testing it in the 1970s, they found that, not only did sharks like to eat the packet, but also 125 packets would be required to protect a person in a body of water the size of a swimming pool. The Navy's rejoinder was that although Shark Chaser was ineffective, it was valuable because people in the water believed in it and therefore had a psychological edge.

This time around, the Navy might be on a better research footing.

Gruber told *Undercurrent* that he and his associates have conducted tests of two common ingredients, sodium dodecyl sulfate and sodium laurel sulfate, on mako, lemon, hammerhead and blue sharks. Both worked as well as the substance exuded by the Moses sole.

They tested several delivery systems. The most effective was a pressurized metal container with a four-foot wand attached. When a shark got within six or seven feet of the diver, they would shoot the substance toward the shark's mouth. The shark would freeze then rush away, not be be seen for the rest of the day. As little as two parts per thousand of the substance were needed to be effective.

Gruber does not believe that the results merit commercial application yet and also that the delivery system needs to be developed. "We are biologists, not divers," he said, "so what served our research may not at all be best for divers."

Gruber questions whether it will be ultimately practical to impregnate a wet suit with the chemical to offer protection. "It is possible, but perhaps not feasible because the shark would have to get too close to the diver for the repellent to work."

The Navy grant has run out for Gruber and he does not know if more money is forthcoming. It is conceivable that the Navy is working on its own delivery system, which could mean that a product might eventually be commercially available. Gruber, himself, would like to get the backing of a diving company to further develop the toxin and the squirt gun.

For private parties to continue with the work, Gruber thinks that $150,000 or more would be needed "not counting design costs, marketing studies, and the production required to bring it to market and assuming whoever does it has some present production capabilities." A delivery system might involve some sort of exciting equipment like a shark dart or a bang stick, or require an entirely new product.

Gruber cautions, however, that "a group of entrepreneurs just could not do it without the production facilities and the knowhow. Just throwing money at it will not work."

As the next article by Dr. Ken Kizer points out, shark attacks are rare. And that leads divers to take more adventurous trips, seeking out bigger and bigger sharks for thrills and photography. So perhaps the value of such a device is not so much in actually having to use it, but rather in just knowing it is there.

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